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THE
SCIENCE AND PRACTICE
OF
MEDICINE.

BY

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THE BRITISH TROOPS AT SCUTARI IN TURKEY
DURING THE CRIMEAN WAR.

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the Geographical Distribution of Diseases.*

VOL. II.

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THE

SCIENCE AND PRACTICE OF MEDICINE.

PART III.—Continued.

CHAPTER XII.

GENERAL NATURE OF LOCAL DISEASES.

THIS class of diseases is intended to comprehend all those which affect the structure of special organs or particular parts of the body, and which lead to more or less marked disturbance of their functions.

Local affections are often accompanied by constitutional symptoms. Such symptoms, when they succeed to and depend upon the existence of the local lesion, are to be regarded as secondary to that lesion; but many lesions are strictly localised as the results of *constitutional* or *specific* diseases; and many such lesions have been already described under the several GENERAL diseases that have been considered in the first volume of this work. The *local affection* in such cases is often so striking and important that it has especially challenged attention, while the *constitutional state* from which it may have sprung is as yet concealed and unknown. The local lesion is then said to be secondary to the general affection. Not a few cutaneous eruptions are of this nature; so also are some forms of dropsy. In all local affections, therefore, it is always necessary to inquire into the constitutional condition and history of the patient, especially with regard to the existence of such diatheses or cachexiæ as may be associated with any of the general diseases considered under Section B. The existence of any such diathesis, cachexia, or temperament may materially modify the diagnosis, prognosis, and treatment of the local disease.

The diseases about to be considered are essentially characterised by more or less defined local lesions, with characteristic morbid phenomena, local symptoms, and physical signs.

The elementary forms of such lesions have been already considered in Part I., under "Topics relative to Pathology," pp. 36 to 294, Vol. I.

CHAPTER XIII.

DISEASES OF THE NERVOUS SYSTEM.

SECTION I.—INTRODUCTION TO THE PATHOLOGY OF DISEASES OF THE NERVOUS SYSTEM.

IT is with the accessory and essential parts of the nervous system contained within the cranium and spinal canal, or accumulated in ganglionic masses as the sympathetic system, and distributed as nerves throughout the body, that we have to deal in describing the local diseases of this section.

The interest which attaches itself to the study of the nervous system cannot be surpassed, either in a physiological or a pathological point of view. The truth and force of this statement will be more especially apparent when it is remembered that the nervous system is one of great extent;—that considered with regard to its functions, it consists of three sets of organs, each acting in a different manner; namely—(1.) *Central*, consisting of various forms of *nerve cells;* (2.) *peripheral*, consisting of certain structures, which may be called *terminal* or *end-organs*, as existing in the organs of special sense, in the skin, the muscles, and perhaps also in the walls of the blood-vessels, and in the ultimate structure of glands; and, (3.) *an intermediate* set of organs, which are the *nerves* in the form of continuous cords distributed through the various organs of the body; and which perform a function chiefly, if not solely, of a communicating or internuncial kind;—that the *nerve cells* are aggregated in certain parts of the body into masses, taking part in the formation of the *cerebro-spinal axis* and various *ganglia;*—that in these central masses changes occur which result either in motion, in control of vessels or of glands, in restraint (inhibition) of the activity of other centres, or in physical phenomena, such as sensation, feeling, volition, intellectual acts, and will;—that the nervous system exercises an influence over other great functions, such as the innervation of the digestive organs, of the heart, liver, and reproductive system;—that the brain is the organ of the Mind;—that the convolutions forming the surface of the hemispheres of the cerebrum are the parts where the mechanism or apparatus is situated which furnishes the material conditions through which the intellectual processes are manifested; and that, lastly, every mental act is associated with some molecular change in the grey matter of the convolutions. Such considerations cannot fail to show that the pathological relations of the nervous system contained within the cranium and spinal canal, and distributed throughout the body, are perhaps the highest and most important, in a scientific, philanthropic, and sanitary point of view, to which the mind of the physician can be directed. About 12 per cent. of those who die are annually cut off by nervous diseases; and the diseases of this system occupy the fourth rank amongst the maladies destructive of human life; being only surpassed in fatality by the contagious, tubercular, and respiratory diseases (ALTHAUS). The student is therefore called upon to give the DISEASES OF THE NERVOUS SYSTEM generally a large share of his study—(1.) Because of the confessedly great difficulty of their investigation; (2.) because they furnish a very large proportion of the more common ailments of humanity, while the condition of the nervous system more or less influences all other diseases.

The varied phenomena connected with the morbid conditions of the nervous system must be examined from the following points of view, namely—(1.) The purely anatomical structure, histology, surroundings, and relations of the brain and nerves; (2.) the chemical composition and properties of the nervous substance; (3.) the physiological relations of the several parts; (4.) the clinical, morbid, and pathological relations. Each of these methods of study and investigation mutually illustrate one another; and it is only from a consideration of all of them conjointly that we can arrive at the natural history of a case, and so act for the best in diagnosis, prognosis, and treatment. Every student knows how very many physiological doctrines regarding the brain and nerves receive elucidation from accurately determined anatomical information; and how such knowledge tends to explain various points in the pathology of cerebral diseases. He need only be here reminded of the phenomena explained by the decussation of the *pyramids* in the *medulla oblongata;* how the continuity of the fibres of the spinal cord upwards to the *cephalic centres* explains various secondary lesions of the brain as a consequence of lesions in the spinal cord altered by paralysis (Drs. BROWN-SÉQUARD, TURCK, WALLER, CHARCOT, and VULPIAN); and of the results of the more recent investigations of Drs. Crichton Browne, Ferrier, H. Major, Sankey, Broadbent, Lauder Brunton, H. Jackson, Dodds, Althaus, Allbutt, and many others. The observations of these men show, to some extent, how accurate anatomical investigations become valuable in the elucidation of morbid phenomena. A knowledge of anatomy is also of great service "in checking the conclusions to which we may have been led by experiment or as the results of disease; for however probable in other respects these may be, if they are not in harmony with well established anatomical facts it is certain they cannot be ultimately maintained" (Dr. W. J. DODDS, *Journ. of Anat. and Phys.*, Vol. XII., p. 636, 1878).

There is, indeed, no department in which "the search for disease has been keener," and in which a greater amount of work has been done during the past ten years than in diseases of the brain and nervous system; and in the minute investigations of their lesions this country may justly claim to have taken a very prominent, if not a foremost rank. It has been found that the causation of any case of nervous disease must be studied from three points of investigation, namely—(1.) By exact anatomical inquiry, so that the organ or part damaged may be sought out, and the seat or "*localisation*" of the disease determined; (2.) by physiological inquiry, so that the defective working of the nervous tissue of the part damaged may be searched into, and the functional affection understood; (3.) by pathological inquiry the processes by which the nutrition of the nerve centres and tissues are impaired are to be traced out (H. Jackson). Records of *post-mortem* appearances in the brain are now required to be given with as much accuracy as is consistent with anatomical information, otherwise valuable cases are lost to those who would make use of them as data for arriving at the localisation of function from the side of pathology, and so of acquiring ultimately a knowledge of the diseases of the nervous system.

The following sketch of the general structural arrangement of the cerebro-spinal system is given (having these points of investigation in view), and it is intended as an aid to the accurate recording of *post-mortem* examinations, and as a guide to the interpretation of symptoms in their pathological relations, and in connection with the clinical history of individual cases of diseases of the nervous system. Lesions affecting the hemispheres should be exactly described upon a definite and orderly method—using an uniform comparative nomenclature to which the topography of every mammalian brain may be referred, as has been done in recording the very important experiments of Dr. Ferrier and others on the brain of living animals, so as to compare them with *post-mortem* results and with clinical records of diseases of the brain and nervous system in the human individual. With this object in view, I have adopted the method of Ecker, Turner, and Ferrier; and would recommend such a general use of their maps or charts in outline as are here reproduced, supplemented by others from the careful dissections of Dr. Allen Thomson, in the last edition of Quain's *Anatomy*. These maps, charts, or plans in outline of the different surfaces and regions of the encephalon are given in order that pathological observations made with care of changes in the cortex of the cerebrum or internal ganglia of the brain should be recorded as accurately as possible; believing that "it is only by accurate clinical study, combined with the most careful *post-mortem* observation, that we shall be able by degrees to arrive at a knowledge of the several regions of the brain's surface and interior, and of the physiological significance of symptoms." It is at first difficult to recognise the constant unity of form which underlies the multiplicity of individual variations of the surface of the brain; and hence the usefulness of such maps or plans as have been devised by Ecker and Professor Turner. General use may also be made of such handy brain-charts as those edited by Drs. Tuke and Howden.*

I.—ANATOMICAL STRUCTURE, HISTOLOGY, SURROUNDINGS, AND RELATIONS OF THE SEVERAL PARTS OF THE NERVOUS SYSTEM.

1. General or Crude Anatomical Constituents of the Nervous System.—These are to be studied as arranged into three great divisions:

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* These charts are published by Maclauchlan and Stewart, Edinburgh, and Robert Hardwicke, 192 Piccadilly, London. They enable the pathologist to note accurately the exact positions of lesions of the brain and membranes; and it is suggested that the sites and limits of apoplexies, apoplectic cysts, softenings, abscesses, bullæ of fluid over atrophied convolutions, limited opacities, adhesions of membranes, tumors, and other abnormal conditions, should be indicated by different colours, as red, black, yellow, blue; and that the diagram so coloured should be cut out of the chart and pasted into the pathological record or case book, alongside the verbal description.

—(1.) As a large quantity of nervous matter collected into one mass, and contained in one cavity, the cerebro-spinal. This mass is called the *Brain and Spinal cord,* or *Cerebro-spinal axis,* or *central part of the nervous system;* and is composed of the encephalon, or brain proper, mainly filling the cranium; and of the *spinal cord,* hanging in the spinal or vertebral canal. The *encephalon* or brain proper includes a number of organs more or less distinct from each other, but connected by numerous bands of nerve-fibres. The largest of these are the *cerebrum* and *cerebellum;* and, overlapped by the former are to be found, from before backwards, the *corpora striata, optic thalami,* and *corpora quadrigemina,* constituting together the *cerebral basal ganglia,* with the various commissural parts which connect symmetrical portions on each side of the mesial plane; and, lastly, of the *pons Varolii.* (2.) The distribution of nerves or of nervous texture arranged in the form of long continuous cords or threads, running in definite directions throughout the body. (3.) An accumulation of peculiar nervous substance, in the form of small, round, and somewhat oval masses, called "*ganglia,*" variously connected with each other and with the surrounding parts, forming what is known as the *sympathetic system,* and which is intimately connected with the cerebro-spinal system, and with all the cranial nerves (except the olfactory, auditory, and optic), either direct or through a ganglion. On its fibres distributed to the viscera there are numerous ganglia (or plexuses in which ganglia exist), and frequently a plexus follows the course of each great blood-vessel. Thus the sympathetic system contributes to the innervation of all the glands, blood-vessels, and viscera in each region of the body. With the first-named central parts are associated—(1.) Three special membranes or meninges, which lie between them and the bones, and which serve for their support and protection; (2.) Vascular arrangements for a most copious supply of blood by vessels communicating in such a way as to maintain adequate nutrition with great functional activity.

2. THE MEMBRANES OF THE BRAIN AND SPINAL CORD are,—(1.) An external fibrous membrane, called the *dura mater,* which closely lines the interior of the skull, and forms a loose sheath in the spinal canal; (2.) an internal areolo-vascular tissue, the *pia mater,* which accurately covers the brain and spinal cord; and, (3) an intermediate membrane, the *arachnoid,* which lies over the pia mater, the two being in some places in close connection, in others separated by a considerable space. As considerable differences of description exist regarding the anatomy and relations of these membranes, the following account is given from QUAIN'S *Anatomy,* 8th Edition, Vol. II., p. 567; and from Professor TURNER'S *Introduction to Human Anatomy,* as to special anatomical facts which are necessary to be remembered in all investigations and descriptions as to the pathological relations of the parts concerned.

The *dura mater* is a very strong, dense, inelastic tissue, of considerable thickness. Its inner surface, turned towards the brain and spinal cord, is smooth and lined with epithelium, which has been generally regarded as constituting a parietal reflection of the arachnoid membrane. The space between the dura mater and arachnoid (formerly in like manner regarded as the sac of the arachnoid) is now conveniently termed the *subdural* space. The outer surface of the dura mater is connected with the surrounding parts in a somewhat different manner in the cranium and in the spinal canal. In the cranium it adheres to the inner surface of the bones, and forms their internal periosteum. The connection between the two depends on blood-vessels and small fibrous processes; so that the dura mater, when detached and allowed to float in water, presents a flocculent appearance on its outer surface, in consequence of the torn parts projecting from it. The adhesion between the membrane and the bone is more intimate opposite the sutures, and also at the base of the skull, which is uneven and perforated by numerous foramina, through which the dura mater is prolonged to the outer surface, being there continuous with the pericranium. The fibrous tissue of the *dura mater* becomes blended with the areolar sheath of the nerves at the foramina which give them exit. It is intimately attached to the margin of the foramen magnum; but within the vertebral canal it forms a loose sheath around the cord (theca), and is not adherent to the bones, which have an independent periosteum. The adhesions between the cranial bones and the dura mater are greater in early life than at mature age, because growth is more active at these parts in infancy and youth. Towards the lower end of the canal, a few fibrous slips proceed from the outer surface of the dura mater to be fixed to the vertebra. The space intervening between the wall of the canal and the dura mater is occupied by loose fat, by watery areolar tissue, and by a plexus of spinal veins. Opposite each intermediate foramen the dura-matral-theca presents two openings, side by side, which give passage to the two roots of the corresponding spinal nerve. It is continued as a tubular prolongation on the nerve, is lost in its sheath, and is connected with the circumference of the foramen by areolar tissue. The fibrous tissue of the dura mater (especially within the skull) is divisible into two distinct layers—at various places separate from each other, leaving intervening channels, called *sinuses* (for venous blood), which are lined with a continuation of the internal membrane of the veins. The dura mater also sends inwards into the cavity of the skull three strong membranous *processes* or *partitions*, which are formed by duplication of its inner layer. Of these, one descends vertically in the median plane into the longitudinal fissure between the two hemispheres of the cerebrum: the *falx cerebri*. The second is an arched or vaulted partition, across the back part of the skull, between the cerebrum and the cerebellum: the *tentorium cerebelli*. Below this another vertical partition, the *falx cerebelli*, passes down between the hemispheres of the cerebellum. The *falx cerebri* is narrow in front, where it is fixed to the crista galli, and broader behind, where it is attached to the middle of the upper surface of the tentorium, along which line of attachment *the straight sinus* is situated. Along its upper convex border, which is attached to the middle line of the inner surface of the cranium, runs the *superior longitudinal sinus*. Its under edge is free, and reaches to within a short distance of the corpus callosum, approaching nearer to it behind. This border contains the *inferior longitudinal sinus*. The *tentorium* or *tent* is elevated in the middle, and declines downwards in all directions towards its circumference, thus corresponding in form with the upper surface of the cerebellum. Its inner border is free and concave, and leaves in front of it an oval opening, through which the *isthmus encephali* descends. It is attached behind and at the sides by its convex border to the horizontal crucial ridges of the occipital bone, and there incloses the lateral sinuses. Farther forward it is connected with the upper edge of the petrous portion of the temporal bone—the *superior petrosal sinus* running along this line of attachment. At the point of the pars petrosa, the external and internal borders meet and intersect each other—the former being then continued inwards to the posterior, and the latter forwards to the anterior clinoid process. The *falx cerebelli* (falx minor) descends from the middle of the posterior border of the tentorium, with which it is connected, along the vertical ridge, named the internal occipital crest, towards the foramen magnum, bifurcating there into two smaller folds. Its attachment to the bony ridge marks the course of the posterior occipital sinus or sinuses. In *structure* the dura mater consists of white fibrous and elastic tissue, arranged in bands and laminæ. A layer of pavement epithelium exists upon its inner surface, which was formerly regarded as belonging to the serous membrane lining it. A similar layer of epithelium covers both sides of the spinal dura mater. The cranial membrane is traversed by numerous blood-vessels chiefly destined for the bones. An extensive system of lymph canals has been described, formed chiefly by the spaces between the fibrous trabeculæ, and bounded by cellular membranes. Minute nervous filaments, derived from the fourth, fifth, and eighth cranial nerves, and from the sympathetic, are described as entering the dura mater of the brain. Nervous filaments have likewise been traced in the dura mater of the spinal column (LUSCHKA and RÜDINGER, quoted by HYRTL). As shown in the experiments of Ferrier and others, the dura mater is extremely sensitive to irritation.

The *pia mater* is a delicate, fibrous, and highly vascular membrane, which immediately invests the brain and spinal cord. It is applied to the entire cortical surface of the convolutions of the hemispheres of the brain, and dips into all the sulci, which thus contain a double layer. From its internal surface numerous small vessels enter the substance of the brain; hence this inner surface is very flocculent, and is named *tomentum cerebri.* On the cerebellum a similar arrangement exists, but the membrane is finer, and the double fold is distinct only in the larger sulci. It is also prolonged through the transverse fissure into the lateral ventricles, and there forms the *velum interpositum* and *choroid plexus;* also into the fourth ventricle, where it forms the *choroid plexus* of the fourth ventricle. In the healthy brain the pia mater may be stripped off without causing injury to itself or to the brain. On the *spinal* cord it has been described by some as a different membrane, under the name of *neurilemma of the cord.* It is thicker, firmer, less vascular, and more adherent to the subjacent nervous matter than is the pia mater of the encephalon; and it cannot be stripped off the cord without causing injury to both. Its greater strength is owing to its containing fibrous tissue, which is arranged in longitudinal shining bundles. A fold of this membrane dips down into the anterior fissure of the cord, and serves to conduct blood-vessels into that part. A thinner process passes into the greater part of the posterior fissure. At the roots of the nerves, both in the spine and in the cranium, the pia mater becomes continuous with the neurilemma, and it presents a conspicuous fibrous band, running down in front over the anterior median fissure, named by Haller *linea splendens.* In *structure* the pia mater consists of interlaced bundles of connective tissue, having a more regular arrangement in the outer and inner layers, while in the middle is a network of fine elastic fibres. On the cord the outer fibres are for the most part parallel and longitudinal, and the inner network consists of peculiar stiff fibres bending suddenly and inclosing angular interspaces. Beneath them is a fine membrane continuous with the neuroglia of the cord; and pigmented cells are sometimes scattered among the elastic fibres. The pia mater contains great numbers of blood-vessels, which subdivide in it before they enter the nervous substance. Each vessel lies in a canal, the walls of which are composed of a more dense arrangement of the fibres of the membrane (perivascular canal). The diameter of the canal may be two or three times that of the contained vessel. A similar sheath, derived from the pia mater, accompanies the vessel into the substance of the brain. At its commencement the sheath is loose and funnel-shaped, and can be injected from the subarachnoid cavity. On the cerebrum the inner layer of the pia mater is adherent to the cortical substance of the convolutions, but on the cerebellum a space exists between the two, traversed by fibres which pass from the cerebellum to the pia mater. This space is continuous with the intervals between the perivascular sheaths and the brain substance. The pia mater contains numerous lymphatic vessels (FOHMANN and ARNOLD). Purkinje describes a retiform arrangement of nervous fibrils, derived from the sympathetic, the third, sixth, facial, pneumogastric, and accessory nerves. The spinal pia mater is supplied by nerves from the sympathetic (KÖLLIKER and others).

The *arachnoid* is a very delicate and transparent membrane which invests the brain and spinal cord outside the pia mater, and much less closely than that membrane. It passes over the various eminences and depressions on the cerebrum and cerebellum, without dipping down into the sulci and smaller fissures. Between it and the pia mater is a distinct space (*subarachnoid space*), in which is a considerable quantity of fluid (subarachnoid fluid). Its free outer surface is in contact with the smooth inner surface of the dura mater, the epithelium upon which has been regarded as a parietal layer of the arachnoid, closely united with the dura mater. At certain recesses, and near the longitudinal sinus, a small amount of loose connective tissue beneath the epithelium may be distinguished from the dense fibres of the dura mater. With this epithelium the arachnoid membrane (or visceral layer, as it was wont to be termed) has been thought to be continuous at the various foramina, the two thus constituting a closed sac similar to that of the pleura. But since the inner surface of the dura mater in the greater part of its extent does not present any distinct tissue which can be regarded as an attached membrane, Kölliker, Henle, and others consider that there is no justification for the assumption of a parietal layer of the arachnoid. Moreover, recent investigations into the relation between the arachnoid and dura mater on the nerve roots as they leave the cranio-vertebral cavity, have shown that there is no such reflection of the arachnoid on to the dura mater as had been supposed (AXEL KEY and RETZIUS). The *subarachnoid space* is wider and more evident in some positions than in others. Thus, in the *longitudinal fissure* the arachnoid does not descend to the bottom, but passes across, immediately below the edge of the falx, at a little distance above the corpus callosum. In the interval thus left, the arteries of the corpus callosum run backwards along that body. Over the *base* of the brain, and in the *spinal canal,* there is a wide interval between the arachnoid and the pia mater. Over the base this subarachnoid space extends in front over the pons and the interpeduncular recess as far forward as the optic nerves; and behind it forms a considerable interval between the cerebellum and the back of the medulla oblongata. In the spinal canal it surrounds the cord by a more loose investment, so that the space is here of considerable extent. It is occupied, in both brain and cord, by trabeculæ and thin membranous extensions of delicate connective tissue, on the one hand with the arachnoid, and on the other with the pia mater. This tissue is most abundant where the space between the two membranes is least. It is dense in the neighbourhood of the vessels, and continuous with the tissue of their walls. The *subarachnoid space* communicates with the ventricles of the brain by means of the foramen of Majendie, the opening into the lower part of the fourth ventricle, through the membrane which closes it. Two other openings through this membrane exist, one on each side, behind the upper roots of the glosso-pharyngeal nerve into the pouch-like extension of the membrane beneath the flocculus (MIERZEJEWSKY). A certain quantity of *limpid cerebro-spinal fluid* is contained between the arachnoid membrane and the dura mater, the chief part of which is lodged in the *subarachnoid space* in the meshes of the trabecular tissue. It varies in quantity from 2 drachms to 2 ounces. It is alkaline, of a specific gravity of 1·005, consisting of 98·5 parts water, the remaining 1·5 per cent. being solid matter—animal and saline. It contains a little albumen, and a substance which (Professor Turner pointed out some years ago) reduces blue oxide of copper to the state of yellow suboxide. When pressure is made upon the brain, the quantity of fluid in the spinal subarachnoid space is increased; and, conversely, it may be forced from the spinal cavity upwards into the cranium. The nerves as they pass from the brain and spinal cord receive two sheaths, an outer from the dura mater, and an inner from the arachnoid. Upon the optic nerve these sheaths remain distinct and separate, so that the space which each incloses may be injected, the outer from the subdural, the inner from the subarachnoid space. On the other nerves the arachnoidal sheath soon ceases, and the single sheath may be injected from either the subdural or subarachnoid cavity. Separate sheaths surround each bundle of the larger nerves, and the injection passes readily along the nerves, even as far as the limbs. There thus exists a continuity between the ventricles of the brain, the subarachnoid space, the perivascular canals of the cerebral substance, and the perineural spaces within the nerve sheaths. In *structure* the arachnoid consists of distinct riband-like bundles of fine fibrous tissue interlaced with one another. The intervals between these bundles are filled up by delicate membranes, composed of expanded cells, the nuclei of which persist and are scattered over the structure. Several layers of this delicate tissue, arranged in a complex way, constitute the arachnoid membrane.

The *ligamentum denticulatum* is a narrow fibrous band which runs along each side of the spinal cord in the subarachnoid space, between the anterior and posterior roots of the nerves, commencing above at the foramen magnum, and reaching down to the lower pointed end of the cord. By its inner edge this band is connected with the pia mater of the cord, while its outer margin is widely denticulated; its denticulations, about twenty-two, are attached by their points to the inner surface of the dura mater, and thus serve to support the cord along the sides, and to maintain it in the middle of the cavity. The first or highest denticulation is fixed opposite the margin of the foramen magnum, between the vertebral artery and the hypoglossal nerve; the others follow in order, alternating with the successive pairs of spinal nerves. The denticulations in the lower cervical region are very long, and ascend slightly to their attachments. At the lower end, the ligamentum denticulatum may be regarded as continued into the terminal filament of the spinal cord, which thus connects it to the dura mater at the lower end of the sheath. The free edge, in the intervals between the denticulations, is slightly thickened, and, in the body, is closely applied to the inner surface of the arachnoid, with which it is often directly connected by fine trabeculæ. The denticulations do not perforate the arachnoid, but receive from it funnel-shaped sheaths, which accompany them to the inner surface of the dura mater (AXEL KEY and RETZIUS, MAX SCHULTZE'S *Archiv.*, 1873). In *structure* it consists of white fibrous tissue, mixed with many exceedingly fine elastic fibres, which are seen on applying acetic acid. Several layers of fine connective-tissue trabeculæ may be traced; they are surrounded by sheaths, composed of delicate nucleated cells, and which here and there expand into membranes. It is continuous, on the one hand, with the fibrous tissue of the pia mater, and on the other with that of the dura mater.

Glandulæ Pacchioni are numerous small pulpy-looking elevations, generally collected into clusters, upon the external surface of the dura mater, in the vicinity of the longitudinal sinus. The inner surface of the calvarium is marked by little pits, which receive these prominences. Similar excrescences are seen on the internal surface of the dura mater, and upon the pia mater on each side of the longitudinal sinus, and also projecting into the interior of that sinus. On a careful examination of the connections of these bodies, it will be found that the elevations found on the outer surface of the dura mater and within the longitudinal sinus, are grape-like bodies which are attached more deeply, and in their growth have perforated the dura mater. Their precise origin and nature were long the subject of conflicting opinions; but it has been satisfactorily shown by Luschka and Cleland that they are only an enlarged condition of normal villi of the arachnoid, and that no other structure is involved in their formation. Each villus is covered by an epitheliated membrane, continuous with the arachnoid. Outside this is another fine membranous sheath, proceeding from the dura mater, and the interval between the two is continuous with the subdural space. Within the villus is a spongy trabecular tissue, continuous with the subarachnoid tissue, and of similar structure (see QUAIN, l. c., p. 576).

3. MASS OF THE BRAIN, ABSOLUTE AND SPECIFIC WEIGHT AND BULK OF THE ENCEPHALON.—The human brain is absolutely bigger and heavier

than the brain of any animal, except the elephant and the larger whales. It is also heavier, relatively to bulk and weight of the body, than are the brains of the lower animals, except in some small birds and mammals. Considerable variations, however, exist in the size and weight of the human brain, not only in different races of mankind, but in individuals of the same race and of the two sexes. The heaviest brains occur in the white races (Turner). The maximum weight of the adult male brain, in a series of 278 cases, was 65 oz.; and the minimum weight, 34 oz. In a series of 191 cases the maximum weight of the adult female brain was 56 oz., and the minimum weight 31 oz. Thus the difference between the extreme weights in the *male* was no less than 31 oz. and in the female 25 oz. But in a very large proportion of cases it is found that the weight of the male brain ranges between 46 and 53 oz.; that of the female brain between 41 and 47 oz.; and by taking a mean, an average weight is obtained of 49½ oz. for the male, and of 44 oz. for the female brain. Although, also, many female brains exceed in weight particular male brains, it is a matter of everyday observation that the head and brain of a woman are (on the average) smaller than those of a man—the average difference in weight being from 5 to 6 oz.; and this general superiority in absolute weight and size of the male over the female head and brain exists at every period of life, amongst all peoples and races, without exception. Amongst Europeans the average weight of the female brain is 10 per cent. less than in men—a difference which is shown to be a fundamental sexual distinction; and in correspondence with this greater size and weight of the male brain, the head of the male infant is bigger than that of the female—a circumstance which not only retards, but even renders more dangerous, both to mother and child, the process of parturition (Quain, Turner).

As to the mass and weight of the brain considered *in relation to the intelligence,* Professor Turner observes that "one almost insensibly associates the ideas of power and strength with size and weight; and when one sees a large-headed, big-brained man, one is disposed to say that such a person must be endowed with great intellectual capacity. This very general and popular conception of the relation between size of brain and degree or force of intellect, to some extent, undoubtedly, rests on a basis of truth. It is supported by the well-known examples of several men distinguished for their intellectual attainment whose brains have been weighed:—the brain of Cuvier weighed 64½ oz.; of Dr. Abercrombie, 63 oz.; of Professor Goodsir, 50·5 oz.; of Spurzheim, 55 oz.; of Sir J. Y. Simpson, 54 oz.; of Daniel Webster, 53·5 oz.; of Agassiz, 53·4 oz.; and of Dr. Chalmers, 53 oz.—weights which, even in the least heavy of these brains, are considerably above the general European male average of 40 or 50 oz." But this popular belief cannot be entirely accepted. It is found that "absolute differences in the size of the brain do not necessarily imply corresponding differences in the degree of intelligence among animals; that the brain being, likewise, an important centre of *sensori-motor* activity, its size and weight in various animals bear a relation to the size and weight of the entire body; and that brain mass and body mass are correlated with each other." The brain of a male child at birth weighs on an average nearly three quarters of a pound, or about 12 oz.; and so rapidly does it grow during the early years of childhood, that already by the age of three years it has attained more than three quarters of its full size (Dr. ALLEN THOMSON). The observations of Sims, Tiedemann, and Reid show that in both sexes the weight of the brain in general increases rapidly up to the seventh year, when it has reached the proportion of nine-tenths of its size; then only by slow and comparatively small gradations it grows to between sixteen and twenty; and, again, still more slowly, to thirty-one and even forty years of age, at which time it reaches its maximum growth. In comparing, therefore, individuals with each other we cannot say that the weight of the brain bears a direct proportion to the weight of the body; for the weight of the body varies considerably at different stages of adult life, and increases also greatly in bulk and weight after forty years of age, without any corresponding fluctuations taking place in the weight of the brain, which after that age appears to undergo a slow but progressive diminution (QUAIN, l. c.) As the body, therefore, has a tendency to increase in weight during the period of maturity, whilst that of the brain remains stationary, the relative weight of the latter during that period is always less than at or before the age of puberty. Also, it does not seem that any very precise conclusions can be drawn as to the existence of a direct relation between stature and brain weight. Professor Turner further observes, that in discussing the size and weight of the entire brain in relation to the intellectual power of the individual or race, it must be kept in mind that the gross weight of the organ represents much more than the weight of the cerebrum, and includes that of the optic lobes, the pons, cerebellum, and medulla, which serve as important *sensori-motor* organs, not directly associated with intellectual activity; and further, that the weight of the cerebrum itself represents not only that of the convolutions, but of the optic thalami and corpora striata. No reliable determinations have yet been made of the exact proportion, as regards bulk and weight, which the convolutions bear to the internal ganglia, the anatomical relations of the one to the other being of such a kind as to make it extremely difficult to estimate them. But data exist which enable us to form an estimate of the relative weight of the pons, cerebellum, and medulla to the entire encephalon. In males between twenty and seventy years of age the relative weight to these parts of the entire brain was found to be alike in males and females, in a ratio of 13 to 100. Hence the smaller weight of the brain in women is due not to a greater proportional reduction in any one part of the organ, but to a diminution in which all its subdivisions participate. In individual brains, however, a departure from this average ratio may and does arise. A brain exceeding the average in size and weight may in some cases owe its increased bulk, not to any special amplification of the convolutions, but to a greater proportional increase in the other subdivisions of the encephalon; and conversely, a brain somewhat below the average may yet have its convolutionary area developed in a greater ratio than the other parts. It also seems certain that if the human brain, even amongst the most uncultivated peoples, falls below 30 oz., such a low weight is not merely incompatible with intellectual power and activity, but is invariably associated with idiocy or imbecility. The human brain has thus a *minimum* weight below which intellectual action is impossible. Amongst the most cultivated peoples the *minimum weight-limit of intelligence* is in all probability higher than 30 oz. It has been placed by Broca and Thurnam at 32 oz. for the female, and 37 for the male; while the weight of the brain in the microcephalous idiot may fall as low as 10·6, 10 oz. and 5 grains, and 8½ oz., as in Theile, Gore, and Marshall's cases respectively (*Phil. Trans.*, 1863, p. 527, quoted by Dr. TURNER, l. c.) On the other hand, brains of idiots have exceeded even 50 oz. In one (a male aged 22) the brain weighed 59·5 oz.; in another male (aged 37) it weighed 60 oz., the cranium having an internal capacity of 110½ cubic inches (Dr. LANGDON DOWN, THURNAM, J. BATTY TUKE, in *Journal of Anat. and Physiol.*, May, 1873, quoted by Dr. TURNER, l. c.) Hence it by no means follows that when a brain exceeds the *minimum* it should be a competent organ of thought. In various forms of insanity the weight of the brain ranges from the *minimum* up to 60½ oz. It has therefore been made clear, that the size and weight of the brain cannot *per se* give an exact method of estimating the intellectual power of the individual, and that a high brain-weight and great intellectual capacity are not necessarily correlated with each other.

Specific Gravity.—The result of recent observations in Germany, France, and Britain, shows that any considerable change in the specific gravity of the cerebral substance is incompatible with a healthy exercise of the nervous functions. To Dr. John Charles Bucknill, Physician to the Devon County Lunatic Asylum, medical science is indebted for the first most extended account of the specific gravity of the cerebral substance, and its relation to disease, and more especially to *atrophy* and *paralysis*. The following are the general results of his observations, for the most part made upon patients labouring under different forms of mental disease:—(1.) Average specific gravity of healthy brain, 1·036. (2.) In paralysis of a chronic character, complicated with insanity, the specific gravity ranged betweeen 1·036 to 1·046. (3.) In some acute cases the specific gravity was as high as 1·052. (4.) In paralysis terminating by coma, 1·040. (5.) In paralysis terminating by syncope or asthenia, 1·036 to 1·039. (6.) In general terms, a higher specific gravity was found when life terminated by coma or asphyxia, than when it ended by syncope or asthenia (*The Lancet*, 25th December, 1852). In addition to these observations, an able and elaborate paper has since been published by Dr. Sankey, showing the relative specific gravity of the *grey* and *white matter* of the brain, and of so extensive a nature as to furnish very copious data for comparing morbid states with the standard of health. The following are the general results of his researches:—(1.) Mean specific gravity of the grey substance of the brain in either sex, 1·034. (2.) In the earlier and later periods of life the specific gravity of the grey matter is below the mean. (3.) The cerebral substance acquires its greatest density in males between the ages of fifteen and thirty, and in females between the ages of twenty and thirty. (4.) The density diminishes with prolonged illness. (5.) It decreases with a lapse of time after death in the ratio of ·001 for every twenty-four hours. (6.) A density of ·006 above the average indicates the existence of the following conditions during life:—Acute cerebral symptoms, or chronic disease with no cerebral symptoms, or only slight delirium; also with conditions associated with hyperæmia. (7.) Mean specific gravity of white matter, 1·041 (*The British and Foreign Medico-Chirurgical Review*, for January, 1853, p. 257).

Both sets of observations referred to above have been made upon the brain as a whole; and as the observations of Dr. Sankey show that no constant relation exists between the absolute weight of the brain and its specific gravity, it is necessary to examine the brain as we do its anatomy—namely, by comparative observations on its *central parts* or *ganglia*. At the time Dr. Bucknill published his observations I was engaged in determining the specific gravity of the central parts of the brain, which are sometimes called the *central ganglia*, and which are now generally regarded as the parts more immediately related to the combined exercise of sensory and motor functions. These centres consist of the *corpora striata; thalami optici; tubercula quadrigemina; and the large mass of grey nervous matter associated with the convolutions of the hemispheres and the substance of the cerebellum.* I found these parts to have a specific gravity as follows:—The central ganglia, 1·040 to 1·047; the cerebrum, 1·030 to 1·048; the cerebellum, 1·038 to 1·049. The same kind of morbid states which modified the specific gravity of the brain substance, as recorded by Drs. Bucknill and Sankey, also manifest their influence on the central parts. Thus death by *coma*, especially in *typhus fever*, was indicated by an extremely high specific gravity; and while it was observed that a slight difference was common in most cases when similar parts on opposite sides were compared, it is presumed that further observation extended in this direction, especially in cases of hemiplegia, may lead to important results. In one case of *choreic hemiplegia* which I had an opportunity of carefully investigating, the specific gravity of the *corpus striatum* and *optic thalamus* on the right side was found to be 1·025, while the specific gravity of the corresponding parts on the left side was observed to be 1·031 (*Glasgow Med. Journal*, No. 1, 1853).

The bulk of the encephalon is found to vary from 65 to 84 cubic inches. The average male brain of 49·5 oz. avoir., at an average specific gravity of 1·040, would give a bulk of 82·5 cubic inches of brain substance; and if an addition of 10 per cent. for loss of membrane and fluid be made, the cranial capacity for such a brain may be calculated at about 90 cubic inches; and conversely, the bulk and weight of the brain may be calculated from the known cranial capacity. The weight of Cuvier's brain at 65 oz. avoir., at a specific gravity of 1·040, would give a brain bulk of 108 cubic inches, and a cranial capacity of 118 cubic inches; while, on the other hand, it is found that when the brain has fallen to 32 oz. (the lowest weight probably compatible with healthy action) it corresponds to a brain bulk of about 53 cubic inches, and a cranial capacity of about 63 cubic inches (Dr. ALLEN THOMSON).

4. THE MASS OF NERVOUS SUBSTANCE WITHIN THE CRANIUM, CALLED THE ENCEPHALON, is anatomically divided into the following parts, namely—(1.) The *medulla oblongata*, continuous with the spinal cord. It rests on the basilar process of the occipital bone; and on its superior or dorsal surface it presents a groove continuous with the central canal of

the spinal cord. (2.) The *cerebellum* lies above and immediately behind the medulla oblongata, with which it is directly continuous. It occupies the posterior fossa of the cranium. By the middle part of its anterior and inferior surface it forms the roof of a space, the floor of which is the grooved posterior surface of the medulla oblongata, and which is named the fourth ventricle of the brain. On each side of this the cerebellum is connected with the medulla oblongata and cerebrum, and also receives the fibres of the pons Varolii. (3.) The *pons Varolii* is a commissure uniting the two hemispheres of the cerebellum, and passing beneath and between the fibres which extend upwards from the medulla oblongata. (4.) The *cerebrum* includes all the remaining and much the largest part of the encephalon. It is united with the parts below by a comparatively narrow and constricted portion or *isthmus*, part of which, forming the *crura cerebri*, descends into the pons Varolii, and through it is continued into the medulla oblongata; whilst another part joins the cerebellum. Situated on the fibres which extend up from the constricted part are a series of eminences (named from behind forwards)—*the corpora quadrigemina, optic thalami*, and *corpora striata;* and springing from the front and outer side of the *corpora striata* are the large convoluted cerebral hemispheres, which expand from this place in all directions, concealing the eminences named, and occupying the entire vault of the cranium, the anterior and middle cranial fossæ, and the superior fossæ, of the occipital bone.

For pathological and physiological purposes it is first of all necessary that the lobes, sulci, and convolutions of the cerebrum should be studied in relation to the outer surface of the skull and head, as expounded by Professor Turner (*Journal of Anatomy and Physiology*, VIII., pp. 142, 359; and *Introduction to Human Anatomy*, p. 293, Edinburgh, 1875). The outer surface of the skull is thus mapped out into regions, just as the thorax and abdomen are conveniently mapped out into regions named in relation to the parts they inclose. The determination of the exact relations of the parts within the skull is "of importance to the physician and the surgeon as a guide to the localisation and estimation of the effects of diseases and injuries of the brain and its coverings." In conducting such an investigation it is necessary to have a clear conception of certain well-defined landmarks, which can be seen or felt when the outer surface of the skull and head are examined. These are,—(1.) The external occipital protuberance (Fig. 1, *o*); (2.) the parietal (P) and frontal (F) eminences; and, (3.) the external angular process of the frontal bone (A). These are easily recognised parts, the position of which can be determined by manipulating the skull, and still more readily on the surface of the skull itself. (4.) The coronal (*c*) and lambdoidal (*l*) sutures can also be felt through the scalp in most heads; and on the skull itself the position of the squamous (*s*), squamoso-sphenoid (*ss*), and parieto-sphenoid (*ps*) fissures, and the curved line of the temporal ridge (*t*) can also, without difficulty, be determined. By the aid of these landmarks each lateral half of the surface of the skull or scalp may be subdivided and mapped out into ten well-defined regions or areas, within which the fissures and convolutions on the outer surface of the hemisphere may be localised. The line of the coronal suture (Fig. 1, *c*) forms on each side the posterior boundary of the *præcoronal* or *frontal area.* An imaginary vertical line (Fig. 1, 2) drawn from the squamous suture (*s*) upwards through the parietal eminence (P) to the sagittal suture or middle line of the skull, lies almost parallel to the coronal suture, and subdivides the parietal region into a *post-coronal* or *antero-parietal* (Fig. 1, SAP + IAP), and a *prælambdoidal* or *post-parietal area* (Fig 1, SPP + IPP). The squamous part of the occipital bone between the lambdoidal (*l*) suture and the occipital protuberance (*o*) and superior curve line, forms the post-lambdoidal or occipital area (Fig. 1, O). These regions may be again subdivided into smaller areas; thus the temporal ridge (*t*), starting from the external frontal process (A), comes backwards across the frontal (præcoronal), antero-parietal (post-coronal), and post-parietal (prælambdoidal) regions to the lateral angle of the occipital bone, and subdivides each of these regions into an upper and a lower area. The lower areas may be termed the *inferior frontal* (IF) or *fronto-temporal,* the *lower antero-parietal* (IAP), and the *lower postero-parietal* (IPP). The inferior frontal, or *fronto-temporal area* (IF), is bounded above by the temporal ridge, behind by the coronal suture, and below by the fronto-sphenoidal suture. The inferior *antero-parietal area* (IAP) is bounded above by the temporal ridge, in front by the coronal suture, below by the squamous and sphenoido-parietal sutures, and behind

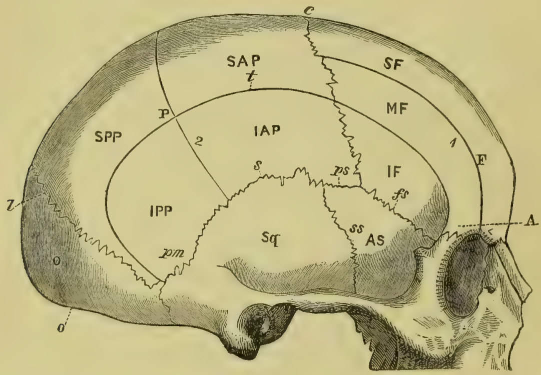

Fig. 1.[*]

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[*] LATERAL VIEW OF THE HUMAN SKULL (after TURNER).—A. The external angular process of the frontal bone; F. The frontal eminence; P. The parietal eminence; *o.* The occipital protuberance; *c.* The coronal suture; *l.* The lambdoidal suture; *s.* The squamous suture; *t.* The temporal ridge; *fs.* The fronto-sphenoid suture; *ps.* The parieto-sphenoid suture; *ss.* The squamoso-sphenoid suture; *pm.* The parieto-mastoid suture; 1. Frontal line; 2. Parietal line; SF, MF, IF. The supero-, mid-, and infero-frontal subdivisions of the frontal area; SAP. The supero-antero-parietal area; IAP. The infero-antero-parietal area; SPP. The supero-postero-parietal area; IPP. The infero-postero-parietal area; O. The occipital area; Sq. The squamoso-temporal area; AS. The ali-sphenoid area.

by the vertical-parietal line through the parietal eminence (P), which divides the parietal region into an anterior and a posterior area. The inferior *postero-parietal area* (IPP) is bounded above and behind by the temporal ridge, in front by the above-named vertical-parietal line, and below by the posterior part of the squamous suture and by the parieto-mastoid suture. The *upper frontal area*, or upper area of the præcoronal region, includes all the frontal regions above the temporal ridge, and may be subdivided by an antero-posterior line (Fig. 1, 1), drawn backwards and upwards parallel to the frontal suture from the superior orbital border, through the frontal eminence (P) to the coronal suture. This divides the upper frontal area into a *supero-frontal* (SF), a *mid-frontal* (MF), and an *inferior frontal* (IF). The upper areas of the post-coronal and præ-lambdoidal regions consist of the parietal bone, above the temporal ridge, and are bounded below by that ridge, above by the sagittal suture, and are separated from each other by the vertical-parietal line already indicated. These may be conveniently named the *upper antero-parietal* (SAP), and the upper *postero-parietal areas* (SPP). Eight areas may thus be defined on each side of the head, situated beneath that portion of the scalp which is so thin as to allow their extent and boundaries to be ascertained, on external manipulation, with a fair amount of precision. The ninth and tenth are more difficult to define, on account of this region of the skull being concealed by the temporal muscle. These areas are situated below the squamoso-parietal, sphenoido-parietal, and fronto-sphenoidal sutures. The lines of the sutures naturally divide this region into a *squamoso-temporal* (Sq) and an *ali-sphenoidal* area (AS). Within these different areas the position of the fissures and convolutions in relation to each other may now be described.

5. THE CEREBRUM.—(*a.*) *Its External Configuration.*—The *cerebrum* (or brain proper) constitutes the highest and much the largest portion of the encephalon, and its external configuration in man is remarkably characteristic. It is made up of two hemispheres united together across the mesial plane by a principal and smaller commissure; by means of which there is inclosed a cavity, which is subdivided into various ventricles, namely, the two lateral, the third and the fifth. (QUAIN, l. c., p. 502.)

The surface of these hemispheres presents a more or less folded appearance, technically called the convolutions or gyri; which are not, as was at one time supposed, irregular and inconstant in their disposition, but possess (as was first systematically described by Gratiolet) definite modes of arrangement. Hence, (1.) Convolutions; (2.) Lobes; and, (3.) Fissures, require careful study in relation to each other, to the skull, and to other surroundings. The human brain can be thus subdivided into five lobes by certain *fissures* seen on the surface of the hemispheres, which lobes have been called *temporo-sphenoidal, occipital, frontal, parietal,* and the *insula* or *central lobe*, or *island of Reil.* Three of these are in contact with the bones after which they are named, though their limits do not correspond with them. The convolutions of the temporo-sphenoidal lobe are arranged in three parallel tiers, from above downwards, and are separated from the fronto-parietal lobes by the well-marked Sylvian fissure. The occipital lobe, small in size, forms the hinder end of the cerebrum, and also possesses, from above downwards, three parallel tiers of convolutions. It is separated from the parietal lobe by the parieto-occipital fissure, which fissure, on the outer surface of the hemisphere at least, is bridged across by two convolutions, named the first and second bridging or annectent gyri (*plis de passage* of Gratiolet).

The great fronto-parietal lobes are subdivided into frontal and parietal portions by a fissure which runs obliquely upwards and backwards over the outer face of the hemisphere, and is named the fissure of Rolando. (Fig. 2, R, and Fig. 3, *c*.) The frontal lobe consists of a large convolution, which ascends immediately in front of the fissure of Rolando (Fig. 2, AP); of three tiers of convolutions, named superior, middle, and inferior frontal convolutions, extending from the anterior end of the cerebrum backwards, and parallel to each other, to join the ascending frontal convolution (Fig. 2, SF, MF, IF); and of some small convolutions on its orbital surface (see Fig. 5, p. 23). The parietal lobe consists of a large convolution,

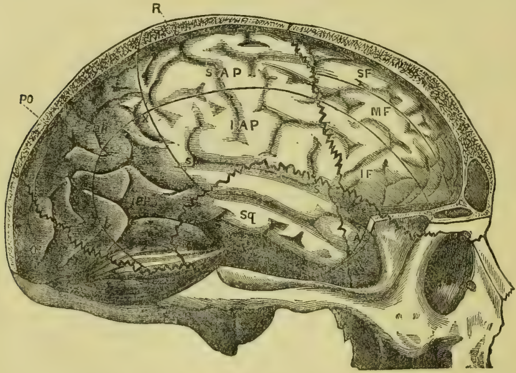

Fig. 2.*

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* Diagram showing the Relations of the Convolutions to the Skull (after Turner).—R. The fissure of Rolando, which separates the frontal from the parietal lobe; PO. The parieto-occipital fissure, between the parietal and occipital lobes; SS. The fissure of Sylvius, which separates the temporo-sphenoidal from the frontal and parietal lobes; SF, MF, IF. The supero-, mid-, and infero-frontal subdivisions of the frontal area of the skull: the letters are placed on the superior, middle, and inferior frontal convolutions; SAP. The supero-antero-parietal area of the skull: S is placed on the ascending parietal convolution, AP on the ascending frontal convolution; IAP. The infero-antero-parietal area of the skull: I is placed on the ascending parietal, AP on the ascending frontal convolution; SPP. The supero-postero-parietal area of the skull: the letters are placed on the angular convolution; IPP. The infero-postero-parietal area of the skull: the letters are placed on the mid-temporo-sphenoidal convolution; X. The convolution of the parietal eminence, or supra-marginal gyrus; O. The occipital area of the skull: the letter is placed on the mid-occipital convolution; Sq. The squamoso-temporal region of the skull: the letters are placed on the mid-temporo-sphenoidal convolution; AS. The ali-sphenoid region of the skull: the letters are placed on the top of the supero-temporo-sphenoidal convolution.

which ascends immediately behind the fissure of Rolando (Fig. 2, S and I), and of three convolutions, named postero-parietal lobule, supra-marginal convolution, and angular convolution, which pass backwards to the parieto-occipital fissure and the posterior extremity of the fissure of Sylvius. The insula, or island of Reil, is composed of from five to seven short gyri, which radiate outwards and backwards from the locus perforatus anticus, and lie concealed within the Sylvian fissure. On the inner face of the hemisphere also a well-defined and long-recognised convolution, the gyrus fornicatus, is seen winding round the corpus callosum (Fig. 6); to its

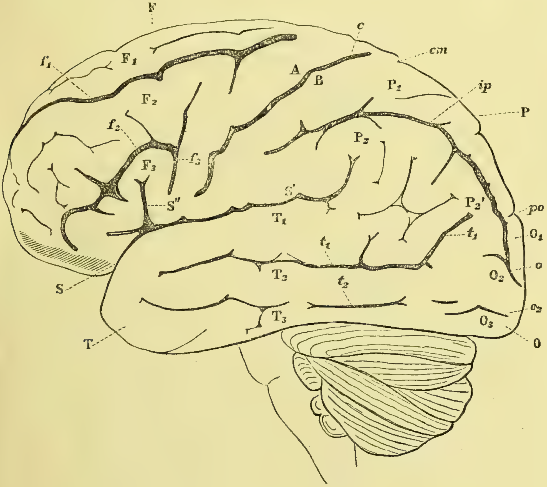

Fig. 3.*

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* MAP OR PLAN IN OUTLINE OF THE ENCEPHALON, looking upon its *left latera* surface (after ECKER). F. Frontal lobe; P. Parietal lobe; O. Occipital lobe; T. Temporo-sphenoidal lobe; S. Fissure of Sylvius; S'. Horizontal, S''. Ascending ramus of the same; *c*. Sulcus centralis, or fissure of Rolando; A. Anterior central convolution (*Antero-parietal gyrus* of Huxley, *Ascending frontal gyrus* of Turner); B. Posterior central convolution (*Postero-parietal gyrus* of Huxley, *Ascending parietal convolution* of Turner); F_1. Superior, F_2. Middle, F_3. Inferior frontal convolution; f_1. Superior, and f_2. Inferior frontal sulcus; f_3. Sulcus præcentralis; P_1. Superior parietal lobule (the *Postero-parietal lobule* of Huxley, Turner, and other English authors); P_2. Inferior parietal lobule—viz., P_2. Gyrus supra-marginalis, and P_2'. Gyrus angularis; *ip*. Sulcus interparietalis (the *Intraparietal fissure* of Turner); *cm*. Termination of the calloso-marginal fissure; O_1. First, O_2. Second, O_3. Third occipital convolutions; *po*. Parieto-occipital fissure; *o*. Sulcus occipitalis transversus; o_2. Sulcus occipitalis longitudinalis inferior; T_1. First, T_2. Second, and T_3. Third temporo-sphenoidal convolutions; t_1. First, t_2. Second temporo-sphenoidal fissures.

posterior and lower end the name of gyrus hippocampi is now not unfrequently applied. Other less extensive, but at the same time well defined, convolutions may also be seen on this inner surface, to be afterwards noticed.

In all brains certain primary fissures (besides the deep *median longitudinal fissure* which divides the two hemispheres) can be recognised on each side, on which the division into *lobes* has been founded. These *interlobar* fissures are three in number, namely—(1.) *The fissure of Sylvius* (Fig. 2, SS, and Fig. 3, S) commences on the under surface of the brain, close to the anterior perforated spot, immediately behind the posterior border of the lesser wing of the sphenoid bone. Thence it passes

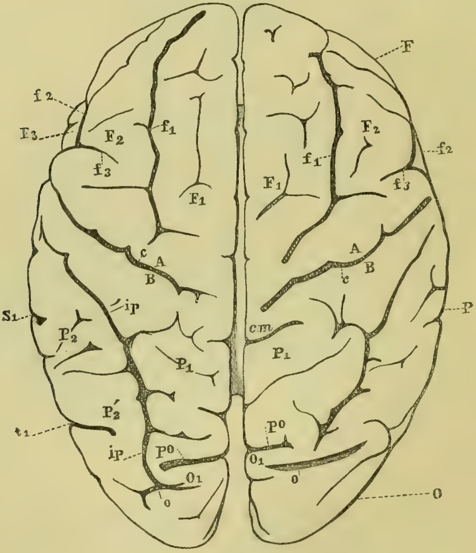

Fig. 4.*

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* Map or Plan in Outline of the Surface of the Brain, seen from above (after Ecker). F. Frontal lobe; P. Parietal lobe; O. Occipital lobe; S_1. End of the horizontal ramus of the fissure of Sylvius; *c*. Central fissure, or fissure of Rolando; A. Anterior, B. Posterior central convolutions (other names, see Fig 3); F_1. Superior, F_2. Middle, F_3. Inferior frontal convolutions; f_1. Superior, f_2. Inferior frontal sulcus; f_3. Sulcus præcentralis; P_1. Superior parietal lobule; P_2. Inferior parietal lobule—viz., P_2. Gyrus supra-marginalis, and P'_2. Gyrus angularis; *ip*. Interparietal fissure; *cm*. Calloso-marginal fissure; P*o*. Parieto-occipital fissure; t_1. Superior temporo-sphenoidal fissure; O_1. First occipital convolution; *o*. Sulcus occipitalis transversus.

transversely outwards to the lateral surface of the hemisphere, where it divides into a short anterior ascending limb, and a larger posterior horizontal limb (Fig. 3, S′), which passes backwards, ascending slightly through the middle third of the hemisphere, and separating the parietal lobe above from the temporo-sphenoidal lobe below it. The anterior or ascending limb (Fig. 3, S″), about an inch in length, runs upwards and forwards into the frontal lobe, the lowest convolution of which curves round it. In its course upwards and backwards the *Sylvian fissure* is covered by the great wing of the sphenoid bone, where it articulates with the anterior inferior angle of the parietal. It then passes obliquely under cover of the anterior superior part of the squamous plate of the temporal, and appears in the lower part of the antero-parietal region, through which either it, or one of the small branches into which it not unfrequently divides, may be continued into the lower postero-parietal region. Within this *Sylvian fissure* is the isolated group of convolutions of the "*Island of Reil,*" the "*Insula*" or "*Central lobe*" (QUAIN and TURNER, l. c.). (2.) The *fissure of Rolando* (Fig. 2, R, Figs. 3 and 4, *c*), or *central sulcus,* extends across the lateral convex surface of the hemisphere. It commences above, behind the vertex, near the great longitudinal fissure, and passes downwards and forwards, to end close behind (but above) the bifurcation of the fissure of Sylvius, into the posterior limb of which it has been very rarely seen to open. Its position and direction are such that the fissures of the two sides, seen from above, form a V-shaped line, open in front. It is rarely interrupted in its course, and is very uniform in man and most of the primates. It appears about the fifth month of fœtal life. It lies in the *post-coronal region,* through which it passes from below obliquely upwards and backwards, so that it traverses both its upper and lower subdivisions. Its distance from the coronal suture varies in different brains, and with the degree of development of the frontal lobe. Professor Turner has seen its upper end as much as 2 inches behind the top of the suture, its lower end an inch and a half behind the inferior part of the suture, and both ends not more than 1·5 and 1·3 inch posterior to this suture. It separates the frontal from the parietal lobe. The parallel convolutions which it separates are named respectively the *ascending* or *tranverse frontal* (Fig. 2, AP, Figs. 3 and 4, A), and the *ascending parietal* convolutions (Fig. 2, I, Figs. 3 and 4, B) (QUAIN and TURNER, l. c.) (3.) The *parieto-occipital fissure* (Figs. 2, PO; 3, *po;* and 4, P*o*), or *perpendicular fissure,* separates the occipital from the parietal lobe. It is best marked on the median surface of the hemisphere, as a deep cleft, extending downwards and a little forwards from the margin of this surface to near the posterior extremity of the corpus callosum. On the convex surface it is continued transversely outwards for a variable distance, generally about an inch, as the *external parieto-occipital fissure.* The size of its external portion depends (inversely) on the size of the convolution which curves round its outer extremity, and connects the parietal with the occipital lobe. It appears about the fourth or fifth month of fœtal life. It lies in the *upper area* of the *prœlambdoidal* region, close to its sagittal border. Its exact distance from the apex of the lambdoidal suture varies, partly from variations in the brain itself, and partly from the not unfrequent variations in the mode of ossification of the upper squamous part of the occipital bone.

About 0·7 or 0·8 of an inch will express its average distance from the apex of that suture.

By these several fissures the outer surface of the hemispheres is divided into the following lobes, which do not precisely correspond to the areas of the cranial bones (Turner) :—(1.) The *frontal lobe* (Figs. 2 and 3) is the anterior portion of the brain in front of the *Sylvian fissure* at the base, and of the *fissure of Rolando* on the outer side. On the median surface there is no corresponding demarcation. The inferior surface of the frontal lobe, which is in contact with the orbital plate, is called the *orbital surface* (Fig. 5); the upper arched aspect is called the *frontal surface.* On this latter surface the convolutions are four in number, three antero-posterior, in parallel rows one above the other, and one transverse behind them—the *ascending or transverse frontal convolution* (Figs. 3 and 4, A), in front of the fissure of Rolando, which it thus bounds. Below it commences just above and behind the bifurcation of the Sylvian fissure, and thence curves upwards and backwards to the margin of the great longitudinal fissure. Usually above, and almost invariably below, it is connected with the ascending parietal convolution (Figs. 3 and 4, B) behind the fissure of Rolando. That fissure is thus isolated. Anterior to this convolution the frontal surface is occupied by convolutions which run more or less in an antero-posterior direction—three in number, an upper, middle, and lower—which may or may not arise superficially from the ascending frontal convolution; and secondary convolutions usually connect them in their course. The *superior*, or *first frontal* convolution (Fig. 2, SF, and Figs. 3 and 4, F_1), lies at the margin of the great longitudinal fissure. It usually commences superficially at the upper end of the ascending frontal convolution, and extends to the anterior extremity of the hemisphere, where, tapering, it passes over to the orbital surface. Its inner aspect appears on the median surface of the hemisphere. The *middle*, or second *frontal convolution* (Fig. 2, MF, and Figs. 3 and 4, F_2), usually broad and much subdivided, arises deeply or superficially from the ascending frontal below the last, and runs thence to the anterior extremity of the hemisphere. The *inferior*, or *third frontal convolution* (Fig. 2, IF, and Figs. 3 and 4, F_3), forms the lower and outer portion of the frontal lobe. It arises from the lower extremity of the ascending convolution, just above the bifurcation of the fissure of Sylvius, arches round the ascending limb of that fissure, and extends to the anterior extremity of the hemisphere.

The *fissures* of the frontal surface are,—(1.) The *transverse frontal fissure* (Figs. 3 and 4, f_3), or "*præcentral sulcus,*" which lies in front of the ascending frontal convolution, and parallel to the fissure of Rolando. If the origins of the superior, middle, and inferior frontal convolutions arise superficially, the fissure is interrupted, and may be inconspicuous; and when the inferior frontal convolution arises deeply, this fissure is continuous with the Sylvian fissure, of which it has, in consequence, been regarded as the ascending limb. (2.) Two *antero-posterior fissures*—*superior* and *inferior frontal* (Figs. 3 and 4, f_1 and f_2)—separate the corresponding convolutions from the middle frontal. They are often very irregular, from bridging over by secondary convolutions.

The orbital surface of the frontal lobe (Fig. 5) presents two *fissures:*—(1.) The *olfactory sulcus* (Fig. 5, O), which is straight, parallel with the great longitudinal fissure, and lodges the olfactory bulbs; (2.) the *orbital sulcus* (Fig. 5, TR), which is irregular, often triradiate, and lying in the centre of the lobe. Its *convolutions* are,—(1.) The *straight convolution* (Fig. 5, 17), which is continuous at its anterior extremity with the superior frontal; (2.) an *inner*, an *anterior*, and an *outer* or *posterior orbital* convolutions are also described as lying around the orbital sulcus.

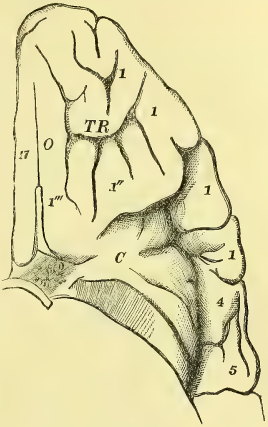

Fig. 5.*

(2.) The *parietal lobe* (Figs. 2, 3, 4) lies behind the frontal, and in front of the occipital lobe. The *temporo-sphenoidal* lobe is below it. In front it is bounded by the fissure of Rolando; behind, by the parieto-occipital fissure and its arbitrary continuation; internally, by the great longitudinal fissure; and externally, by the posterior limb of the fissure of Sylvius, as far as this preserves its horizontal direction and its arbitrary continuation in that direction. The *intraparietal fissure* arches through the parietal lobe, commencing in its anterior inferior angle, where it is sometimes, though rarely, continuous with the fissure of Sylvius. It divides the parietal lobe into two portions—superior and inferior parietal lobules; and is often bridged across by a secondary convolution connecting these lobules. The *convolutions* of the parietal lobe are,—(1.) The *ascending parietal* (Fig. 2, SI, and Figs. 3 and 4, B), which lies behind the fissure of Rolando, and parallel to the ascending frontal convolution, with which it is usually continuous both above and below. Above, it is continuous with the superior parietal lobule (Fig. 3, P_1). (2.) A narrow convolution—the *first connecting* or *annectent convolution*—usually connects the superior parietal lobule with the occipital lobe, outside the extremity of the external parieto-occipital fissure. Two convolutions exist in the inferior parietal lobule,—(3.) A *supra-marginal gyrus* or *convolution* (Fig. 3, P_2), which lies behind the lower end of the intraparietal fissure, beneath which it is usually continuous with the ascending parietal convolution, and passes into, (4.) the *angular gyrus* or *convolution* (Fig. 3 and 4, P'_2), above the extremity of the Sylvian fissure. This convolution is bounded in front by the terminal ascending portion of the fissure of Sylvius; above, by the intraparietal fissure; below it is continuous with the superior (and sometimes with the middle) temporo-sphenoidal convolution; and behind by the occipital lobe, by means of one or two (*second* and *third*) annectent convolutions (QUAIN, l.c.)

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* THE ORBITAL SURFACE OF THE LEFT FRONTAL LOBE AND THE ISLAND OF REIL; the tip of the temporo-sphenoidal lobe has been removed to display the latter (after TURNER). 17, Convolution of the margin of the longitudinal fissure; O. Olfactory fissure, over which the olfactory peduncle and lobe are situated; TR. Triradiate fissure; 1'', 1''', Convolution on the orbital surface; 1, 1, 1, 1, Under surface of infero-frontal convolution; 4, Under surface of ascending frontal, and 5, of ascending parietal convolutions; C. Central lobe or insula.

As regards the contents of these areas in relation to the skull and scalp (Figs. 1 and 2), and taking the fissure of Rolando as the line of demarcation between the frontal and parietal lobes, extending obliquely upwards and backwards in the *post-coronal* region, the great central convolutions, which bound it in front and behind (the ascending frontal and ascending parietal), necessarily occupy a considerable share of the two subdivisions. The *frontal area* is entirely occupied by the frontal lobe, though it does not cover the whole of what is included under the term, inasmuch as the posterior extremities of the three longitudinal frontal convolutions and ascending frontal convolutions lie in the anterior parietal area. The contents of the frontal area are as follow:—(1.) In the *lower antero-parietal area* (Fig. 2, IAP) are to be found the lower third of the ascending frontal convolution (on which the letters AP rest), from which the inferior frontal convolution arises somewhat less than an inch behind the lower end of the coronal suture; also, the lower third of the ascending parietal convolution (on which the letter I of IAP rests), behind which, but separated from it by the intra-parietal fissure, lies a small part of the supra-marginal gyrus or convolution. The relations of the parietal eminence to the surface of the hemisphere are so constant that, in the specimens examined by Professor Turner, the hollow of the bone corresponding to the cerebral surface of this eminence was always occupied by the supra-marginal convolution, hence it may be appropriately named the *convolution of the parietal eminence* (Fig. 2, X). Below the ascending convolutions a short segment of the Sylvian fissure extends; and quite at the lower and posterior boundary of the area a little bit of the superior temporo-sphenoidal convolution appears above the squamous suture. The lower frontal convolution arises in this area from the ascending parietal, somewhat less than an inch behind the lower extremity of the coronal suture; and the posterior extremity of this convolution (Broca's region) lies in this area. (2.) In the *upper antero-parietal area* (Fig. 2, SAP) are to be found the upper two-thirds of the ascending frontal (AP) and parietal (S) gyri, which extend close up to the sagittal suture and the origins of the superior frontal and middle convolutions. From the ascending frontal arises the superior and middle frontal gyri; the former arises 1·2 or 1·3 of an inch behind the coronal suture, the latter about an inch behind the same line. Immediately behind the upper end of the ascending parietal gyrus, in the neighbourhood of the sagittal suture, a little bit of the postero-parietal lobule may or may not be seen; and below this a small portion of the convolution of the parietal eminence (*supra-marginal convolution*) may appear. The contents of the *prælambdoidal region* are,—(1.) *In the lower postero-parietal* area (Fig. 2, IPP) lies the hinder part of the convolution of the parietal eminence (or supra-marginal gyrus), behind which is part of the angular gyrus, and below this the posterior ends of the three convolutions of the temporo-sphenoidal lobe. (2.) *In the upper postero-parietal area* (Fig. 2, SPP), in the region close to the sagittal suture up to the parieto-occipital fissure, lies the postero-parietal lobule; below it lies the upper portion of the angular gyrus (SPP), and a part of the convolution of the parietal eminence, or supra-marginal gyrus (X). In the more posterior part of the region the annectent gyri blend with the three convolutions of the occipital lobe.

Thus the *præcoronal region* is entirely occupied by the *frontal lobe.* In the *inferior* or *temporo-frontal area* is situated a large portion of the inferior frontal convolution, but a small bit of the middle gyrus may be seen at its posterior superior angle. The *middle frontal area* corresponds almost exactly to the middle frontal gyrus, and the *superior frontal area* to the superior gyrus. The *occipital area* (Fig. 2, O) indicates the situation of the occipital lobe, though it does not entirely cover it, inasmuch as part of the occipital lobe extends anteriorly beyond the lambdoidal suture into the postero-parietal area. The *squamoso-temporal area* (Fig. 2, Sq) contains the greater portion of the temporo-sphenoidal convolution; but the superior temporo-sphenoidal convolution, though for the most part under cover of the squamoso-temporal and greater wing of the sphenoid, ascends into both antero- and lower postero-parietal areas. The *ali-sphenoid* area (Fig. 2, AS) contains the lower or anterior extremity of the temporo-sphenoidal lobe. The *central lobe*, or *island* of Reil, does not come to the surface, but lies concealed within the fissure of Sylvius. It is situated behind the upper part of the great wing of the sphenoid, and opposite its line of articulation with the anterior inferior angle of the parietal bone and squamous portion of the temporal. The deep-seated position and direction of the hippocampal region are superficially indicated by the convolutions of the temporo-sphenoidal lobe, contained chiefly in the inferior postero-parietal, squamoso-temporal, and ali-sphenoid regions (TURNER, FERRIER).

Convolutions also exist on the inner and on the under surfaces of the hemispheres, resting on the tentorium. They are to be studied in connection with the *corpus callosum,* or great transverse commissure, which connects the two hemispheres; and with certain fissures situated on these surfaces. The convolution which immediately bounds the corpus callosum (Fig. 6, CC) is termed the *gyrus fornicatus* (G*f*), or arch-shaped gyrus—thus inclosing the corpus callosum within the concavity of its arch, and hence sometimes also called the callosal convolution. It commences at the frontal extremity of the brain, beneath the anterior extremity or *genu* of the corpus callosum, and terminates posteriorly in the *gyrus uncinatus* (uncinate gyrus), or gyrus hippocampi (Fig. 6, H), which ends anteriorly in a crooked-like extremity or crotchet—the *uncus gyri fornicati,* or *subiculum cornu Ammonis* (Figs. 6 and 7, U). Above the gyrus fornicatus, and separated from it by a fissure termed the *calloso-marginal fissure* (Fig. 6, *cm*), lies the *marginal convolution* (Fig. 6, F_1), which forms part of the internal margin of the great longitudinal fissure. It extends forwards from the parieto-occipital fissure to the anterior end of the hemisphere, where it turns back to the *locus perforatus anticus.* It is merely the mesial or internal aspect of the convolutions of the frontal and parietal lobes. Between the posterior extremity of the calloso-marginal fissure and the parieto-occipital fissure (Figs. 6 and 7, *po*) is a lobule of a quadrilateral form—the *quadrilateral lobule* or *præcuneus* (Figs. 6 and 7, Pi), which is the mesial aspect of the postero-parietal lobule; and, inferiorly it is continuous with the gyrus fornicatus. The parieto-occipital fissure leads downwards, and terminates by an acute angle in an offset termed the *calcarine fissure* (Figs. 6 and 7, *oc*), which passes almost horizontally backwards in the occipital lobe, and terminates in a *superior* and *inferior* ramus (Figs. 6 and 7, *oc′* and *oc″*.) It marks the position internally of the *calcar avis* or *hippocampus minor*, in the posterior cornu of the lateral ventricle. The calcarine fissure is *not* continuous anteriorly in the *dentate fissure* (Fig. 6, *h*), or *sulcus hippocampi*. Hence the gyrus fornicatus and gyrus uncinatus are continuous. The dentate fissure, or sulcus hippocampi, marks the position of the hippocampus major, or cornu Ammonis, in the descending cornu of the lateral ventricle. In this fissure lies the *fascia dentata*, or *dentate gyrus*, which forms a boundary to the grey matter of the hippocampus major. In the angle between the parieto-occipital and calcarine fissures, a wedge-shaped lobule, named the *cuneus* (Fig. 6, Oz), or *internal occipital lobule* of Huxley, is marked out on the mesial aspect of the occipital lobe. Running along the internal or mesial aspect of the occipital and temporo-sphenoidal lobes is the *collateral fissure* of Huxley, or *sulcus occipito-temporalis*. It separates the two convolutions from each other which connect the occipital and temporo-sphenoidal lobes—the *occipito-temporal convolutions* (Figs. 6 and 7, T_4 and T_5); and marks the position of the *collateral eminence* in the interior of the ventricle. The upper of these convolutions is termed the *gyrus occipito-temporalis*

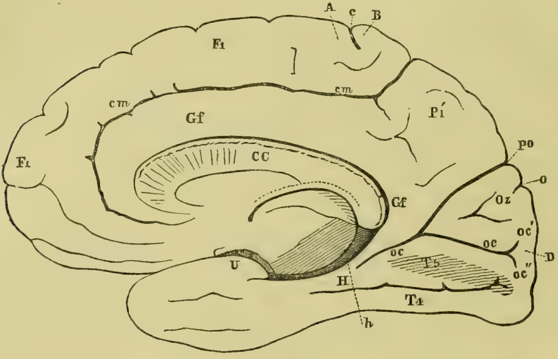

Fig. 6.*

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* Map or Chart in Outline of the Right Hemisphere of the Median Aspect (after Ecker). CC. Corpus callosum longitudinally divided; *Gf.* Gyrus fornicatus; H. Gyrus hippocampi; *h.* Sulcus hippocampi; U. Uncinate gyrus; *cm.* Sulcus calloso-marginalis; F_1. Median aspect of first frontal convolution; *c.* Terminal portion of the sulcus curvatis; A. Anterior, B. Posterior central convolutions; Pí. Præcuneus, Oz. Cuneus, *Po.* Parieto-occipital fissure; *o.* Sulcus occipitalis transversus; *oc.* Calcarine fissure; *oc′.* Superior, *oc″.* Inferior ramus of the same; D. Gyrus descendens; T_4. Gyrus occipito-temporalis lateralis (Lobulus fusiformis); T_5. Gyrus occipito-temporalis medialis (Lobulus lingualis).

medialis, or *lingual lobule* (Fig. 6, T_5); the lower frequently merges with the inferior temporo-sphenoidal convolution, and at other times is marked off by a fissure. It is termed the *gyrus occipito-temporalis lateralis*, or *lobulus fusiformis* (Fig. 6, T_4). Within the lips of the fissure of Sylvius, and concealed by the region included between its ascending and horizontal rami (*operculum*), lies the central lobe, or island of Reil (Fig. 5, C). It covers in the extraventricular nucleus of the corpus striatum; and its surface is marked by certain radiating short convolutions, named the *Gyri breves.*

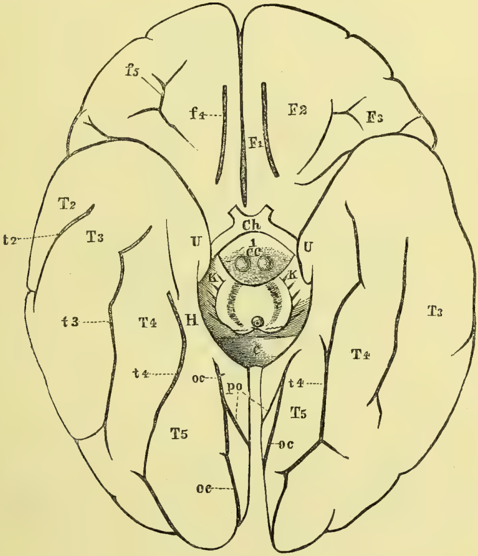

Fig. 7.*

b. Internal Architecture and Minute Structure of the Brain.—The cerebral hemispheres, whose outward conformation has in the preceding paragraphs been topographically laid down in relation to the skull and other parts, form each a sort of hollow shell, cap, or cortex inclosing and overlapping the two great basal ganglia. Of these basal ganglia the posterior pair are called the *optic thalami,* the anterior pair the *corpora striata.* From these ganglia white fibres radiate into the hemispheres of the cerebrum in all directions, so as to form a hollow cone (*corona radiata*). The exact method of distribution of the fibres of the *corona radiata* in the several regions of the cortex, has been variously described, especially by Lockhart Clarke, Meynert, Broadbent, and others. Physiological experiments and clinical observation have also done much to indicate the relations of parts. If a horizontal slice be removed from the upper part of each hemisphere, it will be seen that the cerebrum is composed of *grey* and of *white* matter. The grey matter seen in such a section is peripheral—*the peripheral grey matter of the convolutions,* the various windings of which it closely follows; while the core of each convolution consists of white matter continuous with the mass of white matter in the interior of the hemisphere. If a deeper slice be now made on a level with the plane of the corpus callosum, it will be seen that the white matter of that structure is continuous with the white centre of each basis of the hemisphere (Fig. 8).

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* VIEW OF THE BRAIN FROM BELOW (after ECKER). F_1. Gyrus rectus; F_2. Middle, F_3. Inferior frontal convolutions; f_4. Sulcus olfactorius; f_5. Sulcus orbitalis; T_2. Second or middle, T_3. Third or infero-temporo-sphenoidal convolution; T_4. Gyrus occipito-temporalis lateralis (Lobulus fusiformis); T_5. Gyrus occipito-temporalis medialis (Lobulus lingualis); t_4. Sulcus occipito-temporalis inferior; t_3. Inferior, t_2. Middle temporo-sphenoidal fissure; *po*. Parieto-occipital fissure; *oc*. Calcarine fissure; H. Gyrus hippocampi; U. Gyrus uncinatus; C*h*. Optic chiasma; *cc*. Corpora cortica (albicantia); KK. Crura cerebri; C. Corpus callosum.

The *corpus callosum* consists of bundles of nerve fibres, almost the whole of which pass transversely across the mesial plane between the two hemispheres. These fibres may be traced into the white cores and grey matter (Fig. 8, *a*) of the convolutions; and they are seen to connect the corresponding convolutions and cortex in the opposite hemispheres. Hence the corpus callosum is a connecting or commissural structure, which brings the convolutions of the two hemispheres into anatomical and physiological relation with each other. This commissural structure must be cut through on each side of the mesial line, in order to open into the large cavity or lateral ventricles in each hemisphere (Fig. 8). By so doing the interior of the hemispheres and the two great basal ganglia are exposed. The cavity or interior of the hemispheres follows the same general course as the curve of the hemisphere itself; and at its inner edge, as it winds round the crus cerebri, its grey matter becomes folded into the interior in a reverse manner to the surface convolutions, giving rise to an internal convolution or prominence in the ventricular cavity (FERRIER). This formation (which has certain peculiarities of structure) is termed the *hippocampus major* (Fig. 8, *l*). The lateral ventricles are subdivided into a *space* or body and three bent prolongations or *cornua* on each side. On the floor of the central space may be seen from before backwards the grey upper surface of the pear-shaped *corpus striatum* (Fig. 8, *b* and *b'*), and (to its inner and posterior part) a small portion of the *optic thalamus* (Fig. 8, *d*), whilst between the two is the curved flat band, the *tænia semicircularis* (Fig. 8, *c*). On the upper surface of the thalamus rests the vascular fringe of the velum interpositum, named *choroid plexus;* immediately internal to which is the free edge of the white *posterior pillar of the fornix.* The anterior end of the corpus striatum projects into the anterior cornu. The hippocampus major follows the curvature of the descending cornu to a nodular end, named the *pes hippocampi,* and on its inner border is the white *tænia hippocampi,* continuous with the white posterior pillar of the fornix. If the tænia be drawn on one side the hippocampal fissure is exposed, at the bottom of which the grey matter of the gyrus hippocampi may be seen to form a well-defined dentated border (*fascia dentata*). The choroid plexus is continuous with the pia mater round the gyrus hippocampi, and enters the descending cornu through the great transverse fissure between the tænia hippocampi and optic thalamus. The lateral ventricles

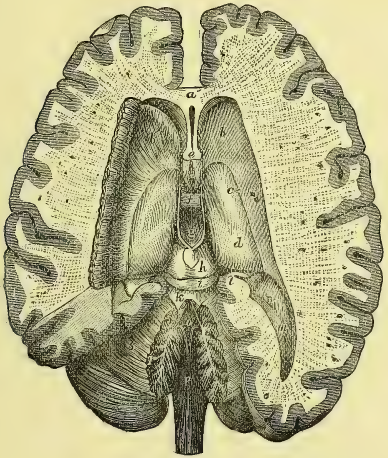

Fig. 8.*

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* Dissection of the Brain from above, exposing the Lateral, the Third and the Fourth Ventricles, with the Surrounding Parts (from Hirschfeld and Leveillé) half size.—*a*. The anterior part or knee of the corpus callosum divided: its fibres are seen spreading on each side into the cerebral hemispheres; *b*. anterior part of the surface of the right corpus striatum in the anterior cornu of the lateral ventricle; *b'*. The same on the left side, in which the grey substance has been dissected so as to show the peduncular medullary fibres spreading through the corpus striatum into the cerebral hemispheres; *c*. Points by a short line to the tænia semicircularis; *d*. Surface of the thalamus opticus; *e*. The anterior pillars of the fornix divided: below they are seen descending in front of the third ventricle, and between them is seen a part of the anterior commissure; above the letter *e* is seen the fifth ventricle, represented as a slit between the two laminæ of the septum lucidum; *f*. is placed on the soft or middle commissure, *g*. In the posterior part of the third ventricle: on either side of this letter is the white striæ or peduncles of the pineal gland; immediately below the latter is the small posterior commissure and the pineal gland; *h*. The upper, and *i*. The lower of the corpora quadrigemina; *k*. Superior peduncle of the cerebellum, and close to this the valve of Vieussens, which is partly divided by a median incision along with the middle lobe of the cerebellum, so as to open up the fourth ventricle; *l*. The hippocampus major and corpus fimbriatum, separated from the posterior pillar of the fornix, and descending into the middle cornu of the lateral ventricle; *m*. The posterior cornu of the lateral ventricle, and hippocampus minor; *n*. Eminentia collateralis; *o*. The cavity of the fourth ventricle; *p*. Posterior surface of the medulla oblongata; *r*. Section of the middle lobe, showing the arbor vitæ; *s*. Upper surface of the cerebellum, brought into view on the left side by the removal of the posterior extremity of the left hemisphere.

are lined by an *ependyma,* which consists of cylindrical epithelium, in many parts ciliated, and which rests on a layer of neuroglia. This lining or ependyma is continuous through the foramen of Munro with that of the third ventricle, which again is continuous with the lining of the fourth ventricle through the aqueduct of Sylvius.

If the corpus callosum is cut across about its middle by a transverse incision, and its posterior half reflected backwards, the body of a structure named the *fornix* is exposed, on which the corpus callosum rests. If the anterior portion of the corpus callosum be reflected forwards, the grey partition, or *septum lucidum,* between the two lateral ventricles, is exposed. It consists of two layers of grey matter, between which is a narrow vertical mesial space—the *fifth ventricle.* On removal of the septum, the anterior part of the fornix is seen. It is an arch-shaped band of fibres, extending in an antero-posterior direction. This system of fibres consists of two lateral halves, one belonging to each hemisphere. The anterior end of the fornix forms the *anterior piers* or pillars of the arch; its *posterior piers* the posterior pillars, whilst the intermediate body of the fornix forms the summit or crown, where the two lateral halves are conjoined. The two anterior pillars descend in front of the third ventricle to the base of the cerebrum, where, by a twist or loop, they form the *corpora albicantia* (Fig. 7, *cc*, and Fig. 11, *a*), after which they radiate into the optic thalamus. Posteriorly the two halves of the fornix diverge much more from each other, and form its posterior pillars, each of which curves away into the descending cornu of the ventricle, and under the name of *tænia hippocampi* forms the free border of the hippocampus major. Thus the *fornix* is a system of fibres which connects the optic thalami and hippocampi. If the body of the fornix be divided by a transverse incision, and the parts separated, the great transverse fissure of the cerebrum is opened into, and the *velum interpositum* is exposed lying in that fissure. It is an expanded fold of the pia mater, continuous with that investing the convolutions, and passes into the interior of the hemispheres through the great transverse fissure. Its lateral margins are fringed by the choroid plexuses, which contain the small choroidal arteries supplying the corpora striata, optic thalami, and corpora quadrigemina—the blood returning by small veins, which join to form the *veins of Galen,* passing along the centre of the velum to open into the straight sinus. The velum interpositum being raised from before backwards, the *optic thalami,* the *third ventricle,* the *pineal gland,* and the corpora quadrigemina (Fig. 8, *h* and *i*) are exposed. Crossing the anterior pillars of the fornix, and passing through the corpora striata, a transverse band of fibres, called the *anterior commissure,* joins the lower temporo-sphenoidal regions with each other; while some of the fibres, forming a loop, directly connect the olfactory bulb tracts.

The *corpus striatum cerebri* (Fig. 8, *b* and *b′*) consists of two masses of grey matter separated from each other by numerous striæ of white fibres, which ascend from below upwards through its substance. The upper mass of grey matter projects into the lateral ventricle as the intraventricular portion, caudate nucleus, or *nucleus caudatus* (Fig. 8). The lower extraventricular portion, or *nucleus lenticularis,* forms the outer and lower part of the corpus striatum, and is separated by the *claustrum* or *tæniform nucleus* (an exceedingly thin plate of grey matter) from the island of Reil. The *cerebral fibres* which enter those nuclei, and occupy the intervals between them, come from all parts of the cerebral surface, but mainly from the anterior half, including the island of Reil and its neighbourhood. The fibres which emerge from them below pass mainly downwards and backwards to form the under superficial or anterior portion or *crusta* of the corresponding cerebral peduncles, within which they become connected with an additional ganglion of the same system—the *locus niger.* The peduncular fibres of the *crura cerebri* are arranged into two groups, named respectively the *crusta* and *tegmentum,* which are separated from each other by the *locus niger.* The fibres of the *crusta* are in greater part continuous with the longitudinal fibres of the pons derived from the anterior pyramids of the medullæ; and receive additional fibres from the grey matter of the locus niger, and from the cells of the Sylvian layer in the corpora quadrigemina. Some of the fibres of the crusta pass directly upwards, as radiating fibres, to the grey cortex of the occipital and temporal lobes; but the larger number terminate in the *nucleus caudatus* and *nucleus lenticularis* of the corpus striatum. From these nuclei a great mass of fibres radiate into the cortex of the fronto-parietal lobes, especially the frontal. A few also pass (*stria cornea*) to the grey matter of the apex of the temporal lobe; and fibres also enter the convolutions of the insula. In addition to the radiating fibres, the grey matter of the corpus striatum gives origin to fibres of the middle root of the olfactory peduncle, and to connecting fibres with the grey matter of the septum lucidum. The *tegmentum* forms the posterior or deeper part of the *crus cerebri.* Its fibres are continuous with the longitudinal fibres of the pons derived from the olivary fasciculi, fasciculi teretes, and posterior pyramids of the medulla. A few fibres enter the corpora quadrigemina and corpora geniculata; but the majority enter the optic thalami, in the grey matter of which many evidently terminate, while some may pass thus into the cortex of the hemispheres as fibres of the corona radiata.

The *optic thalami* (Fig. 8, *d*) (*posterior cerebral ganglia*) are of an oval shape, and rest on the corresponding cerebral crura, which they, as it were, embrace. The inner sides of each are in partial contact; and they present grey substance uncovered with white, and are generally connected together by a transverse portion—the *middle* or *soft commissure* (Fig. 8, *f*) of the third ventricle. The upper surface is free, prominent, and white, partly seen in the lateral ventricle, and partly covered by the fornix. The posterior surface is also white and free, and projects into the descending curve of the lateral ventricle. The *posterior commissure* (Fig. 8, *g*), on which is situated the pineal gland, also joins the two optic thalami. Each optic thalamus forms an almost continuous mass of grey matter traversed by nerve fibres, but they are not connected into definite striæ. The grey matter of the thalamus also gives origin to numerous radiating fibres. Those which arise in its posterior part radiate into the occipital and temporal lobes; whilst those proceeding out of its anterior part radiate into the frontal, parietal, and temporal lobes, and the insula.

The external *corpus geniculatum* consists of alternate layers of grey and white matter, due to the zig-zag folding of the grey matter.

The *corpora quadrigemina* are made up of four tubercles—*nates* (two anterior) and *testes* (two posterior) (Fig. 12, 8). The two anterior are connected with the optic thalamus, and with the optic tracts which spring from them, and wind round the crura cerebri (Fig. 11, II') to the middle line at the base of the brain, and there undergo decussation with each other in the *optic chiasma* or *commissure*. Various opinions are entertained as to the completeness or incompleteness of this decussation in man (Ferrier, l. c., p. 72). The posterior are related to the cerebral peduncle. By the superior peduncle of the cerebellum they are also connected with that organ.

The *grey matter* of the *corpora quadrigemina* consists of two distinct masses. One, the *peripheral layer*, lies near the surface; the other, the *Sylvian* or *central layer*, surrounds the Sylvian fissure, and belongs to the grey matter of the wall of the cerebro-spinal tube, and serves as a centre of origin for the roots of both the third and fourth cranial nerves. The central grey matter of the cerebrum is in series with the grey matter of the floor of the fourth ventricle, and with the grey matter of the spinal cord. It is accumulated around the Sylvian aqueduct, and at the sides and floor of the third ventricle, which forms the cerebral portion of the cerebro-spinal tube. All the nerve fibres from the grey cortex matter of the convolutions (except the commissural fibres connecting opposite sides) converge to the *basal ganglia* and to the *corpora geniculata* and *corpora quadrigemina*, and the *locus niger* in each crus cerebri. They are thence continued (directly or indirectly) either through the superior cerebellar peduncles to the cerebellum, or along the crura cerebri to the medulla oblongata. Thus, the optic thalami, corpora geniculata, and corpora quadrigemina derive fibres from nearly all parts of the cerebral surface, though mainly probably from the posterior and lateral portions.

Thus the grey matter of the cerebrum is disposed in three great groups:—(1.) The grey matter of the cortex of the hemispheres; (2.) the grey matter of the great ganglia of the base of the cerebrum; (3.) the central grey matter which forms the wall of the cerebral end of the cerebro-spinal tube.

The grey matter of the cortex of the hemisphere forms the superficial part of the convolutions. It is known as *the great hemispherical ganglion*, which, in certain localities (as at the *loci perforati antici* and the *septum lucidum*); has received distinctive names. When a convolution is divided vertically across, the grey matter is seen to be confined to the surface, and to be laminated, the layers being arranged parallel to the plane of the surface; and in the axis of the convolution a white core is inclosed, the strands of which radiate into the adjoining grey matter. When thin slices through the white core are examined microscopically, it is seen to consist of nerve fibres and blood-vessels, embedded in a very delicate, richly nucleated protoplasm—an imperfectly differentiated form of connective tissue—which by binding the other constituents together has been named by Virchow the *Neuroglia* or *Nerve-glue*.

The *grey matter of the cortex* consists of five layers (Lockhart Clarke, Meynert, Quain) (Fig. 9), which are composed of the characteristic and pyramidal nerve cells of the cortex of the cerebrum, of nerve fibres, of matrix or neuroglia, and of blood-vessels. The nerve cells vary in relative size, in numbers, and in shape in the different layers of the grey cortex. The pyramidal shape is, however, characteristic. The most superficial layer consists of neuroglia, in which nerve fibres extend parallel to the surface of the convolutions. In the deeper layers are found the pyramidal nerve cells, lying with their long axes vertical to the surface of the convolutions, so that the basal part of the cells is directed towards the white core, while their apices point towards the surface of the convolutions. They contain well-marked nuclei. The largest cells lie in the third and fourth layers. Very fine processes arise from these cells. One proceeding from the base of each is continuous with one of the vertical or radiating nerve fibres of the white core. Other cells are fusiform; and others like stellate corpuscles. The large cells are found in the frontal lobe in considerable numbers; and also in the occipital lobe in fewer numbers, mixed there with small cells nearly uniform in size. The nerve fibres which ascend from the white core

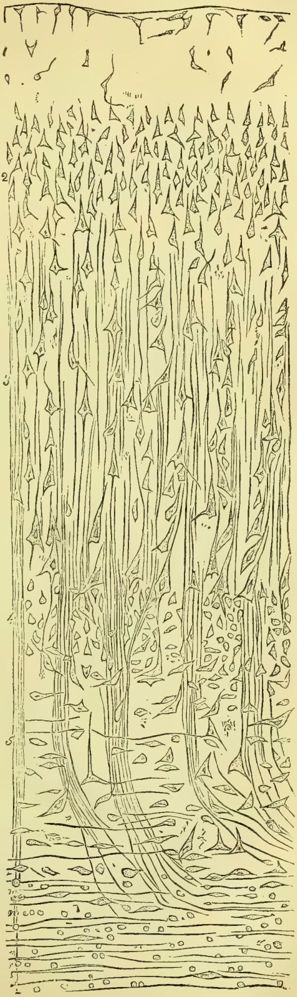

Fig. 9.*

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* SECTION OF THE GREY MATTER OF THE HEMISPHERE, taken from the third frontal convolution (after MEYNERT.)—1. Superficial layer of scattered small cortical corpuscles; 2. Dense close-set layer of small pyramidal or angular cortical corpuscles; 3. Layer of large pyramidal cortical corpuscles (Ammonis horn formation), separated into columns by the radiating nerve fibres; 4. Narrow layer of small close-set irregular corpuscles (granule-like formation); 5. Layer of fusiform and irregular cells in medullary centre (claustrum formation); *m.* Medullary lamina.

of the convolution into the grey matter, radiate into its several layers, and are apparently continuous with the basal axial cylinder processes of the nerve cells. The elongated apices of the cells, which point to the surface of the convolution, are continuous with the nerve fibres situated in the superficial layers of horizontal fibres (CLELLAND). Immediately subjacent to the large pyramidal cells, numerous small, irregularly shaped nerve corpuscles (like those of the internal granular layer of the retina), form the so-called *granule layer* of the grey matter. Fusiform cells, giving off lateral processes, are found in the deepest layer (*claustral layer* of MEYNERT). The later branched processes of those cells are continuous with a network of very fine nerve fibres, similar to what is found in the spinal cord. The neuroglia contains numerous small rounded corpuscles, and small stellate cells powdered with minute branched processes. So pellucid are those cells, that they seem to be only free nuclei, and may belong either to neuroglia or to nerve-cell elements.

The grey matter of the cortex has been accurately measured in various regions by Dr. Herbert Major, by means of a simple instrument devised by him, and described, with the results he obtained by its use, in the second volume of the *West Riding Asylum Medical Reports*, p. 160. The instrument has been named a Tephrylometer (*τεφρά*, ash-coloured, and *ὕλη*, matter), or measure of the ash-coloured matter of the brain. It consists of a graduated glass tube, open at both ends—in length about 5 inches, in thickness the wall of the glass is about that of a goosequill, but at one end it is bevelled off on its outer side to a sharpness necessary for piercing the pia mater and substance of the brain. It is graduated on a fiftieth of an inch scale over half the length of the instrument, commencing at a quarter of an inch from the piercing or bevelled off end, by means of which the grey lamina inclosed may be read off. Each fifth degree (*i.e.*, every tenth inch mark) is longer than the others. The bore of the tube must be everywhere of uniform size. The diameter of the bore may be eight-fifths, six-fifths, or five-fifths of an inch. By this instrument he found that the depth of the grey matter of the cerebrum varies in different situations in accordance with a definite plan. The depth of the cortical layer of the occipital lobes and of the orbital lobes was uniformly markedly less than that of the frontal or parietal lobes; but that the grey layer of the island of Reil is always deep (but more pale), or as deep as the cortex of any part of the hemispherical ganglion (l. c., p. 175).

Microscopic Appearances of the Nerve Cells vary in the followiug accumulation of Grey Matter.—The cortex of the island of Reil and of the convolutions bounding the Sylvian fissure, contains a very large proportion of fusiform cells (MAJOR in *West Riding Asylum Reports*, Vol. VI., p. 6). They form the chief constituent of the grey *claustrum.* Fusiform cells also occur abundantly in the *nucleus amygdale* (a grey mass situated below the corpus striatum), continuous with the grey matter of the inferior temporo-sphenoidal convolution. The grey matter of the locus perforatus anticus contains clusters of minute granules, and a compact arrangement of small nerve cells. Multipolar nerve cells are found in both the caudate and lenticular masses of the corpus striatum; and, in the latter, cells of large size have been seen. In the optic thalamus, the nerve cells are both multipolar and fusiform. In the external corpus geniculatum the nerve cells are multipolar and contain pigment. In the internal corpus geniculatum the cells are smaller in size and fusiform. The peripheral layer of the corpora quadrigemina contains numerous small multipolar nerve cells. The collection of grey matter in the crura cerebri, known as the *locus niger,* consists of multipolar nerve cells containing dark brown or black pigment. The grey matter which surrounds the aqueduct of Sylvius, forming the *Sylvian* or *central* layer in the corpora quadrigemina, contains numerous well-marked multipolar nerve cells, which are largest and most distinct below the aqueduct, where they form well-defined groups; and a remarkable zone-like arrangement of nerve cells lies at the circumference of the sides and upper part of the central mass of grey matter. In vertical transverse sections these cells may be seen to lie sometimes singly, but more usually in groups of two or three, or up to six cells. These cells (at the circumference) are twice, or even three times, as large as the multipolar nerve cells of the central mass. The body of the cell is often globular in form, and from it one or two processes arise. The cells lie in a tract of well-defined nerve fibres, to which they have apparently the same relation that the cells in the ganglion, or the posterior root of a spinal nerve, have to the nerve fibres (TURNER). The pituitary and pineal bodies, besides the nervous matter, contain structures of the type of the glands without ducts.

"There can be little doubt that the larger nerve cells are functionally more active, and evolve more nerve energy than do those of smaller size. Though more extended data in support of the anatomical continuity of the nerve cells of the convolutions with the nerve fibres is to be desired, yet, on physiological grounds, there can be little doubt that each nerve cell has at least one nerve fibre either directly or indirectly continuous with it, so that multitudes of conducting rods are provided in the large nerve centres, along which the energy evolved by the nerve cells can be transmitted. It would be very difficult indeed to conceive how nerve fibres could fulfil the functions which are universally ascribed to them, if they were not anatomically continuous with nerve cells, as without such connection they would be like strings of knotless threads, or the wires of a telegraph cable dissevered from their central battery" (TURNER, *West Riding Asylum Reports,* Vol. III).

White Matter of the Cerebrum.—"In studying the arrangement of the nerve fibres of the cerebrum, it is of great importance," writes Professor Turner, "to determine the portions of the cerebrum, and the groups of nerve cells with which they are continuous. Without this knowledge it is not easy to determine what the parts are which are associated together." The *white matter* of the cerebrum consists of tracts or fasciculi of nerve fibres, of which (*a.*) some connect the cerebrum with the lower divisions of the encephalon; (*b.*) others connect the two hemispheres together; (*c.*) others connect different structures in the same hemisphere; (*d.*) others are roots of origin for the more anterior encephalic nerves. The more generally known and accepted facts regarding these connections are thus given in the words of Professor Turner :—"We know, for example, that a great system of longitudinal or peduncular fibres ascends from the spinal cord and medulla oblongata through the pons and crura cerebri to the optic thalami and corpora striata; from which the fibres may either directly or indirectly, after junction with the nerve cells of those bodies, radiate outwards to the grey cortex of the cerebrum, more especially to that of the frontal lobes. But peduncular fibres also ascend from the grey matter of the spinal cord and medulla to the cerebellum; and others again, arising in the cerebellum, ascend to the corpora quadrigemina, which latter again are connected with the cerebrum proper by peduncular fibres.

"We know also that a great system of transverse fibres passes across the mesial plane from one hemisphere to the other. The most numerous and important of these fibres constitute the corpus callosum, which connects together corresponding convolutions in the opposite hemispheres, and its fibres decussate with the fibres of the peduncular system, passing to the same convolutions. But smaller groups of fibres also cross the mesial plane. The anterior commissure, for example, as was pointed out by Spurzheim half a century ago, passes between and connects together the convolutions of the opposite temporo-sphenoidal lobes, and in its course traverses the two corpora striata, which, in all probability, it also brings into anatomical connection with each other. The posterior commissure, again, connects the two optic thalami.

"But in each hemisphere, also, there are numerous fibres collected together into more or less distinct bands, which, under various descriptive names, as fornix, tænia semicircularis, nerves of Lancisi, fasciculus uncinatus, &c., run in the antero-posterior direction for a greater or less distance, as the case may be, to connect together structures situated on the same side of the mesial plane. Amongst the most important of these fibres are those which connect together adjacent convolutions or groups of convolutions.

"Lastly, from the grey matter in certain of the subdivisions of the encephalon bands of nerve fibres arise, which, under the name of cranial nerves, connect the brain with various sensory and motor peripheral-end-organs. But of the cranial nerves only two—namely, the olfactory and optic, appear to have any immediate connection with the grey matter of the convolutions; all the rest taking their origin in groups of nerve cells, forming the centres which lie nearer the base of the encephalon.

"From these general facts we reach the very important conclusion—that the convolutionary area of the hemispheres does not form a system dissociated from the other nerve centres; that not only are convolutions in the same hemispheres connected together, but that they are anatomically continuous with the various centres from which the cerebro-spinal nerves arise, and through these are brought into relation with the outer world."

6. The Cerebellum occupies a position posterior to the corpora quadrigemina, and is connected to the cerebro-spinal axis by its *peduncles.* These are three—(1.) The *superior peduncles, crura ad cerebrum,* or *processus ad testes.* These enter the posterior extremity of the corpora quadrigemina, converge beneath them, and decussate into the red nuclei or tegmenti, the *sensory* regions of the crura cerebri. Their farther destination is doubtful, but, according to Meynert, they form part of the motor division of the

corona radiata. The *valve* of Vieussens—a lamina stretched between the peduncles and forming the roof of the fourth venticle—directly connects the middle lobe of the cerebellum with the corpora quadrigemina by their posterior tubercles. (2.) The *inferior peduncles, crura ad medullam,* or *restiform tracts,* are the upper extremities of the restiform bodies. (3.) The *middle peduncles,* or *crura ad pontem* (much the largest), are the lateral extremities of the transverse fibres of the pons Varolii, of which they form the great mass. They connect together the two halves of the cerebellum inferiorly. These middle peduncles are developed proportionately to the lateral lobes of the cerebellum. They undergo decussation in the middle line, and enter into connection with the strands which pass to the opposite cerebral hemispheres. Hence *the right half of the cerebellum* becomes functionally related to the *left cerebral hemisphere;* and thus a cross relation is established between the cerebral and cerebellar hemispheres. All these peduncles pass into the interior of the cerebellum at its fore part (QUAIN). In form the cerebellum is divided into lobes—a median and two lateral. Its surface is disposed in the form of laminated folds, and the grey matter which forms its surface exhibits on section (Fig. 12) the appearance of leaflets (*arbor vitæ*). Microscopically the cerebellum has been minutely examined by Obersteiner, Meynert, H. R. Octavius Sankey, and others. The grey matter or outer layer is composed of a beautiful arrangement of granules, cells, and nerve fibres. Its neuroglia is scattered over with round and elongated nuclei. Dr. Sankey's observations mainly confirm those of Obersteiner.

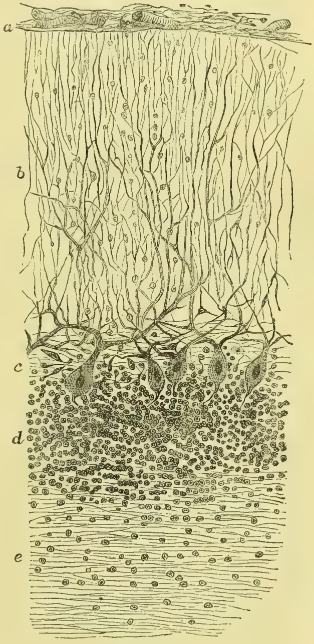

Fig. 10.*

"If in a section made perpendicularly to the surface of the cerebellum, and at right angles to the lamellæ, one of Purkinje's cells be brought into

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[*] STRUCTURE OF THE CORTEX OF THE CEREBELLUM (from a drawing and observation of Mr. H. R. O. SANKEY). *a.* Pia mater; *b.* External layer; *c.* Layer of corpuscles of Purkinje; *d.* Inner or granule layer; *e.* Medullary centre.

view under a magnifying power of 600 diameters, and its peripheral processes traced out, the following appearances will be observed (Fig. 10):—Each cell gives off, in a direction towards the surface, one primary trunk. This soon divides into two secondary trunks, which pass to the right and left in a direction parallel to the surface, and in their progress send off at right angles numerous branches, which also are directed towards the surface or towards the pia mater; and, finally, the secondary branches, having gradually become smaller and smaller in their course, terminate themselves by turning, like the smaller processes, towards the surface. At each point of division there is to be observed a small triangular swelling, which might aptly be compared to a small blot, such as might occur when a line of ink is drawn across another which is still wet. Various opinions have been broached as to the nature of this swelling. . . . The fibres running outwards may be seen to divide and subdivide into very fine branches, dividing not three or four times only, but much more frequently. I have observed one branch to divide more than twenty-five times. The more distant branches are given off at very acute angles, and the fibres, after a very short distance, run a nearly or quite parallel course. The division is not strictly dichotomous; for in many instances small branches are given off from the sides of larger ones, though more usually two branches of equal size result from the division of one. Dichotomous division prevails more as the fibres become smaller. The branches, having thus become finer and finer, are ultimately lost to view. In my preparations the union of the fibres with the protoplasm of the cells, in the pure grey layer, is plain and unmistakable, and resembles very closely the appearance described by Dr. Obersteiner. The fine fibres, on approaching the cells in which they are about to end, are seen in my preparations to enlarge and assume the character of the protoplasm of the cell, into which they pass without line of demarcation. Most of the cells seen in my preparations are not round, but, as Dr. Meynert describes them, triangular or pastille-shaped; and there is no clear space around the cell. These small cells are not only seen to be provided with a process of union connecting them to the fibres in the manner described, but also to give off others three or four in number, which are short, but which divide and subdivide until they are finally lost in the reticulo-molecular ground substance of the part. The 'processes of union,' which are connected with Purkinje's cells, are usually directed toward that layer of cells, but this is not an invariable arrangement. My preparations corroborate Dr. Obersteiner's opinion that the terminal branches of the processes are directly united to cells, and that also side-twigs are given off in a more or less rectangular direction, which also join cells, each fibre making for its cell a kind of stalk. I have, however, more frequently observed the former kind of connection than the latter. There are nuclei of two kinds in the pure grey layer of Kölliker. Firstly, there are those cells which are surrounded by a protoplasmic substance, and which are the most numerous—these are the cells which are connected with the fibres from the cells of Purkinje; and, secondly, there are undoubtedly others around which no protoplasm is to be seen—these are either round or more frequently slightly elongated, and both Dr. Obersteiner and Dr. Meynert regard them as free nuclei belonging to the neuroglia. In my preparations small arteries and veins may be detected, but no capillaries, or, if any, very few, and these but faintly indicated; but by taking a freshly-stained portion of cerebellum and teasing it, the nuclei of the capillaries may be readily detected, and will be found to resemble in appearance, shape, and size the second kind of nuclei referred to above. I am therefore of opinion that this kind of nucleus belongs to the capillaries, and is not an element of the neuroglia, and their numbers are about what such an origin would readily account for" (*West Riding Asylum Reports*, Vol. V., p. 193).

7. PONS VAROLII, MEDULLA OBLONGATA, AND SPINAL CORD.—(*a.*) *The pons Varolii*, or Bridge (Fig. 11, PV), consists of fibres passing in two directions—(1.) Longitudinally, connecting the brain above with the medulla and cord below; (2.) transversely, connecting the lateral hemispheres of the cerebellum through the middle peduncle of that organ. Mixed up with the fibres are various nuclei or deposits of grey matter, into which the roots of several of the cranial nerves may be traced. It is, therefore, commissural or connecting in two directions. Its anterior surface rests on the dorsum Sellæ of the sphenoid bone, and is marked by a median longitudinal groove; its inferior surface receives the pyramidal and olivary tracts of the medulla oblongata; at its superior surface are the two crura cerebri; each lateral surface is in relation to a hemisphere of the cerebellum, and a peduncle passes from the pons into the interior of each hemisphere. The posterior surface forms in part the upper portion of the floor of the fourth ventricle, and in part is in contact with the corpora quadrigemina. The grey matter of the pons is scattered irregularly through its substance, and appears on its posterior but not on its anterior surface, which is composed exclusively of superficial transverse fibres. The nerve cells are multipolar and stellate. Some of the fibres of the crura cerebri end in the nerve cells of the pons, which cells again give origin to fibres that pass outwards to the cerebellum. (*b.*) *The medulla oblongata* rests in the basilar groove, upon the basi-occipital bone. It is bilateral, divided into a right and left half by shallow anterior and posterior median fissures, continuous with corresponding fissures in the spinal cord; the posterior fissure ends above in the fourth ventricle. Each half is subdivided into elongated tracts of nervous matter. Next to and parallel with the anterior fissure is the *anterior pyramid*, continuous below with the spinal cord, on a level with the upper border of the atlas. The place of continuity is further marked by the passage across the fissure of three or four bundles of nerve fibres, from each half of the cord to the opposite anterior pyramid—a crossing known as the decussation of the pyramids. To the outer sides of the pyramid on each side are two smooth masses—the projecting oval-shaped eminences, named the *olivary bodies*, sunk to a considerable depth in the substance of the medulla oblongata. Externally they mainly consist of white fibres, and internally of a grey nucleus—the *corpus dentatum* or *ciliare*, or olivary nucleus—which presents on section a zig-zag light yellow coloration circumscribing a whitish substance within, and intercepted towards the centre of the medulla, through which bundles of nerve fibres proceed from the interior, which aid in the formation of the olivary fasciculus, and in part arch across the mesial plane to join the restiform body on the opposite side, while some apparently join the posterior pyramid (DEITERS and MEYNERT). Behind the olivary bodies, and separate from them by a faint groove, is the strong tracts named the *restiform bodies*, directly continuous with the posterior, and with part of

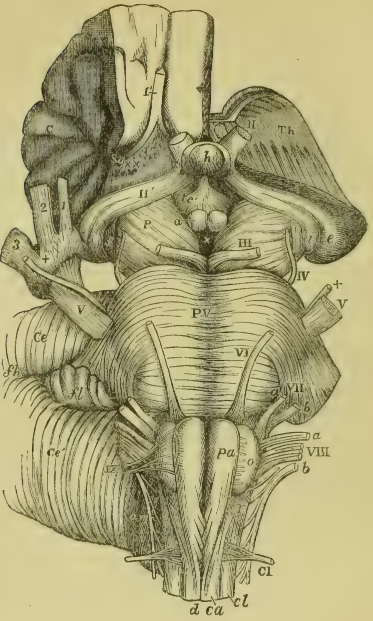

Fig. 11.*

* View from before of the Medulla Oblongata, Pons Varolii, Crura Cerebri, and other Central Portions of the Encephalon (after Allen Thomson). On the right side the convolutions of the central lobe, or island of Reil, have been left, together with a small part of the anterior cerebral convolutions; on the left side these have been removed by an incision carried between the thalamus opticus and the cerebral hemisphere. 1'. The olfactory tract, cut short and lying in its groove between the convolutions; II. The left optic nerve in front of the commissure; II'. The right optic tract; T*h*. The cut surface of the left optic thalamus; C. The central lobe, or island of Reil; S*y*. Fissure of Sylvius; XX. The locus perforatus anterior; *e*. The external, and *i*. The internal corpus geniculatum; *h*. The hypophysis cerebri, or pituitary body; *tc*. Tuber cinereum, with the infundibulum; *a*. Points by a short line to one of the corpora albicantia; P. The cerebral peduncle or crus; *f*. The fillet; III. Close to the left oculo-motor nerve; X. The locus perforatus posticus; PV. Pons Varolii; V. The greater root of the fifth nerve; +. The lesser root: on the right side this + mark is placed on the Gasserian ganglion, and points to the lesser root, where it proceeds to join the inferior maxillary nerve; 1. Ophthalmic division of the fifth nerve; 2. Supra-maxillary division; 3. Inferior maxillary division; VI. The sixth nerve; VII*a*. The facial; VII*b*. The auditory; VIII. The pneumogastric nerve; VIII*a*. The glossopharyngeal; VIII*b*. The spinal accessory nerve; IX. The hypoglossal nerve; *fl*. The flocculus; *fh*. The horizontal fissure of the cerebellum (C*e*); *am*. The amygdala; *pa*. The anterior pyramid; *o*. The olivary body; *r*. The restiform body; *d*. The anterior median fissure of the spinal cord; *cl*. The lateral column of the spinal cord; CI. The sub-occipital or first cervical nerve.

the antero-lateral columns of the cord. They diverge as they ascend, and each passes into the corresponding hemisphere of the cerebellum, and becoming free and prominent, constitutes its inferior peduncles. Thus by the divergence of each the central part of the medulla is opened up, and the lower half of the cavity of the fourth ventricle is formed. There is a considerable quantity of grey matter in their interior. The *posterior pyramids* (*fasciculus gracilis* or *fasciculi graciles*) are the smallest of the four pairs of columns of the medulla oblongata. They are internal to the restiform bodies, and are situated one on each side of the posterior median fissure, and are continuous with the postero-median columns of the cord. They consist of white fibres, and contain much grey matter. The *floor of the fourth ventricle* is the space between the medulla and cerebellum, and is formed by that portion of the back of the medulla oblongata which is situated above the divergence of the posterior pyramids, and upon it the central grey matter of the medulla is opened out to view. It is marked by a median furrow ending inferiorly in the *calamus scriptorius* (Fig. 12), and its lower end is a tubular recess passing down the centre of the medulla for a few lines, and which is the upper expanded portion of the central canal of the spinal cord. The medulla oblongata is thus the prolongation upwards of the spinal cord, which unites it with the brain, and amongst special anatomical facts relating to it, Professor M'Kendrick points out that—

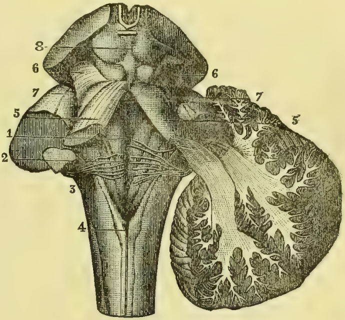

Fig. 12.*

"Nearly the same columns of grey and white substance that exist in the cord are to be recognised in the medulla, but modified in form, structure, and relative position, and combined with several nerve fasciculi and masses of grey matter. The greater part of the fasciculi may be traced upward into the brain, some proceeding towards the cerebellum and others to the cerebrum. It is doubtful whether any fibres pass directly to or from the encephalon and the lower portions of the cord. Many physiologists are of opinion that direct continuation is at all events not the rule; and that the majority of fibres both from above and from below find a new point of departure in the cells of the medulla. Still the tracts along which nervous transmission probably takes place may be traced from below upwards as follows:—(1.) The fibres forming the *anterior columns* divide into two portions, passing to the same side of the encephalon, as (*a.*) a small band which ultimately reaches the cerebellum; and, (*b.*) a portion separated by anatomists into two strands, which reach the corpora quadrigemina and the cerebral hemispheres; (2.) The fibres forming the *lateral column* pass partly to the cerebellum on the same side, partly to the cerebrum on the same side, and partly to the cerebrum on the opposite side; and, (3.) The greater portion of the *posterior column* passes to the cerebellum—a small part of the *fasciculus gracilis* going to the cerebrum. It is important to note that each column of the cord, through the medulla, is thus connected both with the cerebrum and with the cerebellum" (*Outlines of Physiology*, p. 509).

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* View of the Floor of the Fourth Ventricle, with the posterior surface of the medulla oblongata and neighbouring parts (from Sappey, after Hirschfeld and Leveillé). On the left side the three cerebellar peduncles have been cut short; on the right side the white substance of the cerebellum has been preserved in connection with the superior and inferior peduncles, while the middle one has been cut short. 1. Median groove of the fourth ventricle with the fasciculi teretes, one on each side; 2. The same groove at the place where the white striæ of the auditory nerve emerge from it to cross the floor of the ventricle; 3. Inferior peduncle or restiform body; 4. Posterior pyramid, above this the calamus scriptorius; 5. Superior peduncle, or processus a cerebello ad cerebrum; on the right side the direction shows the superior and inferior peduncles crossing each other as they pass into the white centre of the cerebellum; 6. Fillet to the side of the crura cerebri; 7. Lateral grooves of the crura cerebri; 8. Corpora quadrigemina.

The grey matter of the medulla oblongata contains numerous multipolar nerve cells; and is in part continuous with the grey matter of the spinal cord, and in part consists of independent masses. (*c.*) *The spinal cord*, or *spinal marrow*, *medulla spinalis*, lies within the vertebral canal, and extends from the level of the atlas to that of the space between the first and second lumbar vertebræ—varying in length from fifteen to eighteen inches. It is cylindrical, and somewhat flattened before and behind. It presents two enlargements—the upper cervical, or brachial enlargement, which is of greater size and extent than the lower one in the lumbar region —the crural, or lumbar enlargement. These enlargements have a relation to the large size of the nerves which supply the upper and lower limbs, and which are connected with these regions of the cord. It presents an anterior and posterior median fissure; and two lateral furrows or fissures, which correspond with the lines of attachment of the anterior and posterior roots of the spinal nerves. Its internal structure is made up of white and grey nerve substance, of which the white is in the larger proportion, is situated externally, and forms the columns of the cord with the grey in the interior. The spinal cord is continuous above with the medulla oblongata; and below it tapers off into a slender thread—*filum terminale*—which is attached to the back of the coccyx. The two halves of the cord are united by commissures. In a transverse section the grey matter is seen to have in each lateral half a crescentic shape with the convexities of the crescents towards one another. They are joined across the middle by a transverse portion—the *grey* or *posterior commissure* of the cord. The white matter increases in amount from below upwards, so that its absolute quantity is greatest in the cervical part of the cord, and the grey crescents are thicker in the upper and lower enlargements than in the intermediate part. The grey crescentic portion contains nerve cells, nerve fibres, connective tissue, and blood-vessels. The nerve cells are multipolar, and are chiefly collected in the anterior and posterior horns of each crescent. In the anterior they are large, distinct, and stellate, forming well-defined groups of nerve cells. In the posterior cornu the nerve cells are of much smaller size, more elongated, but with stellate-branched processes. They are less distinct in the posterior than in the anterior horn, on account of the abundance of connective tissue with its corpuscles; and at the extreme tips of the posterior horn, which is somewhat enlarged, the grey matter has a peculiar transparent aspect—the *substantia cinerea gelatinosa* of Rolando. Groups of large stellate-branched cells are also to be seen at a short distance from the tips of the posterior cornu. The external columnar white part of the cord consists of nerve fibres, with a supporting reticular framework of connective tissue and blood-vessels, derived from the pia mater. The nerve fibres of the various columns extend longitudinally, lying parallel to each other. Individual fibres vary much in diameter, but in all of them the axial cylinder and medullary sheath can be distinctly seen. Wherever nerve roots enter the cord, the fibres pass horizontally or obliquely inwards to the grey matter, and the fibres of the anterior and posterior commissures, have a horizontal course.

As to the course of the fibres in the cord and their ultimate destination much difference of opinion still exists; but there seems little doubt that the radiating prolongations of the nerve cells are in actual continuity with the axial filaments of nerve fibres, whether proceeding from nerve roots or from different parts of the cord itself. Of the longitudinal fibres, some ascend from below upwards. Other longitudinal fibres descend from the brain and higher regions of the cord to the lower. The horizontal and oblique fibres of an *anterior* (*motor*) *nerve root* enter the grey matter of the anterior cornu, and seem to have the following arrangement:—(*a.*) Some become directly continuous with the axial cylindrical processes of the nerve cells; (*b.*) others pass into the anterior commissure; (*c.*) others extend as far as the grey matter of the posterior horn. The nerve cells of the anterior cornua give origin, therefore, to nerve fibres by their unbranched processes; while the branched processes of those cells become continuous with the network of extremely minute fibres in the grey matter. From this network medullated fibres appear to rise, which leave the grey matter; some of these enter the lateral column, and ascend as the fibres of this structure; others pass as fibres of the *anterior commissures* to the opposite side of the cord, and ascend as the anterior column of that side. The anterior and lateral columns, therefore, are constantly receiving accessions of fibres from the inclosing grey matter. The fibres of a *posterior* (*sensory*) *nerve root* on entering the cord subdivide into two bundles, one of which does not enter the grey matter, but applies itself to the posterior column, of which it forms some of the longitudinal fibres. These fibres may ascend to the brain, or they may at some higher point in the cord enter the grey matter of the posterior horn. The other bundle of posterior root fibres at once enters the posterior horn of grey matter; but the connections and ultimate arrangements of these fibres in the grey matter have not yet been clearly made out. They are known to frequently subdivide on entering the grey matter, and thus they may form the fine nerve-fibre plexus of the grey substance; but any direct continuity between them and the axial cylinder processes of the cells of the posterior horn does not seem to have been observed. From the plexus formed by the much divided processes of these cells fibres arise, which (forming the fibres of the posterior commissure) pass both in front of and behind the central canal to the opposite side, where they ascend towards the brain, "partly in the vertical fasciculi of the posterior cornua, and partly in the posterior columns" (TURNER). Thus the nerve cells in the grey matter of the cord give rise either directly, or through the delicate plexus formed by their branching processes, to nerve fibres, which may either pass out of the cord as the anterior and posterior roots of the spinal nerves, or may ascend to the brain as the columns of the cord. Hence the cord is anatomically continuous, on the one hand (through the nerves which arise from it), with the peripheral end-organs in the skin and muscular system in which these nerves terminate; and, on the other hand, it is continuous with the brain. The two halves of the cord are also anatomically continuous with each other through the nerve fibres of the commissures. Professor M'Kendrick has embodied the general facts relative to these structures in the following useful table, which may help to keep them in remembrance:—

PRINCIPAL FASCICULI OF THE MEDULLARY FIBRES TRACED FROM SPINAL CORD THROUGH THE MEDULLA OBLONGATA INTO THE BRAIN (M'Kendrick).

Columns of Spinal Cord.	Divided into	Continued in the Medulla Oblongata as	And pass into the	
I. ANTERIOR	1. Small division called Band of Solly.	Part of restiform body.	Cerebellum.	...
	2. Portion surrounding olivary nucleus.	Part of olivary fasciculus.	...	Corpora quadrigemina and cerebral hemispheres.
	3. Outer fibres.	Outer part of anterior pyramid.	...	Cerebrum.
II. LATERAL	1. Fibres from surface and deep part.	Part of restiform body.	Cerebellum.	...
	2. Decussating fibres.	Anterior pyramid.	...	Cerebrum.
	3. Remainder.	Fasciculus teres.	...	Cerebrum.
III. POSTERIOR	1. Fasciculus cunietus, or all the fibres with the exception of the posterior median column.	Part of restiform body.	Cerebellum.	...
	2. Posterior median column, or fasciculus gracilis.	Posterior pyramid.	...	Cerebrum.

8. THE ENCEPHALIC OR CRANIAL NERVES arise within the cranium in the upward continuations of the tracts of grey matter from the cord into

the encephalon. One set springs from that which represents the upward continuation of the grey matter of the cord anterior to the spinal canal (*motor* or *efferent*); the other set from that portion of the grey matter which is behind it (*sensory* or *afferent*). But as these tracts are allied in their relative positions in the medulla oblongata and floor of the fourth ventricle, the anterior (*motor* or *efferent*) tract becomes superficial on either side of the middle line in the fasciculi teretes; the posterior (*sensory* or *afferent*) tract spreads out from the floor of the fourth ventricle on either side along the inner aspect of the cerebellar peduncles towards the cerebellum; and at the anterior part of the ventricle they again coalesce as in the cord. The cranial nerves are so named because they pass through foramina in the base of the cranium. They are arranged according to the following systems:—

English System (Willis).	Physiological Name.	Continental System (Soemmering).
First pair,	Olfactory,	First pair.
Second pair,	Optic,	Second pair.
Third pair,	Oculo-motor,	Third pair.
Fourth pair,	Pathetic,	Fourth pair.
Fifth pair,	Trifacial,	Fifth pair.
Sixth pair,	Abducal-ocular,	Sixth pair.
Seventh pair, *portia dura*,	Facial motor,	Seventh pair.
portia mollis,	Auditory,	Eighth pair.
Eighth pair,	Glossopharyngeal,	Ninth pair.
	Pneumogastric or Vagus,	Tenth pair.
	Spinal accessory,	Eleventh pair.
Ninth pair,	Hypoglossal,	Twelfth pair.

The *olfactory*, or first cranial nerve, springs from the olfactory bulb (I, Fig. 13), which appears at the frontal extremity of the orbital region of the hemisphere; and is connected with the hemisphere by a peduncle or tract called the olfactory tract (I′, Fig. 13). The tract and bulb are an offshoot from the hemisphere; for the bulb and tract constitute in reality a lobe of nervous substance composed of grey and white matter, inclosing a central cavity or ventricle continuous (in the embryo and permanently in some of the lower vertebrates) with the cavity of the hemispheres. The olfactory tract is continuous with the uncinate gyrus and the frontal end of the gyrus fornicatus, and great differences are observed in these parts of the brain in different animals. The connections of the tract or peduncle with the hemisphere constitute the roots of the olfactory tract. The outer one can be traced across the *locus perforatus corticus* (XX, Fig. 11) to that part of the brain which forms the lower extremity of the temporo-sphenoidal lobe. The other root joins the hemisphere at its internal margin, just anterior to the optic commissure. The farther course of this root has not been satisfactorily traced. According to Meynert it joins the corpus striatum. By means of the anterior commissure the olfactory bulbs are joined together, a commissure which also connects together only those parts of the hemisphere to which the outer roots are traceable,—namely, the temporo-sphenoidal and occipital lobes, and so they pass to opposite sides, an *olfactory chiasma* being thus formed. From the bulbs arise the olfactory nerves proper, which perforate the cribriform plate. The *second pair*, or optic nerves, terminating in their end-organ (the retina of each eye), proceed by the optic tracts which wind round the crura cerebri (II′, Figs. 11 and 13) to the middle line at the base of the brain, where they undergo (in man incomplete) decussation with each other in the *optic chiasma* or *optic commissure.* They arise in part from the optic thalamus, the corpora geniculata externa, and in part from the corpora geniculata externa, and from the anterior corpora quadrigemina, which may be regarded as containing the nuclei of the optic nerves. In these ganglia radiating fibres, proceeding from the occipital and temporal lobes, converge to form a thick medullary lamina (optic radiations of Gratiolet), covered by a large number of arcuate fasciculi. The optic tracts, in their whole extent,

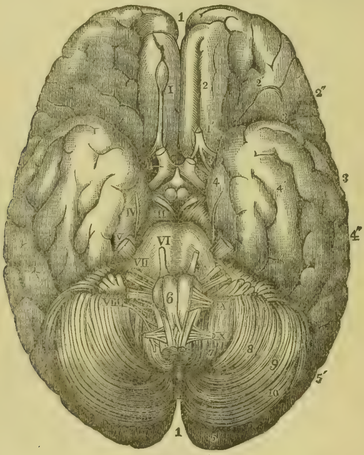

Fig. 13.*

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[*] Base of the Brain, with the origins of the cerebral nerves (after Allen Thomson). Half size. The figure is taken from an adult male brain which had been hardened in alcohol. 1. Superior longitudinal fissure; 2. Within the straight olfactory tract and sulcus for the convolution; 2′. Orbital convolutions; 2″. Inferior frontal convolution; 3, 3, 3. Fissure of Sylvius; 4, 4, 4. Temporo-sphenoidal lobe; 5, 5′. Occipital lobe; 6. On the right anterior pyramid of the medulla oblongata above the decussation; 7. Amygdaloid lobe of the cerebellum; 8. biventral lobe; 9. Lobulus gracilis; 10. Posterior-inferior lobe; +. The inferior vermiform process; I. Olfactory bulb; I′. The tract divided on the left side; II. In the right anterior perforated spot, marks the right optic nerve—the left has been cut shorter; III. On the right crus cerebri, denotes the third pair; IV. The fourth pair; V. The trigeminus; VI. On the pons Varolii, indicates the sixth nerve; VII. Also on the pons Varolii, indicates the facial with the auditory nerve on its outer side; VIII. On the right lobe of the cerebellum below the horizontal fissure and the flocculus, indicates the group of nerves below the auditory—*i.e.*, the glossopharyngeal, pneumogastric, and spinal accessory; IX. On the upper part of the left amygdaloid lobe, denotes the hypoglossal nerve; X. On the same lobe, the sub-occipital nerve.

are intimately connected anatomically with the crura cerebri, especially with the crusta (by its posterior and external fasciculi), and the chiasma with the grey matter lining the third ventricle. And as the most external fasciculi of the crusta passes through the decussation of the pyramids into the posterior column of the cord, it is obvious that the posterior roots of the spinal nerves are represented in the same lobes of the hemispheres as the radiations of the optic tract (DODDS, *Journ. of Anat. and Phys.*, Vol. XII., p. 664, July, 1878). These tracts are represented in the occipital and temporo-sphenoidal lobes, in which are accordingly to be found the *visual* centres, as indicated by anatomy. "A more definite localisation cannot be given with certainty, but there is reason to believe in the correctness of Meynert's description, that the optic radiations pass to the cortex of the fusiform and lingual lobules" (Dr. W. J. DODDS, l. c.). Thus, (1.) the retina; (2.) the olfactory bulb and the posterior afferent, or sensory surfaces connected with the spinal cord, are each represented in the occipital and temporal lobes. The *third pair* of cranial nerves (Figs. 11 and 13, III.), or oculo-motor, and the *fourth pair* (Figs. 11 and 13, IV.), or trochlear, both arise in common from a pair of nuclei situated side by side in the corpora quadrigemina. The fourth nerves encircle the iter in their course, and then, winding round the outer side of the crura cerebri, reach the base of the brain (Figs. 11 and 13). Each third nerve penetrates the subjacent locus niger, and makes its appearance on the inner side of the crus. The other cranial nerves (Figs. 13 and 14)—namely, the fifth, the sixth or abducens oculi, the seventh or facial and auditory, the eighth or vagus, spinal, accessory, and glossopharyngeal, and the ninth or hypoglossal, are all traceable to nuclei in the medulla oblongata, where anatomical connections exist between those which have associated physiological action, and where connections are formed with strands of nerve fibres ascending either to the cerebrum or cerebellum. There seems to be no anatomical observations which indicate exactly the cerebral origin of the auditory nerve.

9. THE SPINAL NERVES consist of thirty-one pairs, each pair connected to the spinal cord by two roots, one of which roots, the anterior (*efferent* or *motor*), arises from the anterior aspect of the cord, having its immediate origin in the groups of large nerve cells contained in the anterior cornua of the cord, and they emerge from the surface of the cord in the fissure which separates the anterior from the lateral columns. The other, the posterior root (*afferent* or *sensory*), appears to originate in the posterior cornua, and makes its appearance superficially in the groove which divides the lateral from the posterior columns. After a short independent course, and the development of a ganglion on the posterior root, the two roots unite and form one trunk, which thereafter becomes a mixed nerve, carrying fibres from the anterior and posterior parts of the cord, to distribute themselves, by minute ramifications, into more or less active *end*-organs at the periphery, each filament remaining distinct in its whole course.

10. THE SYMPATHETIC NERVOUS SYSTEM consists—(1.) Of a pair of gangliated cords, situated one on each side of the spinal column; (2.) of three great ganglionated prævertebral plexuses, situated in the thoracic

and abdominal cavities; (3.) of numerous smaller ganglia, lying more especially in relation with the thoracic and abdominal viscera; (4.) of multitudes of fine communicating and distributing nerve fibres. Each *gangliated cord* extends along the side of the spine from the base of the skull to the coccyx in the front, by which the two cords are united in a single ganglion (*ganglion impar*). Each ganglion is united into a continuous cord of intermediate nerve fibres. In the sacral region there are five ganglia, in the lumbar region five, and in the thorax twelve; but in the cervical region there are only three—*superior*, *middle*, and *inferior*. Of these the superior is very large, representing several smaller ganglia; and from it proceeds an ascending or cranial offshoot through the carotid canal, whence it forms a plexus around the internal carotid artery (*carotid plexus*), and another in the inner wall of the cavernous sinus (*cavernous plexus*). By *communicating branches* the gangliated cord and its intercranial prolongation is connected with most of the cranial and with the anterior (efferent) divisions of all the spinal nerves. Thus the cerebro-spinal and sympathetic systems of nerves are brought into close anatomical and physiological relation with each other (TURNER). The fibres of the sympathetic system consist of two kinds:—(1.) Of grey or gelatinous fibres, usually called "*sympathetic fibres*," destitute of the white substance of Schwann; (2.) of medullated fibres similar to those met with in the cerebro-spinal system of nerves. The grey fibres originate chiefly in the ganglia of the gangliated cord, in which numerous ganglion cells are aggregated, and which are therefore regarded as the principal central organs of the sympathetic system. These cells of the sympathetic ganglia are surrounded by a capsule covered with squamous epithelia (FRÄNTZEL), and they give off a straight fibre and a spiral one, which twists round the former, whilst the tubular fibres are derived from the cerebro-spinal system. Each communicating branch contains both kinds of fibres, so that a double interchange of fibres takes place between the two systems. Thus, the *superior cervical ganglion* and its cranial prolongation communicate with the third and fourth nerves, with the Gasserian ganglion of the fifth, with the sixth, the portio dura of the seventh, the glossopharyngeal and pneumogastric of the eighth, with the hypoglossal or ninth cranial nerve, and with the anterior divisions of the four upper cervical spinal nerves. The *middle cervical ganglion* communicates with the fifth and sixth cervical nerves; the *inferior cervical ganglion* with the seventh and eighth cervical nerves; the twelve thoracic ganglia with the series of intercostal nerves; the five lumbar ganglia with the series of lumbar spinal nerves; the sacral and coccygeal ganglia with the sacral nerves and the coccygeal nerves. *Distributing branches* from the gangliated cord go—(*a.*) to the pharynx, joining the glossopharyngeal and pneumogastric nerves to form the *pharyngeal plexus*; (*b.*) branches to the vertebral articulations; (*c.*) *pulmonary* branches entering into the formation of the posterior pulmonary plexus; (*d.*) *vaso-motor branches*, or *nervi motor*, to the muscular coats of the arteries; (*e.*) *cardiac* branches to join the prævertebral cardiac plexus; (*f.*) *splanchnic branches* to the prævertebral solar plexus and to the renal plexus; (*g.*) *hypogastric branches* to the prævertebral hypogastric plexus.

The sympathetic nervous system is thus especially associated with the blood-vessels and with the thoracic and abdominal viscera, and is the medium of nerve supply to the involuntary muscular apparatus, both in the coats of the vessels and in the walls of the hollow viscera. The tract of distribution of the vaso-motor fibres has also been traced along with the anterior roots of the spinal nerves, through the lateral columns of the cord to the medulla oblongata, in which the vaso-motor centre is described as lying a little to one side of the mesial plane, above the calamus scriptorius (DITTMAR); but numerous centres are also seated in the grey matter of the spinal cord (VULPIAN). It is also important to note the termination of the sympathetic nerves, not only in the blood-vessels of glands, but in connection with the secretory cells themselves; and the communication between the cerebro-spinal and sympathetic systems, not only through the spinal nerves, but also through the pneumogastric, must be kept in mind in considering the effects produced by varying mental conditions on the secretions of the glands (TURNER, l.c., p. 313).

11. VASCULAR SUPPLY TO THE BRAIN.—Professor Turner observes, regarding the brain, that "it is a well-known axiom in physiology that organs which display great functional activity require and receive an abundant supply of blood, so that the work performed by the organ bears a relation to the amount of blood which flows through it in a given time. The brain ranks as one of the most vascular organs, for not only does it receive its blood through four good-sized arteries, the vertebrals and internal carotids, but these vessels communicate so freely with each other within the cranial cavity, as to permit of a ready transmission of blood to the different parts of the organ. In connection with the distribution of the blood to the substance of the brain, it is to be noted that by far the greater number of the blood-vessels penetrate the convolutions in their passage to the deeper parts. As they go through the grey matter they give rise to a very complete network of capillaries, which have been computed to be five times more abundant in the grey cortex than in the white core of the convolutions, a circumstance which substantiates the view that the grey matter is functionally much more active than the white. The arteries derived from the pia mater pass vertically into the grey matter, within which some terminate in a rich capillary plexus. Other vessels, of larger size, after giving off small branches, which join the capillary plexus of the grey matter, pass into the white matter, where they are continuous with a much more scanty capillary plexus than is found in the grey matter. The amount of blood which flows to a given brain, relatively to its size, is a point that ought to be kept in view, not only in connection with its own activity, but in comparing it with other brains. But the arteries of the brain, as elsewhere, are not merely to be regarded as tubes with simple elastic walls capable of dilating under the force of the blood pressure due to the heart's contraction, and recoiling when that contraction has ceased; but as pipes which possess in their own walls a musculo-nervous apparatus (vaso-motor), which accurately regulates the amount of blood transmitted through them and in accordance with the nutritive needs of the parts." The connections and arrangements of this vaso-motor apparatus in its relation to the physiological and pathological changes in the various arterial cerebro-spinal areas are of the greatest importance. The anatomy of those nerves on the blood-vessels and

capillaries has been beautifully illustrated and described by Dr. L. S. Beal, to the Royal Society (May 7, 1863), when he published drawings in the *Philosophical Transactions*, and again, in 1871, when he read a paper to the Royal Microscopical Society, in which he collected his observations together (*Monthly Microscop. Journal*, Jan., 1872). He has traced nerve fibres distributed to the finest capillaries from *ganglia*, from sensitive and motor nerve trunks, from the peripheral ramifications of sensitive and motor nerves, and internally related to the ultimate ramifications of some of the nerves of special sense. His observations also led him to the conclusion that in no case do the finest terminal ramifications of the nerve fibres end in or upon the ultimate structures which they influence; and that the action of the nervous influence is not dependent upon continuity of substance between the nerve and the tissue which it influences. The nerve only runs *near* it, but is distinctly separated from it. The late Professor Laycock also especially pointed out the important relations of this vaso-motor apparatus to the various pathological and physiological changes in the nervous system (*Medical Times and Gazette*, August 19 and Sept. 2, 1871). But the vaso-motor relations of the nerve centres to each other have still to be determined: and the functions of the sympathetic ganglia have still to be made out. There are certain ganglia of this system—*the cervical ganglia*—which regulate the activity of the blood-vessels that supply the encephalon. They have not only to do with the carotid and vertebral systems of blood-vessels, but also with the thyroid body and its relations to the blood-supply of the brain and spinal cord. The thyroid body has also very complex trophic functions, as shown by its twofold blood and nerve supply. Branches of the system of the superior cervical ganglion (*nervi molles*) accompany the arteries of the carotid system. They also accompany the *superior* thyroideal artery as a limb of that system, whereas the *inferior* thyroideal artery, which belongs to the system of the vertebral arteries, receives its nerve fibres from its own ganglion—the middle cervical. It is therefore concluded that as the innervation and arterial supply of the thyroid gland are double, the functions are double. There can now be no doubt that the thyroid gland must be regarded as an agent in regulating the intracranial circulation — an agency which is apparent in the fact that when under excessive muscular exertion the veins of the neck are compressed, and there is danger of venous congestion in the brain, the thyroid gland also becomes swollen by the strong influx of arterial blood (GUYON), and so compresses the carotids, from itself being firmly held against the vertebral column by the contraction of the muscles of the neck (MAINGIEN, NOTHNAGEL). Hence the danger of further distension of the intracranial vessels through the carotids is diminished. That these functions are in connection with those of the other cervical ganglia is probable from the intricate and intimate anatomical relations of the whole chain. The late Professor Laycock seems to have been the first to call attention to the clinical relation of these facts to goitre and cretinism; to vascular bronchocele, and that combined with cardiac neurosis in the form of "Grave's disease;" and to the functions of the ovaria and uterus (*Mind and Brain*, 2nd Ed., p. 476). The blood supply of the nervous system must therefore be the subject of clinical observation in regard to trophic and vaso-motor areas.

A summary of the trophic distribution of the blood-vessels of the nervous system is given as follows, mainly from QUAIN'S *Anatomy*, p. 576:—The *medulla oblongata* and *pons Varolii* are supplied by branches from the anterior spinals, the vertebrals, the basilar and the posterior cerebrals, entering the pons and medulla in two sets of branches,—lateral or radicular, and median—the latter passing in the septum to the grey matter on the upper surface.

The Cerebellum.—Its *under surface* is supplied by the posterior inferior cerebellar arteries from the vertebrals, and the anterior inferior from the basilar. Its *upper* surface is supplied chiefly by the superior cerebellar arteries from the basilar. Its *posterior portion* from the posterior inferior cerebellar. The arteries of the cerebellum form very rich anastomosing networks in its pia mater, from which small vessels pass into its cortex.

The Cerebrum.—(*Convolutions, outer surface, frontal lobe*)—The superior frontal and anterior two-thirds of the middle frontal convolution, with the upper extremity of the ascending frontal, are supplied by the anterior cerebral. The inferior frontal convolution, the posterior extremity of the middle frontal, and the greater part of the ascending frontal convolutions are supplied by the middle cerebral. The orbital surface is supplied, outside the orbital sulcus, by the middle cerebral: within that sulcus (including the olfactory bulb) by the anterior cerebral—the smaller of the two, which anastomoses with its fellow by the anterior communicating artery. Its trunk then turns round the anterior edge of the corpus callosum, and running backwards along the upper surface of that commissure, divides into three principal branches, distributed as already mentioned, as well as to all that part of the inner aspect of the hemispheres which lies between the area of distribution of the first branch and the ascending limb of the fronto-parietal fissure, including the anterior two-thirds of the corpus callosum.

Parietal Lobe.—All the convolutions of the parietal lobe are supplied by the middle cerebral artery; which also, by a number of small vessels, supplies the island of Reil. The middle cerebral artery divides, in the fissure of Sylvius, into four branches, which, radiating within the short convolutions of the island of Reil, supplies them with vessels, and emerges on the *outer* surface of the brain, and are thus distributed:—The anterior branch ramifies over the third frontal convolution only; a second is distributed to a portion of the second frontal convolution, and to almost the whole of the ascending frontal; a third supplies mainly the ascending parietal and superior parietal convolutions—the posterior and lower limit of its distribution being indicated partly by the inter-parietal fissure, and partly by a horizontal line drawn from this to a point on the upper margin of the hemisphere midway between the parieto-frontal and parieto-occipital fissures; a fourth or posterior branch is distributed to the first and second temporal convolutions, and to the gyrus angularis—its posterior limit being determined by a line drawn from the posterior extremity of the second temporal sulcus to the parieto-occipital (BRISTOWE).

The *Occipital Lobe* is supplied by the posterior cerebral artery, which results from division of the basilar, and sending branches into the brain substance at the locus perforatus posticus, it is joined by the posterior communicating artery of the same side, and finally gives off three principal branches—anteriorly to the uncinate convolution; a middle to the third temporal and lateral occipito-temporal convolutions, and to the hinder part of the gyrus fornicatus; and a posterior branch to the median occipito-temporal convolution, which connects the occipital lobe.

Temporo-sphenoidal Lobe.—The superior and upper part of the middle temporo-sphenoidal convolutions are supplied by the middle cerebral artery; the lower portion of the lobe by the posterior cerebral.

Inner Surface.—The whole anterior and upper portion, as far back as the parieto-occipital fissure, is supplied by the anterior cerebral artery; the cuneate lobule and the occipito-temporal region by the posterior cerebral.

The *Grey Substance at the base* of the cerebrum is supplied by small twigs from the adjacent vessels of the circle of Willis, or the commencing cerebral vessels.

Central Parts—Corpus Striatum.—Both nucleus caudatus and nucleus lenticularis are supplied almost exclusively by the middle cerebral artery, which has a wide and important distribution, and is "the artery of cerebral hæmorrhage" (CHARCOT)—the anterior part of the caudate nucleus only being supplied by the anterior cerebral.

The *Optic Thalamus* is supplied by the posterior cerebral artery. It gives also branches to the choroid plexuses and ventricular walls, and the tegmentum. Its inner and outer portion are supplied by the middle cerebral.

The *corpora quadrigemina* and *corpora geniculata* are both supplied by the posterior cerebral artery.

The observations of M. Duret point to the fact that the limits of the distribution of each cerebral artery is very definite and constant to a certain district or area (*Journ. of Anat. and Phys.*, Nov., 1874, p. 201). The study of the vascular supply to the nervous system from this point of view is obviously of great importance in relation to the pathology of embolism, thrombosis, and disease of the cerebral arteries, to apoplexy, epilepsy, and hysteria, and to trophic lesions of the brain (see also LAYCOCK, *Mind and Brain*, 2nd Ed., Vol. II., p. 473). The circle of Willis may be regarded as the only commissural connection of the cerebral blood-vessels. Except through this circle little or no communication takes place between the branches of the cerebral arteries—down to their finest terminal twigs, except by capillary vessels amongst the finer ramifications. Hence if any artery become obstructed, the area to which the obstructed branch is distributed suffers throughout its whole extent, and trophic changes take place as nutrition becomes impaired. The arteries which pass from the pia mater into the cerebral substance have a special arrangement in the grey and white matters of the convolutions. Those which go to the white matter (medullary arteries) spring at right angles from small branches in the pia mater; and, in the section of a convolution of medium size, ten to fifteen may be seen passing in a straight line perpendicular to the surface. Within the grey layers the vessels pass off from the larger ones nearly in the direction of the blood stream, and bend so as to become parallel to the principal bundles of white matter; others follow the direction of the commissural fibres of the convolutions. Near the sulcus a group of five or six arterioles diverge for distribution amongst the white matter of the more distant parts of the centrum ovale.

But the provision for the removal of venous blood is not less essential to cerebral nutrition and activity of function than the supply of arterial blood. M. Duret has shown that the arrangement of the veins of the pia mater is such that a free communication exists between those of the convexity of the hemispheres and those of the base of the brain through the medium of a large vessel—"*the great anastomotic vein*" (of Trolard, who first described it). It passes from the superior petrorsal sinus to the fissure of Sylvius; and another branch of anastomosis is found in the *anterior basilar* vein, springing from the great anastomotic vein, and joining the posterior basilar vein, which ends in the straight sinus. A true venous circle is also often seen within the circle of Willis, completed by the posterior basilar vein communicating with its fellow of the opposite side. The cerebral and cerebellar veins all converge directly or indirectly to the lateral sinuses, so that nearly all the blood of those parts is returned by the internal jugular veins. The ophthalmic vein empties itself into the cavernous sinus on the one hand, and at its peripheral extremity it receives branches of the facial and other veins. The longitudinal sinus communicates with the veins on the exterior of the skull through the parietal foramen, and the lateral sinuses with those of the head and neck through the mastoid foramina. The intracerebral veins spring from small venous branches in the pia mater, and do not exceed six or eight in number in a section of a medium-sized convolution. They do not accompany corresponding arteries, and have a calibre about three times as great as the arteries. The medullary veins pass deeply into the centrum ovale, and communicate with those of the ventricles (*Journal of Anatomy and Physiology*, Nov., 1874, p. 203).

The circulation of blood within the cranial cavity is peculiar in many respects; and now it seems an established fact that the aggregate amount of blood within the cranium at any time is subject to variation. Its quantity is not constant. In the cavity of the skull, just as in other organs, changes take place, not only in the relative amounts of the arterial and venous blood, but also in the aggregate quantity. Changes in the fulness of the cranial vessels have been actually seen to occur in living animals. Dundas, Kussmaul, and Turner have trepanned the skulls of animals, and then fitting tightly in the opening so made a piece of glass, they were able to see the veins of the pia mater dilate and contract under the influence of intermittent pressure made on the veins of the neck of the animal. It is also to be noted that in young children, before the fontanelles are closed the variations of circulation and of cerebral pressure are obvious phenomena recognised by the pulsations in the region of the fontanelle, which are of two kinds—(1.) Those coinciding with the ventricular systole, produced by the pulsations of the arteries at the base of the brain; (2.) those coinciding with expiration. The main peculiarities of the circulation within the cranium is due to the fact "that the contents of the skull are practically incompressible by the forces present in our organism, while, at the same time, they lie inclosed, air-tight, in an inelastic capsule. This being so, it is plain that before any new quantity of matter can find its way into the interior of this capsule a corresponding quantity of its former contents must be displaced; and, conversely, the withdrawal of a portion of the matter formerly present must be made up for by the introduction of a similar quantity from without" (NOTHNAGEL). Hence there are means for regulating the circulation. These consist of—(1.) The anatomical arrangements and contents of the subarachnoid spaces, which communicate freely with each other, and with the space surrounding the spinal cord. These spaces contain fluid (cerebro-spinal), and when the cerebral vessels become distended the fluid recedes from the cavity of the skull to enter it again as the distension becomes less (MAJENDIE, LONGET, ECKER). (2.) The contents of the perivascular lymph spaces act in a similar manner; for there seems evidence to show that they have—along with the ventricles, the subdural and subarachnoid spaces, the vascular plexuses within the ventricles, the external blood and lymph vessels, and lymph glands of the head and neck—connections between them of some sort, the exact nature of which is unknown (*Journal of Anatomy and Physiology*, Nov., 1874, p. 198). In fact it has now been shown that a continuous serous system pervades the entire nervous system from the ventricles of the brain, the subarachnoid spaces, the sheaths of the vessels in the brain and spinal cord, to the extreme ramifications of the peripheral nervous system. Thus, when more blood than usual enters the brain, the perivascular spaces become obliterated, filling up again as the blood recedes (QUAIN, NOTHNAGEL). (3.) The thyroid gland (as already indicated) becomes an important diverticulum and mechanical agent in the regulation of the cranial circulation (LIEBERMEISTER). (4.) The peculiar arrangement of the cerebral sinuses, and the mechanism of the circle of Willis for the circulation and distribution of the blood, combine to prevent venous stasis, on the one hand, and cerebral anæmia, on the other. The different variations of the cerebral circulation must therefore be attended respectively by the following results:—(1.) If there is increase in the quantity of arterial blood, there follows displacement of the cerebro-spinal fluid and of the contents of the perivascular lymph spaces; or by elevation of the intracranial pressure, if the compensation thus made is insufficient. (2.) If there is venous stasis, the thyroid gland becomes a diverticulum, with consequent retardation of the arterial afflux. (3.) If there is diminution in the aggregate quantity of blood, it is followed by an afflux of cerebro-spinal fluid, by dilatation and flooding of the lymph spaces, and when such compensations are insufficient then diminution of intracranial pressure occurs. These details apply equally to the membranes of the brain as to the brain itself (NOTHNAGEL).

As to the arrangements by which waste products are removed from the brain nothing is definitely known, beyond the fact that in some diseases of the nervous system the urine is loaded with chemical products, presumed to come from the vital changes going on in the nervous centres.

II.—CHEMICAL COMPOSITION AND PROPERTIES OF THE BRAIN AND NERVOUS SYSTEM.

There are some grounds for believing that changes in the nerve centres are accompanied by chemical phenomena and the formation of waste products, resulting out of the decomposition or disassociation of the complex chemical substances which form the basis of the nervous tissues. But before we can appreciate any such changes in the living brain, it is necessary to know the properties of the chemical substances existing in it. And even when we do know this, we are not certain that the same proximate principles may manifest the same properties during life as after death. Elaborate reports have been made by Dr. Thudichum to Mr. Simon, the Medical Officer of the Privy Council, on the chemical constitution of the brain (New Series, No. III., 1874), and also by his assistant, Mr. Kingzett (*Chemical News*, Feb. 25, 1876, p. 79), with the following general results :—(1.) Brain substance contains a large quantity of *water* chemically combined with various substances so as to form a colloidal mass, thus permitting great "mobility" of ultimate particles. It also gives penetrability by liquid diffusion, while excluding porosity and its capillary effects. (2.) The brain contains a considerable amount of an *albuminous base*, which is not present in a liquid form, as in serum, but appears to be under the influence of substances peculiar to the brain: the exact condition of the albumen in the brain-matter has not been investigated. (3.) Certain substances are found in large quantities in brain-matter, which, however, are not peculiar to it. These may be arranged into three groups :—(*a*.) *Phosphorised* bodies, containing carbon, hydrogen, nitrogen, oxygen, and phosphorus, and in all of these bodies the phosphorus is in the form of phosphoric acid combined with glycerine; so that under various processes glycerine-phosphoric acid is produced. These phosphorised bodies also differ amongst themselves in the mode in which they contain the nitrogen and the acid radicals; and from this point of view Dr. Thudichum splits these bodies up into the following sub-groups :—*Kephalines*, *Myelines* and *Lecithines.* "The kephalines possess the tendency to be oxidised—oxidisability; the myelines are not easily changed by any agent or influence, and possess, therefore, stability; the lecithines easily fall to pieces—they are afflicted with lability." These phosphorised bodies have also a remarkable diversity of affinities; for example, alkaline affinities for acids, acid affinities for alkalies, and alkaloidal affinities for salts. These affinities are overcome by water in quantity; but the affinity for water is in turn overcome by such metallic oxides as those of lead, copper, manganese, iron, and the like. The inference is, therefore, that if these substances do exist as such in living brain-matter, we have an incalculable number of states or conditions of this matter: and, "it foreshadows on the chemical side the remarkable properties which nerve-matter exhibits in regard of its vital functions. From this it also follows, that nerve-matter (if only as characterised by the phosphorised bodies) must yield obedience to every, even the slightest, external chemical influence which may reach it by way of the blood. It must take up metals, acids, salts, alkalies, and alkaloids presented by the blood; it can retain only oxides when the serum is again free from the combinants; a watery serum will wash the brain, a more watery one will make it swell and displace mechanically, within physiological limits, what it can; a still more watery one will make the brain dropsical, and produce all the conditions of mechanical pressure on the brain."

(*b.*) *Nitrogenised substances*, containing four elements, but no phosphorus, such as *cerebrin*, *stearoconote*, *phrenosin*. As compared with the phosphorised bodies, their peculiarity is slight solubility; they are more stable compounds, and less liable to oxidation. Like the phosphorised bodies, they are colloidal, and do not pass through the septum of the dialyser. (*c.*) *Oxygenated principles*, containing only three elements, namely carbon, hydrogen, and oxygen. The chief of these is *cholesterine*. In this research no fewer than *eighteen* bodies have been discovered for the first time, as ingredients in brain-matter.

Such knowledge regarding the chemistry of the brain and cord must eventually aid in explaining the precise action of neurotic remedies. Hence the practical importance of prosecuting such investigations as far as possible. We know that certain substances act on the brain, others act on the cord; that some act on sensory centres, while others act on motor centres; and that some seem to act by a kind of catalysis, while others split up and disappear. We know nothing definitely as to the precise chemical changes which follow a dose of morphia, or of strychnia, or of arsenic, or of nitrate of silver, or of almost any other active substance. But "when we know the chemical constitution of the substances forming the nerve centres, their chemical affinities, their precise relations to oxygen, their influence over the matters dissolved in the blood, then we may hope to obtain a rational explanation of the action of our neurotics" (*Brit. Med. Journal*, Sept. 2, 1876).

Scarcely anything is known about the chemical constitution of nerves. The axis cylinder seems to be related in its characters to the albuminous bodies. The medullary sheath (whose appearance and behaviour towards solvents indicate a fatty nature) possibly contains no proper fat, but only lecithine ($C_{44}H_{90}NPO_9$) and protagon, the formula for which has not yet been determined (HERMANN'S *Human Physiology*, translated by Dr. GAMGEE, p. 36). Nerve fibres contain in addition cholesterine and creatine.

The chemical composition of ganglion-cells can only be imperfectly and approximately gathered from analysis of the grey substance of the brain. The white substance, which consists essentially of nerve fibres and the connecting neuroglia, is considered to be of the same composition as nerve trunks, the constituents of which have been mainly determined by examinations of the brain and spinal cord. While the reaction to test paper of the white substance is *neutral* or *alkaline*, the grey substance has been found to be *acid*—a circumstance which is probably to be attributed to the rapid changes occurring at the surface of section. The chemical constituents of the white substance are lecithine, protagon, and probably other lecithine bodies; albumen, potassium, albuminates, and globulins; cholesterine and fats; creatine, xanthine, and hypoxanthine; inosite and some anhydride of sugar; lactic acid and volatile fatty acids; salts and water. The grey substance of the brain is distinguished chemically from the white chiefly by containing more water, albumen, lecithine, and lactic acid, and less cholesterine, fat, and protagon (HERMANN, l.c.) The composition of grey and white brain substance is given in the following table (PETROWSKY):—

	Grey Substance.	White Substance.
Water,	81·6 per cent.	68·4 per cent.
Solids,	18·4 ,,	31·6 ,,
	100	100
Chemistry of—		
Albumens and gelatine,	55·4 ,,	24·7 ,,
Lecithine,	17·2 ,,	9·9 ,,
Cholesterine and fats,	18·7 ,,	52·1 ,,
Cerebrine,	0·5 ,,	9·5 ,,
Substances insoluble in ether,	6·7 ,,	3·3 ,,
Salts,	1·5 ,,	0·5 ,,
	100·0	100·0

The salts found are the same as those in the blood, the large amount of phosphates and potash being remarkable—one-twentieth to one-thirtieth of the whole solid matter. The following analysis is given of 100 parts of the salts obtained from cerebral matter (M'Kendrick, *Outlines of Physiology*, p. 154):—

Potash,	32·42
Soda,	10·69
Magnesia,	1·23
Lime,	0·72
Chloride of sodium,	5·74
Phosphate of iron,	1·23
Phosphoric acid, in combination ,.	39·02
Phosphoric acid, free,	8·78
Sulphuric acid,	0·75
Silica,	0·42
Total,	100·0

From the fact that the venous blood of the brain and spinal cord is so poor in oxygen and rich in carbonic acid (*i. e.*, as dark in colour as the venous blood of any other region of the body), there is every probability that processes of oxidation take place in ganglion-cells, as in all other organs. But we are quite ignorant as to whether such processes of oxidation are concerned in the activity of the ganglion cells, or if so to what extent. Nor do we know whether that activity is not dependent upon processes of decomposition similar to those which occur in muscles and nerves; or what the results of such oxidations or decompositions are. But during functional and morbid changes in the nervous system there can be no doubt that a large amount of the phosphorised compounds are continually being metamorphosed; and the amount of alkaline phosphates in the urine may still be regarded as in some measure an estimate of the amount of nerve-tissue disintegration (the earthy phosphates being disregarded, inasmuch as they have been shown mainly to depend on the quantity taken in the food). Hence one mode of elimination of the products of metamorphosis of the nervous system is, that the phosphoric compounds, set free by disintegration of nervous tissue, unite as an acid with alkaline bases in the blood, whence it is separated in the kidneys and discharged with the urine.

III.—PHYSIOLOGICAL RELATIONS AND FUNCTIONS OF THE SEVERAL PARTS OF THE BRAIN AND NERVOUS SYSTEM.

The cortex of the cerebrum must be regarded as the chief, if not the exclusive, seat of psychical activity; so that the functions of the cerebral convolutions are evidently psychical in nature. They intervene, and intervene only, in those operations of the nervous system in which an intelligent consciousness and volition play a part (Dr. M. FOSTER). The following are the principal reasons for this belief:—(1.) In the animal series the cerebrum is found to be more fully developed in comparison with the mass of the body, and with the encephalon as a whole, in those classes in which the individuals approach in mental powers the condition of man. The degree of development is judged of by the weight of the cerebrum, and by the number of gyri or convolutions. Hence, the purpose served by convoluting the surface of the cerebrum is to bestow on it a far greater superficial area of grey matter than is possessed by a smooth-surfaced brain. Hence, also, facts relative to depth of grey matter and depth of the sulci between the convolutions are of importance to be determined, as an indication of the proportion of nerve cells, or ganglion cells, in one brain as compared with another: and the differentiation of the surface into definite primary convolutions gives such precision to the method of its arrangement, as to map it out into more or less formal and recognisable areas, and so helps to bestow upon any definite part or group of nerve cells a local habitation and a name. It is only since the topography of the convolutions has been more precisely understood, that we have been in a position to institute a strictly scientific inquiry into their organology (TURNER). (2.) In cases where the cerebrum is abnormally small from birth (microcephalus, cretenismus), or where it is diseased (hydrocephalus, &c.), there is noticed a corresponding diminution in the higher mental powers, or idiotcy. (3.) Injuries, compression, and diseases of the cerebrum are almost always accompanied by mental disturbance, peculiarity of demeanour, insensibility, somnolence, or excitement; and inflammation of the membranes covering the surface of the brain (meningitis, especially if it involve the superficial layer of grey matter) causes delirium, and afterwards coma. (4.) Removal of the cerebral hemispheres induces a condition resembling that of sleep, in which all voluntary movement ceases. Nevertheless movements of a reflex nature occur in response to the stimulation of sense organs; but so regular is the order of their occurrence that they may be predicted. When the cerebrum is removed in slices, a gradual loss of all the powers of the mind is said to take place (FLOURENS). (5.) In some cases sudden depression of a portion of the cranium into a convolution, or a sudden blow on the head, has either arrested the mental faculties as a whole, or has affected the action of one only, as, for example, the memory. (6.) Pathological states of the grey matter, such as congestions, softenings, degenerations, or the formation of new products, are always associated with the various forms of insanity; but the exact pathological changes peculiar to each form—for instance, melancholia, as contrasted with mania or dementia—are still imperfectly known (HERMANN, M'KENDRICK). It has not yet been shown, however, that the convolutions are distinct organs, notwithstanding the claims at one time put forward by Gall, Spurzheim, and other writers of the school of Phrenology, to subdivide the surface of the hemispheres into areas or organs, each of which is the seat of a particular faculty. There are no such structural and functional differences amongst the convolutions as to justify us in regarding them as distinct organs. There can be no doubt, however, that the history of the progress of the physiology and pathology of the nervous system shows that our present knowledge owes its development to the mode in which those men attempted to prove the truth of the doctrines they taught. Their object was the same as that which is now considered most rational; and we are greatly indebted to a distinguished and accomplished physician, Dr. Samuel Wilks, of Guy's Hospital, for a "History of the Physiology of the Nervous System," in which he has not suffered the spark to be lost in the flame it has served to kindle (*Guy's Hospital Reports* for 1879, Vol. XXIV., p. 59). He shows that the phrenologists looked upon the brain as a compound and complex organ. They were the first to replace the old method of slicing the brain by unfolding and dissecting it; but, by too hastily framing a system, they provoked such discussions and investigations as have not only not ceased since 1825, but are being daily carried on with increasing energy and enthusiasm in every part of the world where anatomy, physiology, and pathology are cultivated. Aphasia was clearly recognised by them, and language they located in the region where physiologists are now agreed to place it. They held that the brain was double; that although its surface is the seat of mental processes, it had other properties attached to it. They also associated the will and the movements with the anterior parts of the brain. Solly also testifies, in his work on the brain, that Gall dissected and unravelled that organ with a significance that had never before been accomplished. In regarding the hemispheres as containing the organs of the Mind, he traced out the columns from below into the hemispheres, and so unrolled the whole mass in a systematic manner. Thus Gall gave an impulse and direction to the dissection of the brain altogether new. The late Mr. Hilton, of Guy's Hospital, seems to have been the first in this country to adopt the new method; and Dr. Wilks considers himself fortunate to have commenced the study of anatomy when the old method of "slicing" the brain to exhibit the *centrum ovale, majus* and *minor*, had been discarded. Instead of this he witnessed Mr. Hilton unravel the fibres of the brain, and trace them in their course in the beautiful manner which his preparations and wax models display so well to this day in the unrivalled Museum of Guy's Hospital. Spurzheim, the colleague of Gall, pursued the same method; and in 1804 Gall had shown the decussation of the pyramidal bodies—their passage through the pons—the existence of several layers of longitudinal and transverse fibres in the pons—the continuation of the optic nerves to the anterior pair of the corpora quadrigemina—the diverging of fibres in the brain, and other facts of a like kind. A knowledge of the use of the double brain is also to be learned from the phrenological writings; but it has not yet received the attention which its importance demands. The nerve centres are fused into one only where their action is common to the two sides; but are distinct where their actions are independent of one another, as in the limbs. "And as we have two halves of the body, in part united and in part independent, so we have two brains, in part acting together and in part acting alone. . . . The independent limb and the independent brain are correlative and mutually connected." Volitional acts may thus proceed from each side of the brain; and if these are associated with sensation and mental operations, the latter also can take place on each side separately. The double nature of the brain also affords an explanation of the great amount of disease which may affect one side of the organ without markedly interfering with the intellectual faculties; also how, in a not uncommon form of hysteria, the patient will lose the power of motion on one side, as well as feeling, while the special senses at the same time are dulled; as if one side of the brain had become suddenly inert, the patient all the while retaining her consciousness, and the other half of the body its full activity. From the brain being thus made up of two halves, it is instrumental in affording us a much wider view of the external world around us. The connection between the two sides is most intimate, so that their working results in a single consciousness, which hides from us the important fact that our bodies are to a great extent double, and therefore our brains and senses double also. For the most part we see double: we do not see single, except by such parts of the retinæ as are associated together by means of the optic chiasma. We look at objects from different points of view, and merely place the images side by side. So also do we discriminate sounds with both ears, taste different substances at the same time on each side of the tongue, and receive distinct impressions of each of two different objects touched by each hand. A consideration of man's body as thus made up of two animal frames combined, and therefore coming in contact with its surroundings in two different aspects, will give a clearer insight into the functions and attributes of the body, than if we regard it merely as a single mass. At all events, it seems clear that a double body needs a double brain; or, at least, that body and brain should correspond in every respect. It seems also evident, that the separation of the hemispheres has an intimate relation to the independent action of the limbs, since disease in one of them produces a lateral paralysis, and an irritation of its convolutions a corresponding spasm. Not only does each side of the brain rule over the movements of the limbs, but when in action produces also mental phenomena—explaining, to some extent, how the instincts of animals are closely allied to the forms and uses of their extremities; and so naturalists have wisely classified animals according to the forms of their limbs, as these imply a corresponding habit of the animal, both limb and habit being closely represented in the convolutions of the brain (WILKS, l. c., p. 66). But although many convolutions exhibit special and constant forms, yet they are not so individualised as to be disconnected from each other. The grey matter on the surface of one convolution forms invariably a continuous layer with the grey matter of the surface of all the convolutions which immediately surround it; so that if a convoluted brain were unfolded, it would present a uniform and continuous surface of grey matter similar to a naturally smooth-surfaced brain. Modifications do undoubtedly occur in various of the convolutions as to the size, the relative numbers, and to some extent also as to the shape of the nerve cells; and in so far such modifications would tend to support the view that functional differences may exist between them, were it not that such modifications pointed rather to differences in the degree than in the kind of action which takes place in the convolutions—that the differences are quantitative rather than qualitative. There is also evidence that differences exist in the connections which are established between the convolutions and the great ganglia of the base of the brain, and between the different convolutions themselves, through the agency of the internuncial nerve fibres which form the white core of each gyrus; and in the differences in their commissural connections we may look for an agreement in favour of the existence of functional differences. Similarly, the fact that communications are established between certain convolutions and not between others, points to the inference that certain gyri are not only anatomically but physiologically associated directly with each other. It is also possible, not only that particular combinations of convolutions may condition (through an interchange of commissural fibres) a particular state of intellectual activity, but that these combinations associate various convolutions together in the performance of a given intellectual act. (TURNER, l. c.) Hence, a study of the deeper connection of the convolutions appears to Professor Turner to offer a more promising line of inquiry in its bearings on the question of the functional independence of the convolutions, or of groups of convolutions, than an investigation into the arrangement and physical properties of the nerve cells. Finally, Professor Turner concludes that,—

> "To some extent data have now been collected which show that some men of high intellectual capacity have possessed brains the convolutions of which were complex in arrangement from the increased development of the secondary and tertiary gyri, and from the greater tortuosity of the convolutions generally. The brains of the eminent mathematicians Gauss and Dirichlet, so beautifully figured by Rudolph Wagner, are illustrative examples. Careful measurements of the convolutions, conducted by Hermann Wagner, have shown that the superficial area of the convolutions in the brains of Gauss and of Professor Fuchs was considerably greater than in an ordinary man or woman. But, as Wagner has himself pointed out, great intellectual activity is not of necessity limited to brains which possess great complexity in the arrangement of the convolutions; and he adduces, in illustration of this fact, Hermann the philologist and Hausmann the mineralogist, in neither of whom were the brain weights above the average, nor the convolutions arranged in a complex manner. Thus, the convolutions may, to a considerable extent, vary in size in the human brain without their functional activity being appreciably affected; though we do not possess the same numerical data to determine what the minimum size may be which is compatible with intellectual vigour, as we do in the case of the minimum weight. This, however, we do know, that when reduced to a state of tenuity from atrophy or congenital mal-development, serious mental defects are occasioned. On the other hand, it by no means follows, when the convolutions are normal in size and shape and relative position, that they should be perfect in function. For not only may we find the convolutions of the brains of the insane well formed and normally arranged, but even in idiots and imbeciles, where there is a positive absence of intellectual power—though this serious defect is not unfrequently associated with some grave error in development—the convolutions have in many cases possessed their normal form and arrangement. Hence it is clear that a correspondence in morphological configuration by no means necessitates either equality or similarity in functional power.

In estimating the value of the convolutions, therefore, either when the brains of men are compared with each other or with those of animals, other factors are to be considered than those afforded by size, or weight, or form, or modes of arrangement. It is generally admitted that the nerve fibres, whether they occupy the white core or the grey cortex, are conducting rods, protected by an insulating investment—the white substance of Schwann—just as the copper wires in a telegraph cable are surrounded by insulating substances; whilst the characteristic cells of the grey cortex are the structures in which the molecular changes occur which occasion the evolution or disengagement of the special form of energy named nerve-energy or nerve-force."

It may be generally accepted that cerebral functions are essentially founded upon the type of reflex actions (LAYCOCK, LUYS). The typical anatomy of the arrangement for such functions are seen in their simplest form in "the much referred to ascidian," in which two sets of fibres meet in a centre or ganglion. The one fibre is *afferent*, the other *efferent*—the ganglion receiving the impression and emitting nerve force. This simple mechanism thus consists of a centripetal fibre, a sensory cell, an intercentral fibre, a motor cell, and a centrifugal fibre, through which all nerve operations are carried on. This simple mechanism becomes more complex as the scale of life passes upwards to man, and so the same type is maintained throughout. In the higher grades, the greater specialisation implies an increase of ganglia and additional central connections, with ever-increasing complexity and more complete integration. In man the parts of the spinal cord are connected with each other by communicating fibres; and are connected also with higher ganglia in the brain by fibres which pass directly to them from below. The lower centres are co-ordinated with the nervous mechanism of other organs in these upper ganglia; and in this way, besides greater control of the functions of inferior centres by the higher, and the possibility of greater unity of action, there is also the capacity of greater specialisation of movements. The general plan of construction of the brain is thus in harmony with the belief that there are in it arrangements for successive integrations and differentiations of function between the cord and the convolutions, in the great ganglionic masses that intervene, particularly those of the medulla and pons, the ganglia in or near the lateral ventricles, and those in the cortical grey matter itself (Dr. ALEXANDER ROBERTSON, *Glasgow Medical Journal*, No. XII., Dec., 1879). This doctrine of increasing specialisation, associated with integration, to be seen in the progression from inferior to upper ganglia, attaining its highest degree of completeness in the supreme centres of all—the hemispherical ganglia—has important bearings regarding the increasingly important question of localisation of functions in the convolutions. On this subject, Dr. Hughlings Jackson has come to the conclusion, from the study of cases of disease, that "the higher centres are evolved out of the lower, receiving intercalations as they ascend from the spinal cord to the cerebrum. The higher centre re-represents *more especially* the impressions and movements already represented, generally in the one below it. The co-ordinations are continually being re-co-ordinated; for example, those of the pons and medulla are re-co-ordinated in the cerebrum. There are, in the lower centres, sensori-motor processes for very *general* purposes, but in *their* higher representatives for the more special. A rude symbolisation would be to suppose the pons Varolii to represent the simple sensori-motor processes of the cord raised to the fifth power, and the cerebral hemispheres those processes suddenly raised again, let us say, to the fiftieth power." Hence even in the highest centre of all there may be localisation of function. The merit of having first (in 1864) distinctly propounded the proposition that there are definite motor centres for associated movements in the convolutions of the entire brain belongs to Hughlings Jackson. His conclusions were based on the observations of disease; and it is now a matter of history how they have been supported by the discovery, in 1870, of Fritsch and Hitzig, that certain portions of the brain's surface were sensitive to electricity, and that there existed certain motor centres in the cerebral cortex; and now these statements have been confirmed, and at the same time modified by the valuable experiments of Ferrier since 1873, and others in this country. The experiments have been made (using the Faradic current) on dogs, monkeys, jackals, cats, rabbits, guinea-pigs, and rats—also on pigeons, frogs, and fish. These observers found that the local application of the current to particular areas on the surface of the convolutions gave rise to definite co-ordinate movements of various groups of muscles. Stimulation of one spot was followed by movements in the muscles of the neck; of another, by extension of the fore-leg; of a third, by movements of the hind leg; and of a fourth, by movements of the eyeballs and of the muscles of the face. Ferrier, especially, has mapped out definite areas, the stimulation of each of which causes precise movement; and he has also found convulsions to result from simultaneous stimulation of several. These areas have been termed *motor centres;* and it has been supposed by some that influences pass from them to special groups of muscles in the phenomena of voluntary motions. Other observers have ascertained that removal of one of these areas of grey matter is followed by paralysis or weakening of the muscles which contract on the stimulation of the area. The term "motor centres" has been an unfortunate one, as it is shown to be misleading, "since it suggests that the brain-surface in a given area is largely occupied in giving rise to the co-ordinate nervous impulses, which carry out the movement resulting from stimulation of the area, just as the respiratory centre, for instance, is occupied in giving rise to the co-ordinate respiratory impulses. The real interest in the results of electric stimulation of the brain-surface, attaches not so much to the question as to which are the exact movements resulting from the stimulation of this or of that area, as to the broad fact that different results follow upon stimulation of different regions, thus serving to indicate that there is after all 'a localisation of functions' in the brain-surface" (Dr. M. FOSTER, *Text-book of Physiology*, p. 573). The only case of direct electrical stimulation of the cortex of the human brain is one in America recorded by Bartholow (*American Journ. of Medical Sciences,* April, 1874). A cancerous ulceration had exposed the surface of the brain. Stimulation of a part (shown, after death, to be the postero-parietal lobule) was attended by movements of the extremities of the opposite side, and by less definite movements of the neck, orbicularis, and pupils. An increase in the strength of the current produced epileptiform convulsions, beginning in the arm. The next day the patient had another attack—now on the same side as had been stimulated—followed by complete hemiplegia. So far as the movements of the leg and arm are concerned, the case is in agreement with the results of stimulation of corresponding regions in the monkey; but the coincident presence of other movements, and the fact that subsequent epileptiform convulsions and hemiplegia were not on the opposite side, as we should have expected, take away much from the value of its evidence (DODDS, l. c., p. 345; FERRIER, *on Functions of Brain*, p. 296).

It is now generally admitted that electrical irritation of certain parts of the brain is followed by movements, often of a localised and special character; that on the electrisation of certain convolutions, particularly those bounding the fissure of Rolando, uniform and definite purposive movements always ensue; and also, that no movements of any kind are induced by stimulating most other portions of the cortex outside the proper motor area, such as the fore and back part of the brain in monkeys. But in spite of some discordance among various authors as to the exact position and extent of the several "areas," that there is a connection between electric stimulation of certain areas of the brain-surface and certain bodily movements must be regarded as fully established. Doubt still remains as to the interpretation of them; and the exact nature of the connection is still very obscure.

It is not yet so certain, however, that the effects of such stimulation may be so localised as to call forth simply the function of the part operated on. An important objection has been raised against Dr. Ferrier's method in the fact that diffusion of the currents forms a possible explanation of some of the phenomena of brain electrisation; and that the danger of this must be directly as the tension of the electricity used. Dr. Ferrier has, however, fairly met this objection; and urges that if the movements following electrical stimulation of the surface were due merely to conduction to the corpus striatum, and not to vital excitation of the cortical motor centres, then these movements should be of a general character, and involve the whole of the opposite side of the body, such as follow the immediate stimulation of those great centres; whereas, on the contrary, these movements are highly differentiated. He also puts the question,—How, on the theory of simple electrical conduction, is there no muscular action when the electrodes are applied to the island of Reil, which is so much nearer the corpus striatum than most parts of the motor convolutions? Nevertheless, as Dr. Dodds observes, what strikes one most in performing any of these experiments, is that they have amply demonstrated that stimulation of certain parts will very generally produce definite movements; and that similar stimulation of adjacent parts is followed by no movement; and that general diffusion may, with sufficient precaution, be effectually guarded against if minimal currents only be used. The effects of the stimulation are those of a local action, whatever its nature may be—an action which affects either the grey matter alone, or both grey and white matter, or the white matter alone, by which its action is propagated downwards. It has been shown experimentally (BURDON-SANDERSON, CARVILLE, DURET, and HERMANN) that the phenomena can be called forth without the agency of the grey matter. Hence the question arises,—Are the results due to a vital excitation of the cortex, or to an excitation of some deeper part by extrapolar currents conducted down the fibres of the corona radiata? The special facts of brain stimulation seem, however, to be unaccountable on the theory that they are produced by extrapolar currents; and the cortex must be regarded in one sense as an end-organ most delicately adapted for psychic functions. Specialisation of function attains its highest degree in the convolutions. Nerve-cells are there combined for acts which require particular training, and which are habitually executed at the instigation of the will. It is probably the most sensitive of all the nervous ganglia, being most easily affected by feeble streams of nerve force; and the experimental phenomena are, in the case of minimal electric currents, due to the excitation of the nerve force in the nerve fibres or cells; but whether the irritation of the cortex acts on its cells as well as on its fibres is not yet known. Hence, also, comes the question,—Are the effects of the stimulation of the so-called "motor" areas due to the stimulation of motor or sensory cells?—the movements in the latter case being of a reflex character. Destructive lesions of the cortex illustrate, to some extent, the significance of the "centres" or "cortical areas." Ferrier is the chief advocate of the motor functions of the cortical centres; others regard them and the cortex generally as purely sensory, having sensory centripetal and sensory centrifugal fibres connected with the same centre. But, generally, the evidence rather tends to show that the so-called "motor" centres are either directly motor, or are so closely connected with motor cells governing corresponding movements, that we can scarcely but regard them as motor. The chief difficulty in the acceptance of the motor view is founded on the fact, that after extirpation of the centres in many animals there is rapidly and apparently complete recovery. Disease destroying centres in man has been also followed by recovery. An explanation is furnished by one or other of the following hypotheses: that the function is taken up (1.) by the corresponding part of the opposite hemisphere; or, (2.) by some part of the same hemisphere; or, (3) by some of the lower centres. Still there is much to be said in favour of motor and sensory elements being associated in those parts of the cortex; and, generally, as to the nature of the cortical centres, the opinion arrived at by Dr. Dodds in his admirable critical review of the localisation of brain function, is that the phenomena are most satisfactorily accounted for on the theory that the centres are motor centres; and he agrees generally with Dr. Hughlings Jackson, that it is impossible to "conceive of what other materials the *organs of mind* can be composed, than of processes representing both movements and impressions" (l. c., p. 462). Looking to the anatomy of the brain, it has been shown that certain regions are distinguished from others by broad differences in structure; hence it may be fairly inferred that such structural differences have their counterpart in a diversity of functions. The so-called giant cells in the brain are chiefly found in the motor region of the experimentalists; the convolutions on either side of the fissure of Rolando at the vertex—in the ascending frontal and ascending parietal convolutions. In this position the cells of the fourth layer are very large (BATTY TUKE). Thus the evidence of anatomy and physiology, while it lends support in many ways to the localisation of centres held by Hitzig, Ferrier, and others, points at the same time to a localisation and interconnection of centres very much more complicated than any yet indicated by physiological experiment (DODDS, l. c., p. 659). Another most competent authority, Dr. Alexander Robertson of the City Parochial Asylum of Glasgow, considers the evidence sufficient to establish the two following positions:—(1.) Though it cannot be held that the theory of motor centres in the convolutions is fully established, yet it is highly probable that there are such centres in what is known as the motor region. (2.) It is probable that there are also separate centres for special and general sensation, but their locality is much less certain than the locality for the centre for movements (*Glasgow Med. Journ.*, Dec., 1879).

The region between the surface of the cerebral hemispheres and the medulla oblongata contains a number of extensive grey masses—the ganglia at the base of the brain—and a highly complicated system of white fibres, the general distribution of which has been already noticed. These "basal ganglia"—the corpora striata anteriorly and thalamic optici posteriorly—are undoubtedly the great means of communication between the cerebral hemispheres, on the one hand, and the crura cerebri on the other. Though some fibres do pass from the crura by or through the ganglia to the cerebral convolutions without being connected with the nerve cells of those ganglia, the great mass of the peduncular fibres are probably connected with the superficial grey matter of the hemispheres in an indirect manner only, the lower or anterior, or motor fibres (*crusta*), passing first into the corpora striata, and the upper or posterior fibres, or sensory (*tegmentum*), into the optic thalami. This anatomical disposition might indicate that these bodies have important functions in mediating between the psychical operations of the cerebral convolutions, on the one hand, and the sensori-motor machinery of the middle and hind brain on the other; and the separate courses taken by the peduncular fibres would further lead us to expect that the functions of the corpora striata differ fundamentally from those of the optic thalami (Dr. M. FOSTER); and that the optic thalami were ganglia of the sensory tract, and the corpora striata of the foot or motor tract. The ganglionic substance of the corpora striata clusters round the peduncular expansion in the form of two great masses on either side, the one of which, projecting into the centre of the lateral ventricle, receives the name of the *nucleus caudatus*, or intraventricular nucleus; the other, situated externally in immediate proximity to the island of Reil, receives the name of the *lenticular ganglion*, or extraventricular nucleus of the corpus striatum. The peduncular fibres and the ganglionic substance must be regarded as one complex whole, impossible to be differentiated. In like manner, the medullary fibres which converge to or diverge from the optic thalami are apparently distributed in the posterior and temporo-sphenoidal regions of the hemisphere (FERRIER, l. c.). But these ganglia are also not only in communication with the grey substance of the cord and medulla (and through these with almost all the peripheral organs of the body), but are also connected with the nerves of the higher senses, whereby they are concerned in much more manifold and complicated centripetal excitations than are the simpler reflex apparatuses of the cord. Hence they are the seat of reflex operations and co-ordinations of correspondingly greater complexity; for the complexity of the efferent (centrifugal) impulses to activity must increase with the number of afferent (centripetal) stimulations. The region has also the power of inhibiting reflex action, and also of adjusting equilibrium. The participation of the higher nerves of special sense in the functional powers possessed by the ganglia at the base of the brain, may be exemplified by the objects in the field of vision, as well as the muscular sense of the muscles of the eye powerfully influencing our movements; also, that the auditory nerve is most probably connected with peripheral apparatuses which serve to explain the position of the head. The effects of experiments and lesions of these parts are as follow:—(1.) Unilateral lesions may give rise to extremely abnormal movements—"*uncontrollable movements;*" the chief forms of which are—(*a.*) movement forwards at the periphery of a circle—"circus movements;" (*b.*) rotation about the longitudinal axis of the body—"rolling or wallowing movements;" (*c.*) movement of the anterior portion of the body about the fixed posterior portion. Such phenomena follow injuries to the corpora striata, optic thalami, crus cerebri, pons Varolii, and certain portions of the medulla oblongata and cerebellum. Injuries to the conducting paths may, under certain circumstances, result in similar movements, and so also may galvanism of the head, which at the same time causes dizziness (PURKINJE, HITZIG, quoted by HERMANN, l. c., p. 506). "When a lesion occurs in the human subject which involves both the corpus striatum and thalamus opticus on one side of the brain, the result is a loss of sensation in, and voluntary power over, the opposite side of the body and face—a hemiplegia—which may be absolutely complete without any impairment whatever of the intellectual faculties. The will and the power to receive impressions are present in their entirety, but neither efferent nor afferent impulses can make their way to or from the peripheral organs and the cerebral convolutions (except through the hemisphere which is whole). The injury to the basal ganglia (on the one side) blocks the way. In the majority of cases the *anæsthesia* (loss of sensation) and *akinesia* (loss of movement) are absolutely confined to the opposite side of the body; and the cases in which a lesion of the basal ganglia of one side of the brain affects the same side of the body, or both sides, must be regarded as exceptional, and explicable as the results of the action of one side of the brain on the other side, either of the brain or of some region of the cerebro-spinal axis. Experiments and clinical observations conclusively show that lesions of the corpora striata and optic thalami produce their effect on the opposite side of the body; but the present state of information does not enable any dogmatic statement to be made as to the nature of the mediation which either the corpus striatum or thalamus opticus effects between the cortex of the hemispheres and the rest of the brain. We only know that they do so act as media between these parts (Dr. M. FOSTER, p. 584). The thalami optici do not admit of experimental investigation without the most extensive injury of other parts of the brain; and as lesions of the optic thalami produce the peculiar movements already noticed, it is supposed to be through the optic thalami that the eye influences co-ordinated movements. The functions of the optic thalami are also concerned with the conscious perception of visual impressions. Facts are wanting as to the functions of the corpora striata and lenticular nucleus. Lesions of the lenticular nucleus invariably cause hemiplegia (HERMANN, *Human Physiology*, p. 507); and destructive lesion, generally, of the corpus striatum, produces hemiplegia of the opposite side, sensation remaining uninterrupted—one of the best established facts in human pathology (FERRIER, l. c., p. 237). Electrical stimulation in animals, by Ferrier, causes unilateral tonic contraction of the muscles of the face, neck, trunk, and limbs—a condition of pleurosthotonus in which the body is bent to the opposite side, and the limbs maintained in a position indicating the predominance of the flexor over the extensor muscles, an experience confirmed by Carville and Duret (FERRIER, l. c.) Electrical irritation of the optic thalami causes no motor manifestations; but there is conclusive proof of the abolition of cutaneous sensation by an injury in and round this ganglionic mass; and as regards the consciousness of sensory impressions, it has been most conclusively shown that hemi-anæsthesia occurs in man when the posterior parts of the peduncular expansion, or internal capsule external to the optic thalamus, are destroyed by disease, as in the cases reported by Türck, Charcot, and others. Dr. Ferrier's experiments and reasoning further show that the only path for the transmission of sensory impressions from the periphery to the hemispheres is through the *tegumentum cruris cerebri*, the optic thalamus and its medullary connections with the cortex; and with the exception of the olfactory tract, there is no other medium of connection between the hemispheres and the organs of sense but the one specified.

The differences in the clinical symptoms of disease of the optic thalami depend on the extent of the lesion. The optic thalamus is situated at a point where the sensory and motor tracts of the crus cerebri have not as yet become clearly separated and differentiated from each other—the two being still more or less bound up together. Both experimental observations and the facts of disease of the spinal cord show that it is much more difficult to interrupt the paths of centripetal or sensory impressions than those of centrifugal or motor impulses. A much more complete disorganisation of the sensory paths is necessary in order that sensation be entirely abolished. The same law holds in regard to the thalamus opticus. A lesion extensive enough to cause interruption of the not as yet completely separated motor paths may cause hemiplegia without loss of sensation. Loss of sensation only follows complete disintegration; and in such cases only will hemi-anæsthesia result along with hemiplegia. The recorded instances, therefore, of so-called destruction of the optic thalamus without loss of sensation, must be put down as only partial lesions; and unless more satisfactory evidence is brought than the mere naked eye appearances of the optic thalamus in such cases, they cannot for a moment be placed against the positive cases, more numerous, in which loss of sensation has been demonstrated to result from lesions situated in this ganglion" (l. c., p. 243). There is also clinical evidence which strongly corroborates the view arrived at on other grounds, that the optic thalamus contains the sensory paths of all the nerves of sensation which take origin below the crura cerebri. It seems further probable that the effects of lesions of the optic thalamus upon vision will vary according to the exact position of the lesion. We should expect hemiopia of both eyes from a lesion occupying the region of the corpora geniculata; total unilateral blindness in the opposite eye if the lesion is central; and if the lesion is very extensive, perhaps total loss of vision in the opposite eye and nasal hemiopia on the same side (FERRIER, l. c., p. 246).

Our knowledge regarding the special functions of individual ganglia is exceedingly incomplete. The corpora quadrigemina are connected both with the sensory and motor tracts and centres. They are in relation with the motor columns of the spinal cord on each side by a strand of fibres thrust aside by the decussation of the pyramids, and which, passing upwards, and embracing the olivary body, ascends with the other longitudinal tracts through the pons, and ultimately enters the corpora quadrigemina at their inferior and lateral aspect. These two strands enter the grey matter of the quadrigemina, and then decussate with each other on the upper surface, and thus form the roof of the canal, or *aqueduct of Sylvius*, which passes below these ganglia and connects the cerebral ventricles with the fourth ventrical and spinal canal (Fig. 12, p. 41). They are also structurally connected with the segmentum and optic thalami. The facts of anatomy and physiological experiment mutually support the view, that though not the centres of conscious vision, the corpora quadrigemina are centres of a most complex co-ordination of retinal impressions with special motor reactions, requiring for its carrying out a distinct nervous machinery. Such machinery exists either in certain parts of the corpora quadrigemina or in the underlying structures. In the nates a common centre exists for both eyes, stimulation of the right side producing movements of both eyes to the left, and of the left side movements to the right; while stimulation in the middle line behind causes a downward movement of both eyes with convergence of the axes; and, in the front, an upward movement with return to parallelism, both accompanied by the naturally associated movements of the pupil. Stimulation of various parts of the nates causes various movements depending on the position of the spot stimulated. After an incision in the middle line, stimulation of the nervous centre, on one side, produces movements in the eye of the same side only (ADAMUK, quoted by Dr. M. FOSTER, p. 506). The centre of co-ordination for the movements of the eyeballs, and that for contraction of the pupil, thus lie in the vicinity of the nates or anterior tubercles of the corpora quadrigemina; and it is probable that the real or immediate centres of those movements lie beneath the corpora quadrigemina, in the front part of the floor of the aqueduct of Sylvius, and therefore are affected in an indirect manner only when the corpora quadrigemina are stimulated. It is still further inferred, from the experiments of Flourens, that "visual sensory impulses become transformed into visual sensations in the corpora quadrigemina; or, in other words, that these nervous structures are centres of sight. But they are so in a limited sense only, for destruction or injury of the cerebral hemispheres profoundly affects vision; and in the absence of the cerebral convolutions, a crude vision, devoid of distinct visual perceptions, is probably all that is possible. The processes constituting distinct and perfect vision, in fact, begin in the retina, and are partially elaborated in the corpora quadrigemina, and possibly in the optic thalami; but do not become completely developed until the cerebral convolutions have been called into operation (Dr. M. FOSTER, l. c., p. 589). Hence it by no means follows that these ganglia are the centres of vision, even though it should be found that destruction of them produces blindness. Destruction of the optic lobes breaks the continuity of the optic tracts, and so cuts off the eyes from the perceptive centres; and that these ganglia are not the centres of consciousness of visual impressions, or *sense* of sight, has been shown by Dr. Ferrier's experimental demonstration—a fact still further borne out by the anatomical investigations of Gratiolet and Meynert. These have led them to conclude that the optic tracts likewise enter into relation with the optic thalami, and with radiating fibres, which proceed to the lateral and posterior regions of the cerebral hemispheres (FERRIER). It is only by such a mechanism that we can account for the co-ordinated movements seen in some cases of somnambulism, where the individual is apparently quite unconscious of any external impressions, but, with eyes open, guides his movements even in difficult and dangerous situations. Flourens and subsequent observers have also noticed that injury or removal of the corpora quadrigemina on one side frequently caused forced movements, and that removal of the whole mass led to great want of co-ordination; but, as to the exact nature of this co-ordination, and what relations are borne in this respect by the corpora quadrigemina to the cerebellum, cerebri, and pons Varolii, we have, at present, no exact knowledge (Dr. M. FOSTER, l. c.) When the optic lobes on both sides are destroyed vision is completely abolished, and the pupils cease to contract when light is thrown on the retina—a proof that in the optic lobes there is an organic connection between the optic and the oculo-motor nerve, which supplies the circular or constringent muscle of the iris—a connection having an anatomical basis in the fact that the central nucleus of the oculo-motor or third nerve is situated in the corpora quadrigemina, beneath the aqueduct of Sylvius, or canal, which, as it were, tunnels these ganglia (FERRIER, p. 71). When one optic lobe is destroyed, blindness of the opposite eye is the result; and, as regards the action of the pupils, it is observed that bilateral contraction is capable of being induced through the one optic nerve. The action is more marked, however, in the pupil of the eye the retina of which is directly stimulated by light. This fact illustrates an important law, of which other examples are to be seen in the spinal centres, and which, as Dr. Broadbent has shown, seems to explain many facts of cerebral paralysis, namely:—"*That movements which are normally associated together are bilaterally co-ordinated in each centre.*" The pupils are usually associated in action; and hence, owing to the bilateral co-ordination in each lobe the destruction of one optic tract does not induce complete paralysis of the opposite pupil. The blindness of the opposite eye which results from destruction of one side of the corpora quadrigemina, or one optic lobe, is readily accounted for by the decussation of the optic tracts in the optic chiasma or commissure (FERRIER, l. c., p. 71.) Ferrier's varied experiments upon animals show that marked disturbances of equilibrium and locomotor co-ordination result from destructive lesions of the corpora quadrigemina or optic lobes; that the most prominent effects are—blindness, paralysis of irido-motor and of some oculo-motor reactions; disorder of equilibrium and locomotion; and annihilation of certain forms of emotional expression. They are susceptible also of irritation by various kinds of stimuli—mechanical, chemical, or electrical—being extremely sensitive to this latter kind of irritation; and the phenomena vary according to the position of the electrodes on the nates or testes. In monkeys irritation of the surface of the nates or anterior tubercles is attended with the following results:—

> "Irritation of one side causes the opposite pupil to become widely dilated, followed almost immediately by dilatation of the pupil on the same side. The eyes are widely opened and the eyebrows elevated. The eyeballs are directed upwards and to the opposite side. If the left tubercle is irritated, the eyes are directed to the right and up. The head is moved in the direction of the eyes. The ears are strongly retracted. With continuance of the irritation the tail becomes elevated, the legs extended, and the jaws firmly clenched, with the angles of the mouth retracted to their utmost. The arms are approximated to the sides, and drawn back flexed at the elbows. Ultimately, when stimulation is kept up, a state of complete episthotonus is produced. Irritation of the posterior tubercles (*testes*) produces the same effects, but in addition cries are excited, varying in character from a short bark, caused by the slightest contact of the electrodes, to all varieties of vocalisation, when the stimulation is continued. The motor effects are shown first on the opposite side of the body, but ultimately both sides become affected by the unilateral irritation."

Generally, the destruction of the corpora quadrigemina annihilates the manifestation of those functions which still remain practically unimpaired by removal of the cerebral hemispheres—namely, equilibration, locomotion, and, in some measure, emotional expression; and therefore Dr. Ferrier concludes that there is reason to believe that the corpora quadrigemina form an essential part of the central mechanism by which these are rendered possible; and, that there is an evident relation between the development of those ganglia and the degree of independence with which some of these functions are manifested after removal of the hemispheres. Dr. Ferrier makes no attempt to differentiate between the optic ganglia as such, and the underlying tracts, for he does not think it possible to determine experimentally what are the functions of the mesencephalic ganglia and cerebellum, apart from their connections and relations to the crura and pons. It may be said that the effects described are really due to the conduction of the currents to underlying motor tracts or centres, but the strength of current sufficient to cause these movements is so excessively weak that it is scarcely perceptible when applied to the tip of the tongue; and, that certain forms of irritation, in which conduction can play no part, likewise produce similar effects,—such, for example, as the vital irritation of inflammatory processes affecting these structures. Dr. Ferrier is also of opinion that the phenomena of electrical irritation of the corpora quadrigemina are mainly of a reflex nature, depending on the transference of irritation of sensory to motor centres and tracts; while both anatomical and physiological considerations justify the view expressed by Flourens, that the optic lobes are essentially similar in constitution to the spinal cord. The optic tracts which originate from these ganglia may be regarded as homologous with the posterior root of a spinal nerve.

And although all the sensory relations of these ganglia are not quite clear, other special sensory tracts besides those of the eyes are brought into relation with motor centres in these ganglia. Meynert has traced, for example, one of the roots of the fifth nerve to a layer of large cells which surround the central canal, or aqueduct of Sylvius. The movements of the trunk and limbs which are produced by excitation of the corpora quadrigemina are such as are concerned in the maintenance of the normal attitude, and for purposes of progression. There is some evidence, also, to support the view that these ganglia are the centres especially concerned in the reflex expression of feeling or emotion. Danilewisky, Drs. Lauder Brunton, and Ferrier have independently found that electrical irritation of the interior of the corpora quadrigemina causes a great rise in the blood pressure, with slowing of the heart and amplification of the pulse waves; while modifications of the functions of circulation and respiration are the most frequent concomitants of states of feeling or emotion. The respiratory rhythm is altered in a marked degree—irritation causing a deep inspiration, followed by prolonged and powerful expiratory efforts. The irritation of the corpora quadrigemina has also been shown to have a direct influence on the viscera, causing contractions of the stomach, intestines, and bladder, with sudden expulsion of their contents;—also, evidence of the relation of these parts to the reflex manifestation of emotion (FERRIER, l. c., p. 84).

The CEREBELLUM is also probably concerned in the co-ordination of the muscular movements of the eyeballs, with reference to binocular vision, and of the muscles generally in locomotion; but the mechanism by which the co-ordination of these orderly movements is accomplished is as yet unknown. Hence the functions of the cerebellum still furnish some of the most obscure, disputed, and unsettled questions in cerebral physiology, and even the positive facts of disease have been appealed to in support of diametrically opposed conclusions. Experimental research on the cerebellum furnishes no evidence in support of the existence of any form of spontaneous or self-determined activity, characteristic of animals possessing their cerebral hemispheres; so that animals retaining their cerebellum, along with the mesencephalic ganglia, only react to immediate sensory impressions, but otherwise remain in absolute inaction, manifesting neither feeling, desire, nor will, until death ensues, unless nutrition is artificially kept up. Lesions of the cerebellum cause very remarkable disturbances, both as regards equilibrium and locomotion, whether the hemispheres exist or not. Superficial lesions cause disharmony of movements, a disharmony which increases in proportion to the extent of the lesion; while total destruction of the cerebellum entails entire loss of all power of progression—disorders of movement resembling those of intoxication in varying degree, from " the walk of a man slightly drunk " to complete loss of power. But these effects are not the result of the muscles being paralysed, nor that the co-ordinated movements of locomotion are rendered impossible, for the animal still retains the power of voluntary motion, and the co-ordinated combinations of muscular contractions concerned in locomotion may be willed or excited as before; but these are no longer co-ordinated with the position of the body in space, and hence the animal, in its attempts at locomotion, reels, staggers, or flounders about in the most helpless manner (FERRIER, l. c.). It has, in general, been found that lateral or unsymmetrical lesions and incisions produce a greater effect than those which are median or symmetrical. Section of the middle peduncle on one side almost invariably gives rise to a forced movement, the animal rolling rapidly round its own longitudinal axis. The rotation is generally, though not always, towards the side operated upon; and is accompanied by peculiar rolling movements of the eyes (*nystagmus*), suggestive of vertigo. Frequently an eye is moved in one direction—*e.g.*, inwards and downwards, and the other in a different or opposite direction—*e.g.*, outwards and upwards. It is also probable, though not proven, that the functions of the cerebellum are especially connected with afferent impulses proceeding from the semicircular canals (Dr. M. FOSTER, l. c., p. 591). There is no evidence that this part of the brain is connected with the sexual functions, as was once the popular belief.

The parts of the encephalon whose functions have been thus shortly noticed—namely, the cerebellum and corpora quadrigemina, together with the pons Varolii—lie between the cerebral hemispheres and the medulla oblongata, and are sometimes considered together under the name of the "*mesencephalon*." With grey matter abundant in the CRURA CEREBRI and PONS VAROLII, it must be inferred that they possess important functions; but we hardly know more concerning them than that the former serve as the great means of communication between the spinal cord and the higher parts of the brain, and that both are intimately connected with the co-ordination of movements. Forced or disorderly movements are the frequent results of section of either of them; and the possession of those parts intact, in the absence of the cerebral hemispheres, is sufficient to carry out the most complex bodily movements. The peduncles contain both sensory and motor fibres. They are intermediate between the pons, medulla oblongata, and the voluntary centres in the brain; while they also establish a connection between the cerebellum and the cortex of the cerebrum. Destruction of one peduncle causes the animal to move to the side opposite the lesion, describing the "circus movement;" and irritation of these peduncles may cause pain, or movements of various groups of muscles, according to the part irritated. The PONS VAROLII forms a transverse commissural band of fibres, connecting the two hemispheres of the cerebellum, containing nuclei of grey matter and numerous fibres passing from below upwards, for the transmission of motor or centrifugal impressions, principally in the anterior part of the pons. Any unilateral lesion causes paralysis of motion on the opposite side, and paralysis of facial on the same side of the face as the disease and the opposite side to the paralysis of the limbs. Since paralysis of the face, seen in cases of hemiplegia from disease of the corpus striatum, is on the same side as that of the body paralysis, it follows that the impulses proceeding along the cranial nerves cross over like those of the spinal nerves. Hence, when paralysis of the face occurs on the opposite side to that of the body paralysis, it may be inferred that the injury or disease has affected the cranial nerve (or nerves) in a part of its course before decussation has taken place; and it is probable that the decussation which begins in the spinal cord is only gradually completed as the impulses pass through the medulla oblongata and pons Varolii (to be finally completed in the peduncles of the brain); and it is also probable that the roots of the facial decussate in the lower part of the pons, while the other motor tracts decussate in the medulla. In disease of the pons loss of sensibility is a much more rare result than loss of motion, and is always on the opposite side; for, sensory or centripetal impressions cross chiefly in the cord. As a reflex centre, it has been observed that movements of the limbs, and even co-ordinated movements, take place after removal of all parts in advance of the pons (LONGET). By Nothnagel the pons is described as the region of cramps—a convulsive centre irritation of which causes very severe convulsions. It may be excited by an excess of carbonic acid, or the absence of oxygen in the blood, as seen in asphyxia; and, it is in close connection with other centres situated in the medulla oblongata.

There are the experiences of disease which practically detach the hemispheres from their mesencephalic connections, leaving thought and speech intact, so that we can obtain direct testimony as regards the consciousness of impressions. Such an experiment is performed by a lesion of the crus cerebri, or of the posterior part of the peduncular expansion, giving rise to phenomena not infrequent in clinical experience. When such a lesion occurs *the individual has absolutely no consciousness of tactile impressions made on the opposite side of the body, however much he may strain his attention to receive them.* In the mesencephale alone, therefore, sensory impressions are not correlated with modifications of consciousness; hence it is concluded that sensation is a function of the higher centres, and sensation proper is abolished by removal of the cerebral hemispheres (FLOURENS, FERRIER). Nothing whatever is known as to the physiological position and functions of the numerous grey tracts of the pons Varolii, nor of the significance of the pituitary body and pineal gland.

The functional manifestations expressed by the parts already described consist of,—1. The function of equilibration, or maintenance of the bodily equilibrium; 2. co-ordination of locomotion; 3. emotional expression. The first and second of these are intimately bound up together; and the maintenance of equilibrium is a combined function of the mesencephalic centres. It involves the conjoint operation of, (1.) A system of afferent nerves; (2.) a co-ordinating centre; (3.) efferent tracts in connection with the muscular apparatus concerned in the action. Lesions of one of either, or conjointly of all of them, will overthrow the faculty of equilibrium. The afferent apparatus consists of three systems, namely—(1.) Organs of reception and transmission of tactile impressions; (2.) organs for the reception and transmission of visual impressions; (3.) the semicircular canals of the internal ear and their afferent nerves. The influence of each of these will be afterwards considered under the pathological relation of brain and nervous system.

The MEDULLA OBLONGATA, as a continuation of the spinal cord, contains tracts for sensory and motor transmission, and likewise a series of reflex centres for special movements—centres of reflex co-ordination of a more complex character than in the cord, and more intimately related to essential vital functions. The transmission of motor or centrifugal impressions occurs chiefly in the anterior pyramids; and it is of importance to remember that it crosses or decussates. All the motor fibres passing from one side of the brain which are distributed to the muscles on the opposite side of the body, cross or decussate in the anterior pyramids, while those supplying the face cross in the pons Varolii; and in man the decussation is usually complete. Hence it is that disease which causes rupture of motor fibres passing from one cerebral hemisphere, is followed by paralysis of motion on the side of the body opposite to the seat of the lesion. Nothing precise is known as to the transmission of sensory or centripetal impressions through the medulla oblongata. Section of one half does not cause complete loss of sensibility. It is probable that the sensory fibres pass up through the grey matter of the medulla (M'KENDRICK). The majority of the sensory fibres decussate in the cord, so that both motor and sensory fibres in connection with one of the cerebral hemispheres are in connection with the opposite side of the body; but the motor fibres cross in the anterior pyramids of the medulla, and the sensory fibres cross in the cord. "When we consider the comparatively small size of the medulla, nothing is more surprising than the number of centres said by physiologists to exist in it; and one cannot help suspecting that at present we know only a few empirical facts, and that the hypothesis by which to explain the seeming complexity of function with simplicity of structure is still wanting" (M'KENDRICK). With the exception of the first four cranial nerves, all the other cranial nerves are directly connected with the grey centres of the medulla oblongata. Hence from this anatomical distribution alone we might infer that the medulla oblongata would be the centre of reflex co-ordination of such actions as are manifested in the regions of distribution of these various nerves, singly or combined. It is the co-ordinating centre of reflex actions essential to the maintenance of life. "If all the centres above the medulla be removed, life may continue, the respiratory movements may go on with their accustomed rhythm, the heart may continue to beat, and the circulation be maintained; the animal may swallow if food be introduced into the mouth, may react to impressions made on its sensory nerves (withdrawing its limbs, or making an irregular spring if pinched, or even utter a cry as if in pain), and yet will be merely a non-sentient, non-intelligent, reflex mechanism" (FERRIER). The functions in which the medulla is concerned are, therefore, of such importance to the whole organism, that injury or disease of this portion of the encephalon is more dangerous to life than those of any other part of the brain or spinal cord. The following centres are described as being located in this part of the nervous system:—(1.) *The Centres for the Involuntary Movements of Respiration, and for the Convulsions of Dyspnœa.* These are two in number, expiratory and inspiratory, connected with the roots of the pneumogastric nerve. It is probable that these centres are situated in the upper part of the cord as well as in the medulla. Their destruction at once causes cessation of respiratory movements and death. The region has been named "*le nœud vital,*" or vital knot of Flourens. It extends on both sides of the middle line downwards, in the floor of the fourth ventricle, at the apex of the calamus scriptorius. The centres also possess accelerating and inhibitory nerves. (2.) *The*

Centres for the Regulation of the Heart's Action are two in number:—(*a.*) *inhibitory,* whence the inhibitory vagus-fibres for the heart receive their rhythmical stimulation; (*b.*) *accelerating,* associated with the sympathetic. The precise situation of these centres is not yet known; but it is evident that some connection exists between the cardiac inhibitory and the respiratory centres. (3.) *The Vaso-Motor Centre* is situated bilaterally at some distance from the middle line, in that portion of the medulla which contains the continuation of the lateral spinal columns, in which the vaso-motor and "pressor" fibres run. It contains some large multipolar ganglion cells; and is constantly in action, its activity being either automatic or merely reflex. It governs all the smaller vessels throughout the body, along with fibres from the sympathetic system, so as to keep them in a state of tonus. (4.) *The Centre for the Dilatation of the Pupils, and for the Movements of the other Smooth Muscles of the Eye* (the exact position of which is unknown) is constantly active, possibly owing to reflex stimulation. It is under similar influences to the respiratory and vaso-motor centres, dyspnœa, for example, being accompanied by dilatation of the pupils, whilst the optic vessels become pale.

Many circumstances indicate a close connection between the four centres just mentioned, especially their stimulation by certain morbid conditions of the blood, and the coincidence in the rhythm of their action (HERMANN).

(5.) *The Centre for the Movement of Swallowing.*—Its exact situation is not yet determined; but it is associated with the sensory and motor filaments associated in this process—namely, the various afferent and efferent nerves concerned in the mechanism;—namely, the glosso-pharyngeal, the hypoglossal, the facial, and the fifth, all springing directly from the medulla oblongata; and when that portion of the nervous system from which they spring is under stimulation, convulsive movements of swallowing take place. The centre is only called into activity by reflex stimulation. (6.) *It contains the Centre for Voice and Articulate Speech,* probably in connection with the external root of the spinal accessory; and although it may not be possible to obtain direct experimental proof that the medulla oblongata is the co-ordinating centre of the various muscular movements concerned in the production of articulate speech, yet this is rendered probable—(*a.*) because the muscles concerned in articulation are directly innervated from the medulla oblongata, and the individual nuclei of those various nerves are anatomically connected with each other; (*b.*) in the disease known as *bulbar paralysis,* sometimes also called *Duchenne's paralysis,* or *glosso-labio-laryngeal paralysis,* the lesions institute a remarkable selection of the centres of innervation of the muscles of articulation; so that there is gradual and progressive paralysis of the tongue, palate, lips, and laryngeal muscles, rendering articulation, and ultimately deglutition, impossible. These lesions are found to implicate the centres or nuclei of the hypoglossal, facial, accessorio-vagus, and glosso-pharyngeal, which undergo progressive degeneration. This selection indicates a functional as well as an anatomical correlation of these various centres, but how they are co-ordinated remains unsolved. Schroeder Van der Kolk was of opinion that the connections of the hypoglossal, facial, and fifth nerves with the olivary bodies pointed to these ganglia (Fig. 11, p. 40, *ante*) as the co-ordinating centres of articulation; but his views are not confirmed either by histological observations, nor by pathology (FERRIER, l. c., p. 26). (7.) *The Centre for the Movements of Mastication* derives proofs of its existence in the medulla from conditions similar to those mentioned under (5.) and (6.)—namely, the occurrence of trismus or cramps of the masticatory muscles under similar circumstances to those already described. (8.) *The Centre influencing Glycogenesis or Diabetes.*—Bernard discovered that a puncture or lesion in the floor of the fourth ventricle, near the middle line, and extending from a little above the calamus scriptorius to the broadest part of the groove, causes transitory diabetes mellitus or sometimes merely increased secretion of urine—diabetes insipidus. This centre is probably associated with the roots of the vagi and of the vaso-motor centre. (9.) *A Centre directly influencing Salivary Secretion,* from which originate those fibres of the facial forming the chorda tympani and lesser superficial petrorsal nerves, which are distributed to the glands.

The medulla also receives fibres from the higher centres, by which all of the centres just noticed may be more or less influenced.

The SPINAL CORD acts (1.) as a transmitter of motor (centrifugal or efferent) impressions and of sensory (centripetal or afferent) impressions between the encephalon and the periphery; and, (2.) as a reflex centre. The brain is thus brought into relation with the periphery by thirty-one pairs of spinal and twelve cranial nerves. These nerves or cords of communication are also separable into two great divisions. One set carry the impressions *from the periphery* to the cord and brain, and are therefore called *afferent* nerves; while the other set carry impulses *from the brain and cord* to the periphery, and are therefore called *efferent* nerves. Sensory impressions and motor stimuli are thus respectively conveyed; and hence the terms *sensory* and *motor* are frequently employed in place of afferent and efferent. The spinal nerves are connected to the spinal cord by two roots, one of which (proved to be motor) arises from the antero-lateral columns of the cord; the posterior (proved to be sensory) is connected with the posterior surface (Sir C. BELL). After a very short independent course, and the development of a ganglion on the posterior (or sensory) root, the two roots unite to form one trunk, which is therefore a mixed nerve containing both motor (efferent) and sensory (afferent) fibres, which distribute themselves by minute ramifications in the receptive and active organs at the periphery, each filament remaining distinct throughout its whole course, thus maintaining the phenomena of isolated conduction. Everywhere throughout the body the sensific ramifications of the mixed nerve pass to the surface of the part which is moved by the muscles receiving their motor fibres from the same compound nerve; so that, while the former supply sensation to the surface, the latter convey the stimulus to excite the act of motion (VAN DER KOLK). From this anatomical arrangement of parts, it is clear that the spinal cord is (with the exception of the sympathetic communication) the only connection between the brain and the nerves of the trunk and extremities. It must therefore contain the paths for the conduction of all voluntary movements of the trunk and extremities, for all sensations in those parts, and for all actions not psychical, or other cranial centre, such, for example, as the respiratory centre. It is also anatomically proven, that the nerves of the trunk in the spinal cord do not simply run to the brain; but that all of them (or at least the motor nerves) are first of all connected with ganglion cells. But no well ascertained anatomical facts have as yet been discovered which throw light on the conduction of impressions from motor and sensory ganglion-cells to the brain. It is most probable that those cells are immediately connected with a complicated network of fibres, which continues uninterruptedly to the brain, but from which fibres continually separate and run to the brain, isolated in the white substance (HERMANN, l. c., p. 476). Our knowledge of the paths of conduction in the cord has been gained partly by direct experiment; partly from observation of pathological conditions; and, partly from a study of anatomical arrangement. The experiments consist mainly in observing the effects of partial section of the cord, such as unilateral; or, of individual white or grey columns, and sections at different levels on the same or opposite sides. The general results may be thus stated:—

"From the effects of partial division of the cord in various ways (BROWN-SEQUARD, SCHIFF, SETSCHENOW, and others), the following conclusions have been drawn:—(1.) The conduction of localised sensory impressions, and of motor expressions, which are voluntarily restricted to definite sets of muscles, is effected through the white substance. Partial section of the latter severs the connection between the mind and individual regions of the skin and individual groups of muscles; hence result insensibility to tactile impressions, anæsthesia, and paralysis of voluntary motion. The paths which subserve conduction remain on the same side of the cord as far as the brain—*i.e.*, they do not cross the middle line of the cord. The conduction of sensory (or centripetal) impressions takes place through the posterior white columns, that of motor (or centrifugal) impressions through the anterior and lateral white columns" (HERMANN.) "If the anterior columns be cut by an incision extending into the grey matter, leaving the posterior columns intact, voluntary movements disappear in the parts below the section. Again, section of the posterior columns and grey matter, leaving the anterior uninjured, enfeebles but does not destroy the power of voluntary motion below the section. Finally, section of an antero-lateral column on one side paralyses voluntary motion on the same side."

From these facts it is inferred that, (*a.*) the motor tracts passing from the brain to the periphery are in the antero-lateral column. "But the recent and careful experiments of Ludwig and Woroschiloff place the motor paths in the lateral columns only; and the anterior columns are regarded more as commissural connections between the motor nerves and the adjacent segments, and not at least the direct paths of motor impulses proceeding from the brain" (FERRIER, l. c., p. 4.) (*b.*) That in the cord the fibres forming these tracts are distributed to the same side of the body (M'KENDRICK.) (2.) The posterior roots of the spinal nerves pass into the posterior columns, and into the grey matter in the posterior cornua. These roots have been proved to be sensory. A complete transverse section of the posterior columns does not abolish sensibility in the parts below; but there is a loss of power of the co-ordinated movements. Section of the posterior columns and of the antero-lateral columns (leaving only the grey matter in the centre of the cord intact) does not abolish sensibility. Again, section of the antero-lateral columns, and of the whole of the grey matter, leaving only the posterior columns uninjured, is followed by complete loss of sensibility in the parts beneath. The inference therefore is that sensory impressions must be conducted through the grey matter. It is manifestly impossible to cut the grey matter without also injuring the columns ; but by a process of exclusion such as has been followed above, the proof is complete (M'Kendrick). (3.) The conduction of painful impressions and of involuntary (especially reflex) motor impulses, takes place through the grey substance in its whole extent, without any distinction of sensory and motor tracts. Section of the grey substance, therefore, produces amongst other things a condition in which painful operations cause sensations or merely appreciation of touch, but not of pain—"*analgesia.*" Such a condition frequently supervenes during the narcosis of chloroform, in which case the knife of the surgeon is felt, but no sensation of pain is experienced. This result of section of the grey substance is not restricted to sharply defined regions of the body, as are the results of section of the white substance ; but is somewhat regular in all the parts supplied by nerves which enter the cord below the point of section, and it is the more complete according as the section has included more of the grey substance (Hermann). "The chief differences of opinion are as regards the exact localisation of the sensory tracts. Schiff would distinguish between the paths of tactile impressions and those of painful impressions. The former he places in the posterior columns, the latter in the central grey matter. Ludwig and Woroschiloff place the sensory with the motor in the lateral columns—a differentiation of the two not being anatomically possible. The posterior columns, like the anterior, become only commissural connections between the sensory roots and adjacent segments, and do not constitute the long or direct sensory paths to the brain. Their researches also confirm what other experimenters have found—namely, that even after destruction of large areas of the lateral columns neither motion nor sensation is absolutely paralysed in any particular part; whence it is argued that a vicarious interchange of functions potentially exists between different parts of the cord. Though the main results of section of different parts of the cord agree, many more observations and experiments will have to be made before it can be said that the sensory and motor paths have been exactly defined and all views reconciled" (Ferrier, l. c., p. 5.) (4.) Brown-Séquard has found that sensory tracts decussate to a considerable extent in the posterior part of the cord. He found that hemisection of the cord involving the grey matter enfeebled sensibility *on the opposite side* more and more as the section became deep ; that a vertical section in the posterior median fissure caused loss of sensibility on both sides ; and that a lateral section, whilst it caused loss of sensibility (anæsthesia) on the opposite side, was followed by increase of sensibility (hyperæsthesia) on the same side—presumed to be due to irritation caused by vaso-paralytic distension of the vessels of the cord on the side of the section. (5.) The grey matter of the lower cervical, dorsal, and lumbar regions of the cord may be regarded as composed of groups of reflex centres associated with the general movements of the body ; whilst in the upper cervical region, and in other portions of the cord, there appear to be differentiated centres corresponding to special actions. The initial excitation for *reflex action* may commence in any sensory nerves passing *to the cord, or in sympathetic filaments.* When the impression reaches the cord a minute interval of time elapses before a muscular response is given. This response may consist either in the movements of muscles specially co-ordinated to a particular centre; or, in more general movements of groups of muscles; and, so exact may the co-ordination be as to produce movements simulating those of a conscious or voluntary character. The reflex excitability of the cord is increased by removal of the influence of encephalic centres, as by decapitation; or in normal circumstances, as during sleep, or when the mind is in abstraction or reverie. It is also influenced by various drugs. Strychnine and the alkaloids of opium increase reflex excitability; while aconite, hydrocyanic acid, ether, chloral, and chloroform have an opposite effect" (M'KENDRICK). Marshall Hall was the first to formulate the reflex function of the spinal cord—foreshadowed by Unzer and Prochaska. When, as the result of injury or disease, there is a solution of the continuity of the cord at any point, all the parts deriving their nervous supply from the cord below the seat of lesion become paralysed, both as regards voluntary motion and sensation. If, however, the soles of the feet be tickled, the legs will be thrown into convulsive action, of which the individual is not conscious, and which it is out of his power in the slightest degree to control. (6.) Special reflex centres exist in the cord —(*a.*) In connection with the maintenance of the tonus of smooth muscles (as in the tonic contraction of the dilator fibres of the pupil, and the smooth optic muscles of Müller), which cease on section of the sympathetic cord in the neck. The centre for this influence is said to be in the neighbourhood of the lower cervical and upper thoracic vertebræ (between the sixth cervical and third dorsal nerves—"centrum cilio-spinale" of Budge; "centrum oculo-spinale" of Bernard), because the sympathetic cord derives the fibres concerned (and which control the radiating fibres of the iris) from the anterior root of the spinal nerve of that locality. A condition of paralysis or of irritation of the cord in that vicinity is accompanied by corresponding phenomena in the eye—paralysis, for example, being marked by contraction of the pupil. These circumstances prove that the region of the cord referred to is concerned in the *conduction* of the necessary impressions, and not that it is their *place of origin.* (*b.*) Centres exist for maintaining the *tonus* of arteries. Unilateral section of the cord causes dilatation of all the arteries of the same side below the level of section, so that the *tonus* depends on the integrity of fibres derived from the spinal cord. A centre for all these fibres is situated in the medulla oblongata; and the grey substance of the cord appears to contain proximate centres of a reflex nature, to which the descending vaso-motor centres first proceed. The vaso-motor nerve descending from the medulla oblongata, as well as the "pressor" fibres proceeding to it, are contained in the lateral columns of the cord (DITTMAR). (*c.*) Accelerating centres exist connected with cardiac movements, originating in the upper part of the cord, connected with those fibres passing through the sympathetic that quicken the heart's movements (M'KENDRICK). (*d.*) Respiratory centres.—Section of the cord above the eighth dorsal nerve paralyses the abdominal muscles; above the first dorsal, the intercostals; above the fifth cervical, the serratus magnus and the pectorals; and above the fourth cervical (by paralysing the phrenics) arrests the action of the diaphragm. Doubts still exist as to the exact position of these centres. (*e.*) A genito-spinal centre is associated with the movements of the genital organs, and exists in the lumbar region. The activity of this centre may be affected by sensory impressions transmitted by other channels than the ordinary sensory nerves of the part. (*f.*) Ano-spinal and vesico-spinal centres exist for the sphincter-ani, and for the bladder, in the lower portion of the dorsal or upper part of the lumbar region; but the tonic closure of the rectum and bladder is regulated by fibres proceeding from the brain (MARIUS), just as are the other regular reflex acts over which the spinal cord presides" (HERMANN and M'KENDRICK). These phenomena have sometimes been described as due to the *automatic action* of the spinal cord; but they are in a great measure reflex actions of a constantly operative character, and due to latent centripetal influences always proceeding from the periphery (FERRIER, l. c., p. 22).

There is also to be noticed here the existence of influences which, (*a.*) render more difficult the production of reflex acts; and, (*b.*) render keener the powers of localising sensations and voluntary movements. After it had been noticed that the power of reflex action in the region belonging to the spinal cord became more regular and stronger after separation of the brain, certain organs were discovered to exist in the latter which constantly act prejudicially upon the reflex power of the cord. These are known as Setschenow's "*inhibitory centres for reflex movements;*" and, in the optic lobes there is found to exist some such power which exerts a constant inhibitory action upon the reflex functions of the spinal cord; and the operation of certain depressors of reflex power, such as morphia, digitaline, and the like, depends upon stimulation of these inhibitory centres. There are said to be three kinds of influence inhibitory of reflex movement, namely,—1. Those centres already mentioned in the brain; 2. that exerted by the power of the will or mind; as when by a strong effort of the will, some persons may succeed in restraining the movements of the legs which would otherwise result from tickling the soles of the feet, and it has been proved experimentally in the lower animals that the encephalic centres exercise a restraining inhibitory influence over the reflex action of the cord (FERRIER, l. c., p. 18); 3. that exerted by centripetal fibres (HERMANN, l. c., p. 481). Generally, it may be said that "the spinal cord, and indeed the whole cerebral nervous system, may be regarded as an intricate mechanism in which the direct effects of stimulation or automatic activity are modified and governed by the checks of inhibitory influences; but we have yet much to learn before we can speak with certainty as to the exact manner in which inhibition is brought about. In the ordinary actions of life, the spinal cord is to a large extent a mere instrument of the cerebral hemispheres; hence we may readily expect that regulative inhibitory influences passing from the latter to the former would be of frequent occurrence (Dr. M. FOSTER, l. c., p. 541; also Dr. LAUDER BRUNTON, in *West Riding Reports,* Vol. IV.). (7.) As to decussation of fibres, it may be stated generally, that "in the mass of white substance extending between the ganglia at the base of the brain and the central grey substance of the cord, an apparently complete crossing of the fibres from one side to the other takes place. The physiological proof of this lies in the circumstance that pathological changes and injuries situated in one of the cerebral hemispheres are followed by anæsthesia, or paralysis of voluntary motion, only in portions of the *opposite* side of the body. Not until the lesion has affected by pressure, &c., the cranial nerves situated at the base of the brain, do symptoms manifest themselves in portions of the head situated on the *same* side as that of the seat of injury. As to the exact situation where the crossing takes place, the discoveries of anatomists and physiologists do not quite agree. For example, while dissection shows an intercrossing of fibres of the white columns in the anterior white commissure of the cord, unilateral sections of those columns are only followed by paralysis of the same side (Volkmann, Von Bezold). The cross commissural fibres are, therefore, possibly merely co-ordinative in function. Fibres cross over from one side to the other in various situations of the spinal cord :—(*a.*) Between the pyramids, where, to judge from the connection with the columns of the spinal cord, both motor and sensory fibres are concerned in the crossing, the former, however, passing over at a lower point; (*b.*) in the white raphe or white lamina occupying the mesial plane of the medulla, where occurs especially the crossing of connecting columns between the grey nuclei of the cranial nerves and the cerebellum, as well as of the fibres running from the nuclei to the peduncles of the brain; (*c.*) in the pons Varolii, about the crossing of the fibres of which but little is known; (*d.*) the crossing is completed in the peduncles of the brain."

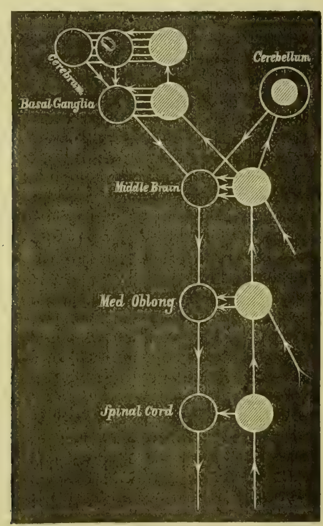

Fig. 14. Diagrammatic representation of the cerebro-spinal nerve centres. The white discs represent *sensory*, and the black ones *motor* ganglia. The arrow heads or darts show the direction of impulses and impressions (after Ferrier and Coats).*

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* Spinal Centre.—A, sensory or afferent, A′ motor or efferent divisions, joined by the intracentral communication, *a*. The afferent nerve or nerves leading to A are

The great importance, from a practical point of view, which attaches to a minute and accurate knowledge of the anatomical structure and mutual functional relations of the encephalic centres and spinal cord, must be my

indicated by *a*, the direction of the arrow or *darts* (as the printer calls them) indicating the centripetal character of the fibres in this as in the higher regions. The other line *a′* is the centrifugal or motor nerve. An impression *a* causes the action *a′* in a reflex or excito-motor manner, quite independently of the higher centres. Hence all the parts of the cerebro-spinal system above A, A′, may be removed without destroying reflex action. This independent activity of the spinal centres he designates by the term *kentro-kinetic*—the Greek equivalent for excito-motor—*κέντρον*, spur; *κινέα*, I move.

Medulla Oblongata Centres, represented by B, the sensory, and B′, the motor divisions. The independent functional activity of these centres is of greater complexity than that of the spinal cord, but of essentially the same nature—namely, reflex or excito-motor; and on account of this greater complexity and multiplicity of sensory and motor co-ordinations, they may be described as the regions of compound reflex action (*syn-kentro-kinetic*). The diagram shows how the impression *a*, which in the spinal centres A, A′, causes the action *a′*, carried to the medulla as *b*, may produce through B, B, B′, and *b*, precisely the same muscular action *a′*, but as part of a higher and special co-ordination; and in the same manner as the reflex functions of the spinal cord may continue to be performed after removal of all above A, A′, so the compound reflex actions of the medulla oblongata may continue to be performed after removal of all the centres situated above B, B′. The diagram also shows that section below A, A′, or between A, A′ and B, B′, must cause paralysis of the functional manifestations of the centres situated higher up, in so far as they are concerned with *a* and *a′*.

The Mesencephalic Centres are centres of co-ordination of a still more complex form of activity, such as of locomotion, emotional expression, and the like, elicited more especially in response to impressions made on the organs of special sense; and usually termed consensual or sensori-motor (Carpenter). On account of the ambiguity involved in the use of this term, and inasmuch as sensation in the psychological acceptation of the term (consciousness of sensory impressions) cannot be predicated of these centres, Dr. Ferrier proposes the term *æsthetiko-kinetic*, to signify the activity of the mesencephalic ganglia—arbitrarily defining *æsthesis* as a physical in contradistinction to a psychical modification of special sensory nerve centres. In so far as the action *a′* is part of the manifestation of the functional activity of the mesencephalic centres, it is the result of *æsthetiko-kinesis*, while the same action conditioned by the spinal or medullary centres is kentro-kinetic, or syn-kentro-kinetic, the signification of the action differing according to the centre by which it is conditioned. It is further shown that all above the centres C and C′ may be removed without destroying æsthetiko-kinetic action; but (as Dr. Ferrier shows) the development of these centres, and degree of independence which they possess, are inversely proportional to the development of the cerebral hemispheres. Hence, in different animals the effects of removal of the cerebral hemispheres vary in accordance with this law. The paths of æsthetiko-kinesis are not independent of those concerned in kentro- or syn-kentro-kinesis; and therefore a lesion which destroys the centres or paths of these forms of activity must, *ipso facto*, paralyse the manifestation of æsthetiko-kinetic action.

Cerebellum.—Here the sensory, D, and the motor, D′, centres are represented as included the one within the other, the two not having been definitely anatomically differentiated from each other. The sensory and motor relations are indicated by the arrows *d* and *d′* respectively. The activity of the cerebellum is simply a branch or division of æsthetiko-kinesis, and is intimately bound up with the activity of the mesencephalic centres, though capable, to some extent, of complete differentiation. This action of the cerebellum especially relates to the function of equilibration. The diagram is intended to show how the centres and paths concerned in this form of activity may be removed without destroying the functional manifestations of the mesencephalic ganglia, or of the cerebral hemispheres proper. The action *a′*, therefore, in so far as it is concerned in the function of equilibration, becomes paralysed, *qua* this function only, by destruction of D, D′ or *d*, *d′*. Its excitation from the cerebral hemispheres in volition, or its excitation reflexly through the spinal centres, is left intact. So also section of the afferent fibres going to the cerebellar centres does not necessarily impede the transmission of sensory impressions to the centres of consciousness. The diagram shows that neither sensation nor voluntary motion need be affected by disease of the cerebellum. All above C, C′ and D, D′ may be removed by section through the tracts E, E′, which correspond to the crura cerebri, without destroying the functions of equilibration, co-ordinated locomotion, and the like—facts experimentally demonstrated in excuse for giving so much space to this subject. The rapid progress of experimental discoveries in relation to the Brain and Nervous System in recent years, has rendered a knowledge of the results of these discoveries still more important and necessary to be fully considered. In relation to diseases of the Brain and Nervous System, our information is perhaps the most rapidly progressing part of the Science of Medicine, notwithstanding the many objections which have been recently made against Experimental Physiology, or Vivisection, as it is now the fashion to call it, and the development of a knowledge which can be learned in *no* other way.

The doctrine of cerebral localisation has long engaged the attention of Lockhart Clarke, Hughlings Jackson, and Broadbent in this country. It received a great impulse from the anatomico-physiological labours of Meynert, of Hitzig, and of Fritsch in Germany; and of Ferrier in this country; and more recently by the energetic clinical expositions of Charcot in France, and by the school of young physicians which he inspires. Symptoms of brain disease, such as some kinds of convulsions, "are really experiments on parts of the human brain analogous to those which Ferrier, Fritsch, and Hitzig have made on the brains of animals—the artificial rousing up of particular parts of the encephalon in them by direct Faradisation, as contrasted by experiments made by disease in man. The clinical study of diseases of the nervous system is really a study of quasi-anatomical and physiological experiments" (Hughlings Jackson, *Brit. Med. Journ.*, May 10, 1873). That the study from this point of view has made any real progress of late, is mainly because physicians have found themselves in possession of a method of

the lower animals, in which the mesencephalic centres reach a high development in comparison with the cerebral hemispheres.

The Basal Ganglia, consisting of the optic thalami and corpora striata, are represented by E and E′ respectively, and their connections with the sensory and motor tracts of the crus by *e* and *e*′ respectively. The intracentral connections are indicated by the parallel lines *e*. These basal ganglia are the centres of a form of activity, subordinate to that of the hemispheres proper, frequently termed secondary reflex or automatic action. The various sensory and motor centres which are differentiated in the hemispheres are integrated in these ganglia, and organic nexuses may be established between them; so that actions at first requiring volitional education and conscious exertion become organised, as it were, reflexly or automatically in these ganglia. Their functional activity is below the domain of consciousness; and destruction of the centres E, E′ cuts off all communication between the hemispheres and the sensory and motor tracts, and necessarily therefore destroys the action between them and the hemispheres, but destroys also *true sensation* and *voluntary motion*—functions belonging only to the hemispheres.

The Cerebral Hemispheres, in their sensory regions, are represented by F, and in their motor regions by F′, while G indicates the associating fibres between sensory and motor centres. By G are indicated the frontal regions, the centres of inhibition, or inhibitory motor centres. The respective communications between these centres and the basal ganglia are represented by the arrows *f*, *f*′, *g*, the direction of the arrow indicating the centripetal or centrifugal direction. The reaction between the sensory and motor centres F, F′ involves consciousness, which may be differentiated into two kinds—namely, one kind indicated by actions conditioned by revived or ideal impressions; and others which are the results of compound associations. But the activity of the hemispheres is under the control of the centres G, which are the basis of attention.

Though the various spinal and encephalic centres have thus their own special modes of activity, capable, more or less, of individual and independent separation, yet they form only parts of a complex whole, and not acting disconnectedly or out of relation to each other, but all more or less engaged in the most ordinary and apparently most simple forms of functional manifestation (Ferrier, l. c., p. 295).

procedure based on topographical anatomy. From this point of view the older observations are very defective, because of want of precision in description in this respect. Hence lesions have been and still are indicated vaguely, and their situation is badly defined, for want of a sufficiently complete anatomical nomenclature. Ecker, Turner, and Allen Thomson have now furnished us with the means of accurately fixing the seat of lesions, based on a definite topography of the brain, which are necessary to be used in solving the many questions concerned with cerebral localisation, whether of normal functions, or of diseased processes. From this point of view, it is necessary to master the researches of Ferrier and others, as detailed in his recent great work on *Localisation of the Functions of the Brain.* It deals with a subject which lies at the foundation of the clinical study of diseases of the nervous system; and of such importance that, combined with such clinical experiences as those of Hughlings Jackson and Dr. Alexander Robertson of Glasgow, furnishes the basis for a new departure in the study of cerebral disease. The results of these workers have been more appreciated abroad than at home, where they have hardly yet become generally known. Then the all-important question is sure to arise—Has there been, or is there likely to be, any practical application of the theory or doctrines attempted to be taught by these and such like experiments? Shall we be any the better able to treat morbid states of the nervous system by the knowledge that there are motor and sensory areas in the cortex of the brain, or any more definite knowledge of its anatomy and functions than we have known before? In reply to these questions, Dr. Alexander Robertson observes that already, especially in France, in several cases motor symptoms have been taken by surgeons as guides to the seat of morbid action; and with successful results the skull has been trephined over the part of the brain which the character of the movements pointed out. The operation of trephining (which has long fallen into comparative disuse) may now, especially in these antiseptic operation days, be performed with greater precision, and greater probability of success than in the past—a result due to the experiments and observations relating to the localisation of motor function, and to Lister's antiseptic teachings. Should there be fixed pain in the head in the motor region, the localisation of disease by symptoms becomes of course more certain. Such pain is often associated with disease of the skull—generally syphilitic. But even though no pain is felt by the patient, it may be elicited at the diseased part by percussion of the skull with the point of the finger. Dr. Alexander Robertson demonstrated this fact on two patients shown at the meetings of the Pathological and Clinical Society of Glasgow, in Dec., 1877. In one of these cases of localised convulsions, it occurred to Dr. Robertson that if the disease were on the surface of the brain, implicating the inner membranes (though these parts were not sensitive in health, they would be so if they were the seat of morbid change), it might be possible, without hurting the patient, to transmit as much force by that means as would irritate the diseased part into sensibility. The result was as described—pain was elicited by percussion. He has in four cases since developed pain of this kind—the patients being unconscious of any unpleasant sensation before percussion; and in all of them (particularly in three) the effect of counter-irritation over the painful part was highly beneficial; and in two of the cases the application of percussion strikingly supported Hitzig and Ferrier's views. In percussing the skull, great care should be taken to apply the same amount of force at each tap; and to avoid suggesting to the patient, by remark or otherwise, any particular point of the skull. Dr. Robertson is in the habit of first tapping the whole head with the finger, and then asking the patient if he feels pain at one place more than another. Should this be so, the observation is to be verified by a second percussion. Diagnosis by percussion may thus be of value in cases where there is the least ground for suspecting the presence of disease on the surface of the brain, or in its membranes. In the first part of the journal called "*Brain*," Dr. Ferrier corroborates and illustrates the value of percussion of the skull as an aid in diagnosis.

IV.—MORBID ANATOMY AND PATHOLOGICAL RELATIONS OF THE SEVERAL PARTS OF THE BRAIN AND NERVOUS SYSTEM, FURNISHING GUIDES TO THE CLINICAL INVESTIGATION AND DIAGNOSIS OF DISEASE.

In dealing with the diseases of the NERVOUS SYSTEM, as of other parts of the body, it is incumbent on the physician to ascertain, as correctly as possible, the *locality of the lesion, the nature of the affection,* and of the *morbid condition of the part affected.* Although it has been sometimes asserted that it is of little practical importance to discriminate accurately between diseases of one part of the brain or of its membranes and those of another, because the treatment may be the same for all; yet, for the sake of science—because "knowledge is power," and because the acquisition of such knowledge must eventually alleviate the sufferings and lessen the sorrows of humanity—the sooner such doctrines are ignored the better for the Science of Medicine; and, moreover, the majority of the medical profession are beginning to appreciate the principle that diagnosis should be carried as far as possible. "Though the practical gain directly to be ascribed to Hitzig's and Ferrier's observations on the localisation of cerebral function may not as yet appear to be great, still, as in many another important discovery, the gain may afterwards become apparent by richer fruits. Inquiry, at least, is stimulated, and new fields of investigation are opened up, some practical, and others speculative. The latter, the speculative class, relate to some of the deepest problems that can engage our thoughts—those, namely, that are concerned with the union between mind and matter. Objection has been taken to such studies for that very reason. It is urged that they lead to materialistic views of mind. But there is no reason why they should have that tendency; we are yet profoundly ignorant of the nature of mind, and of how it is associated with matter" (Dr. ALEXANDER ROBERTSON in *Glasgow Med. Journ.,* Dec., 1879, p. 421). And even "if the association of separate mental faculties with distinct convolutions were satisfactorily determined, we should be no nearer the solution of the mysterious problem, why the energy evolved from the molecular changes taking place in one group of nerve cells produces one kind of intellectual phenomena, and another group another kind. Or why from one brain proceeds a wealth of fancy and imagination which stamps its producer as a true poet; from another, speculation and discoveries on the constitution of mind and matter, which mark an epoch in science and philosophy; from a third, the merest commonplaces, or even absurdities, which prove their author to be a mediocrity or a fool; or why man, more than the brutes which perish, is endowed with a self-consciousness, with the power of distinguishing good from evil, right from wrong. All these are problems which lie beyond the range of anatomico-physiological inquiry; and, regarding them we might indeed say, in terms not unlike those used by the prophet of old—that 'eye hath not seen, nor ear heard, and it hath not entered into the mind of man to understand'" (TURNER, *West Riding Asylum Reports*, Vol. III., p. 29).

The following are elementary facts which must be kept in view:—

(1.) "*The nervous force, nervous power*, or *nervous influence*," which makes itself known by *sensation and motion*, also by the various ways in which the *intellectual and emotional* functions are expressed, constitutes the primary elements of inquiry as to *sensory, motor, co-ordinating, intellectual and emotional* phenomena. Each nerve-fibre in a fasciculus acts quite independently from end to end,—quite isolated from the others in its vicinity; and thus at once we have the enunciation of three distinct sets of phenomena associated with the diseases of the brain and nerves, to be accurately investigated. These are,—

First, Phenomena of Isolated Conduction.—Impaired, exalted, or perverted action is presented by those nerve-fibres only which are affected by the irritating or depressing cause; and the adjoining fibres, though in ever such close approximation, are not implicated.

Second, Phenomena of Sympathy or Irradiation of Sensations.—That irritation may be propagated from a fibre originally excited to other centripetal nerves.

Third, The Phenomena of Intelligence.—The brain, through its cortex and central ganglia, furnishes the conditions necessary for the manifestation of the intellectual faculties properly so called, such as the Emotions, Passions, Volition, and it is at the same time essential to Sensation. The evolution of power or nerve-force immediately connected with Mind, emanates from the supreme centres, the hemispherical ganglia; and clinical observation points out that in those cases in which disease has been found to commence at the circumference of the brain, and proceed towards the centre, the mental faculties are affected *first* (e.g., in *meningitis*); whereas in those diseases which commence at the central parts of the organ, and proceed towards the circumference, the mental faculties are affected last—e.g., *tumors* in the central white substance.

(2.) The spinal cord, by its connection with the brain, furnishes conditions necessary for combined movements; and clinical observation shows that in cases where the *central portion* of the cord is affected previous to the external portion, the individual retains the sensibility and power of moving the limbs, but wants the power to stand or walk; whereas, when disease commences in the *meninges* of the cord, pain, twitching, convulsions, numbness or paralysis, indicate lesion in the white conducting matter.

(3.) The brain alone furnishes conditions necessary for *Intelligence;* and with the other parts of the nervous system those conditions which are concerned with the *balancing and co-ordination of motor and sensific power.*

Any part of the nervous system may be the seat of disease, giving rise to symptoms indicating the special functions of the parts affected, and the extent of the lesion. The symptoms may be significant of the specific nature of the disease, and vary according as the following parts are affected :—(1.) Portions of the motor tracts; (2.) portions of the co-ordinating tracts and centres; (3.) portions of the sensory tract; (4.) the peripheral nerves; (5.) the cord; (6.) the intracranial centres.

I. *Localisation of Brain Disease.*—The grounds of diagnosis of the locality of nervous diseases generally may be summed up as follows, supplementary to the functional results obtained by Dr. Ferrier :—

(A.) The *brain* is presumed to be the seat of lesion when several of the special senses are simultaneously affected ; when *Perception, Ideation, Volition,* and *special Sensation* are affected; when the muscles and general sensory nerves are implicated longitudinally and unilaterally (hemiplegia); when muscles situated so high as those of the face and tongue are involved, and the *orbicularis* of the eyelids does not share in their affection. In those rare cases of bilateral (or transverse) paralysis (*paraplegia*) resulting from some cerebral change, the symptoms at some period of the case have generally referred to the head (by their special character), so that, by a combination of the two classes of observations, the general diagnosis may almost universally be established.

(B.) The *spinal cord* is presumed to be the organ affected when the symptoms of motory and sensory character are distributed transversely or bilaterally, inducing paraplegia or transverse spasms ; the mental functions being unchanged. The precise locality may be estimated sometimes from a knowledge of the anatomy of the spinal nerves. If the lesion or disease is high, speech, deglutition, or respiration may be impaired. There is often erection of the penis, and the retention or voluntary discharge of fæces or urine.

(C.) The *nerve-trunks* are presumed to be the seat of lesion when the symptoms are referable to an isolated muscle, or group of muscles, or to a small portion of the sensory surface.

In diagnosing the locality of diseases of the brain generally, it is necessary to distinguish, in the first instance, the *intrinsic diseases* of the nervous system ; also, local diseases from nervous complications of other diseases not of a local kind. It is necessary also to distinguish affections of the *brain, spinal cord,* and *nerves,* as much as possible from each other ; and, lastly, to separate diseases of the *meninges* from *cerebral lesions.*

CHARACTERS OF MENINGEAL AS DISTINGUISHED FROM CEREBRAL DISEASE.

CEREBRAL DISEASE.	MENINGEAL DISEASE.
1. From the outset, or from a very early stage of development, there is loss of some one or more of the proper nervous functions, such as paralysis, anæsthesia, loss of memory.	1. It is not till some time after the detection of signs of disease that diminution or loss of nervous function takes place.

2. Cerebral disease is not commonly attended by high-marked exaggeration of function, such as furious delirium, convulsions, intense hyperæsthesia, pain or tenderness.	2. The subsequent diminution or loss of nervous function which succeeds the prolonged existence of "head symptoms" is generally preceded, in cases of meningeal disease, by extremely severe excitement or exaggeration of functions, such as pain, tenderness, furious delirium, or convulsions.
3. Little vascular excitement attends cerebral disease, nor is there frequently any highly marked general disturbance.	3. In meningeal affections there is usually much local vascular excitement, with general disturbance.
4. Paralysis and anæsthesia, losses of volition, ideation, perception, and the like, characterise cerebral disease.	4. Spasms, convulsions, pain, and delirium, are the general features of meningeal disease.

It is chiefly by the history of the case that nervous symptoms peculiar to the *Specific* or *Constitutional* class of diseases are to be distinguished. It is also generally worthy of notice that symptoms referable to altered nervous functions, are the earliest indications of intrinsic or local diseases of the organs of the nervous system; and that when general disease exists of a *Specific* or *Constitutional* kind, the nervous symptoms are secondary in relation to the time of their appearance, compared with the earliest manifestation of symptoms of ill-health.

The diagnostic value of *vomiting*, as a symptom of cerebral disease, is one which must be thoroughly appreciated and never lost sight of. The intimate sympathy subsisting between the stomach and the head is a matter of daily observation. Headache from gastric disturbance is as common as vomiting from cerebral derangement. In children especially, the existence of obstinate but easily accomplished vomiting is more frequently indicative of head rather than of stomach disease.

A consideration of the following points (for comparison tabulated) may be useful as showing that while vomiting may depend upon derangement in the gastro-intestinal canal, it may also depend upon increased sensory or reflex action, and is thus a valuable indication of cerebral disease.

CONTRAST BETWEEN GASTRIC AND CEREBRAL VOMITING.

GASTRIC OR HEPATIC VOMITING.	CEREBRAL VOMITING.
1. There is nausea, which is relieved, at all events temporarily, by the discharge.	1. Little or no nausea, and the vomiting continues, in spite of the complete discharge of its contents by the stomach, so soon as anything (liquid or solid) is introduced.
2. The tongue is foul, the conjunctivæ often yellowish, and the headache secondary in respect of time.	2. The tongue may be clean, the conjunctivæ colourless or injected, and the headache primary.
3. Griping pain in the abdomen, diarrhœa, and disordered evacuations frequently attend the *gastric* or *hepatic vomiting*.	3. Obstinate constipation generally attends *cerebral* vomiting.
4. Retching and increased salivation attend *gastric* or *hepatic vomiting*.	4. In *cerebral* vomiting the stomach is emptied almost without effort, and without any increase of the salivary secretion.

Dr. David Ferrier having so far localised "the functions of the brain," as described in previous pages, proceeds to localise cerebral disease, as the complement of his previous investigations, from a clinical and pathological standpoint of these functions; and the question which attracts attention at present is, "Whether different regions of the cerebral hemispheres have different functions? and whether, therefore, the symptoms of cerebral disease vary with the locality of the lesion?" In the face of many difficulties which attend this part of the inquiry, Dr. Ferrier states some principles which must guide research and determine conclusions. These are:—(*a.*) We cannot assume any causal relation, unless the lesion in question is constantly (or more frequently than chance could account for) associated with the same symptoms; (*b.*) sound induction being once established, any statement in opposition must be regarded as incredible. In considering localisation of brain-function, it is necessary to bear in mind that the *functions* and *diseases* of the brain manifest themselves under two aspects; namely,—(*a.*) the psychological, and (*b.*) the physiological, the phenomena of each of which appeal to two distinct methods of investigation, subjective and objective. The following *axioms* also commend themselves, otherwise they would not be axioms:—1st Axiom,—The brain is the organ of the mind; 2nd Axiom,—When mental aberrations exist, the brain is diseased organically; 3rd Axiom,—Brain is necessary to sensory perception and voluntary motion. But as far as investigation has gone, "diseases of the brain do not affect psychological and physiological functions equally." Diseases which produce very obvious affection of motility and sensibility, cause no obvious mental disorder; and diseases which are capable of producing the most profound mental disturbances do not necessarily affect motions and sensation. 4th, Almost an axiom has been taught since the time of Aretæus, and been accepted alike by physiologists and physicians—namely, that "When paralysis results from lesion of cerebral hemispheres, it occurs on the side opposite the lesion." But there are also cases (1 in 1000) in which the paralysis is direct, or on the same side. Hence exceptions to the "all but" universal law have been noted, so that what otherwise would be an absolute rule is converted into an "approximate generalisation." It is a possible occurrence, explicable in special cases from the unusual development of motor strands in the brain.

Hughlings Jackson has shown that convulsions of cerebral origin are dependent on irritation of *discharging lesions* of certain *convolutions near*, and *functionally related to, the corpus striatum.* He thus indicated for the first time, in 1864, the motor functions of certain regions of the cortex; and so explained the phenomena of unilateral cerebral convulsions, as in hemiplegic epilepsy, where consciousness is observed to be retained during the fits. He regarded certain convolutions as themselves motor, and capable (by irritation) of motor discharge. In 1870 Fritsch and Hitzig made their important experiments, which showed that "though electricity might be applied to some portions of the cortex without producing movements, there were other portions of the cortex excitation of which invariably produced movements of the opposite side;" and, also, that "certain movements could uniformly be caused by excitation of certain definite regions"—facts that have been verified on many animals, and also on man. Having regard, therefore, to the significance of these facts, it is desirable to restrict observation to those movements which result from excitation of a certain region of the brain—namely, the motor region (page 93, *ante*)—so as to avoid confusion with other movements which are indications of sensation. The phenomena in question are the result of excitation of the functional activity of the *grey matter* of the hemispheres, and the direct relation of the *cortical grey matter* to movements has been established beyond a doubt—(1.) By experiments on local excitation by electricity; (2.) by localised destruction of the cortex; (3.) by the facts of human pathology.

The effects of localised destructions of the cortex are clear and decisive in the same direction as the experiments of excitation. These experiments were made mainly on monkeys, confirmed by observations made regarding disease on man himself. In monkeys, *destruction of those regions* of the cortex excitation of which gives rise to definite movements of the limbs, causes paralysis of voluntary motion, complete and enduring, and restricted to those very movements the centres of which are specially destroyed. What is true of monkeys is also true of man. The great fact towards which all these experiments on different animals point, is that the same movements have a plurality of causes, and are represented with different significations in different centres,—higher and lower,—(1.) Those movements which involve conscious discrimination (volitional in the strict sense of the term) are those alone which are necessarily paralysed by destruction of the cortex: (2.) those movements which are variously described as automatic, instinctive or responsive (including all the motor adjustments concerned in equilibration, locomotor co-ordination, and instinctive emotional expression), are more or less completely and independently organised in the centres situated below the cortex. We must therefore, (1.) clearly distinguish between the different KINDS of movements and their respective centres; and, (2.) regard paralysis of truly *volitional movements*, or those involving conscious discrimination, as the only essential feature of *cortical lesions*. Thus Goltz's experiments on dogs show that, though a dog's paw is not permanently paralysed as an organ of locomotion by destruction of the cortex, yet it remains *permanently paralysed for all those actions* in which it is employed as a *hand*. The poor dog cannot pull or push the door to open it with the fore-paw which he once used as a hand for that purpose. The conclusion to be drawn from the results of experimental physiology, and which are justified by the facts of human pathology are—(1.) That there are certain regions in the cortex to which definite functions can be assigned; (2.) that the phenomena of cortical lesions vary, (*a.*) according to their seat, (*b.*) according to their character—viz., as irritative lesions, or destructive lesions, two classes into which all the lesions may be theoretically reduced.

The experiments of physiology necessitate the strictest topographical accuracy in the position and limits of individual centres. It is also of vital importance that the same accuracy should be observed in respect to the situation of lesions in the human brain. Most of the older records of cerebral disease have to be discarded unless supplemented by drawings, specimens, or descriptions of the position of the lesion in relation to certain fixed points. Many of such cases can now be read in the light which recent experiments throw upon them—but may not be used to establish the laws,—only to confirm them. It is necessary—(1.) To exclude as valueless all observations in which the topography of the lesion is not indicated with rigorous exactitude; (2.) to select and reject the majority of cases of multiple lesions, and all those in which the lesion is diffuse; (3.) to accept with extreme caution cases of tumor compressing without destroying the cerebral convolutions; for the effects of pressure may be felt at a distance from the seat of lesion, and complicate the results (FERRIER, l. c.)

The lesions which are of special value to be studied are—cases of wounds, lacerations, or loss of substance, with various forms of chronic degeneration, such as atrophy, necrosis, and the like; and the results of hæmorrhage and inflammation, which, though at first complex, subside into local lesions, such as cicatrices, cysts, softenings, abscesses; or, in general, all lesions which exclude meningo-encephalitis, mechanical compression, or general cerebral disturbance. The exact nature of the lesion is not of importance, except in so far as it is one causing, (*a.*) irritation, or (*b.*) destruction of cerebral tissue. The results of physiological experimentation are still, however, in advance of pathology as regards precision and exactitude.

The following are some of the pathological results:—

A. *Lesions of the Frontal Lobes.*—The frontal lobe includes,—*Superior*, *middle*, and *inferior frontal* convolutions, and the *ascending frontal* or *præcentral convolution*, together with the *orbital* and internal aspect of the corresponding region. But for physiological and pathological purposes it is necessary to subdivide this region, and to term that part which, in its relation to the skull, is roughly bounded by the coronal suture, the *præfrontal lobe*, or *antero-frontal region.* (*a.*) Effects of experiments on the monkey in this region by electrical irritation are negative—*they cause no motor reaction.* (*b.*) Sudden and extensive lacerations may be made in the *præfrontal region*, and large portions of the brain-substance may be lost without causing impairment either of sensation or of motion, and without very evident disturbance of any kind, bodily or mental, especially if the lesion be unilateral. There are multitudes of cases on record in which these regions have been the seat of extensive disease on one or both sides with a like negative result as regards sensation or motion; and recovery has taken place after the most frightful lacerations and loss of cerebral substance—*e.g.*, American crowbar case, and many other cases quoted by Ferrier. (*c.*) Of cases of softening or abscess in this region (præfrontal) many are on record without any objective symptoms (FERRIER, *Localisation of Brain Disease*, p. 33). (*d.*) Marked mental deficiency, however (without any objective symptoms as regards motility or sensibility), has been frequently noted in connection with arrested development, or atrophy of the frontal lobes; and the frequent association of idiocy with absence or impaired development of the frontal lobes is a commonly recognised fact.

Generally, as to the præfrontal lobes, we cannot attach objective motor or sensory functions to them; for when injured or experimented upon or diseased, there is a total absence of affection of motility or sensibility, whether in connection with sudden or slowly progressive lesions. Hence, positive indications of disease of the præfrontal lobes must be very obscure, and cases in which sensation or motion has been affected with lesions in the præfrontal lobes cannot be regarded as other than cases of coexistent or multiple lesions. Between lesions of the præfrontal lobes and such symptoms we have no grounds for assuming even indirect causal relationship. Such relationship must, however, be admitted in the case of tumors, because they influence regions at a distance from their actual site, or in the case of disease—such as encephalitis or meningitis spreading backwards—*i.e.*, to the invasion of the motor area of the brain.

The diagnosis of lesions in the præfrontal region is to be made chiefly from indirect or accessory symptoms—*e.g.*, indications from the history of some traumatic lesion, or from the proximity of some affection of the nasal fossa, or from some disturbance of the sense of smell. But in the absence of all these, the question arises, "Are there any symptoms directly indicating disease of the præfrontal lobes?" Although none are pathognomonic, experimental physiology and the observation of injuries have brought into prominence certain facts which are of importance in answering this question. "Removal or destruction by the cautery of the antero-frontal lobes is not followed by any definite physiological results. The animals retain the appetites and instincts, and are capable of exhibiting emotional feeling. The sensory faculties—sight, hearing, touch, taste, smell—remain unimpaired. The powers of voluntary motion are retained in their integrity, and there is little to indicate the presence of such an extensive lesion, or a removal of so large a part of the brain. And yet, notwithstanding this apparent absence of physiological symptoms, I could perceive a very decided alteration in the animals' character and behaviour, though it is difficult to state in precise terms the nature of the change. The animals operated on were selected on account of their intelligent character. After the operation, though they might seem to one (who had not compared their present with their past) fairly up to the average of monkey intelligence, they had undergone a considerable psychological alteration. Instead of, as before, being actively interested in their surroundings, and curiously prying into all that came within the field of their observation, they remained apathetic or dull, or dozed off to sleep, responding only to the sensations or impressions of the moment, or varying their listlessness with restless and purposeless wanderings to and fro. While not actually deprived of intelligence, they had lost, to all appearance, the faculty of attention and intelligent observation" (FERRIER'S *Functions of Brain*, p. 230). With regard also to the psychical characters of some of the cases of disease and injury of the frontal lobes in man, they are in many respects similar to those seen in monkeys after removal of the præfrontal lobes; as, for example, the American crowbar case, and many others mentioned by Ferrier. The observations of Dr. Crichton Browne on general paralysis of the insane "also tend to show that the earlier symptoms, *inter alia*, are—general restlessness and unsteadiness of mind, with impairment of attention, alternating with apathy and drowsiness, which coincide with the morbid changes occurring at this stage, more particularly in the frontal regions" (*West Riding Reports*, p. 223).

The psychical phenomena are all-important here with diagrams of lesions of the præfrontal lobes.

B. *Lesions of the Motor Regions.*—These regions, called now "the motor area of the brain," have been definitely determined by experiments on monkeys. The area includes the bases of the three frontal convolutions, with those bounding the fissure of *Rolando*—namely, the *ascending* frontal and *ascending parietal* with its superior continuation, termed the *postero-*parietal, together with the inner aspect of the same, called the *paracentral lobule* by the French pathologists. In this region there are situated certain definable areas, stimulation of which by the electric current gives rise to certain definite movements *on the opposite side*—namely, of the leg and hand, facial, oral, and lingual muscles; while complete destruction of the defined area causes complete paralysis of all those movements, and limited or dissociated paralysis if individual areas only are destroyed—the paralysis in this case being confined to those movements which are excited by irritation of the same. It is of importance also to bear in mind how this region of the brain is supplied with blood—namely, by the middle cerebral or Sylvian artery, by four or five branches, each of which nourishes a special area (see page 51, *ante*). This artery may be occluded in its main trunk, or in its individual branches; and the arrangement is such that the arteries of the *basal ganglia* may still remain pervious, though the cortical arteries are occluded by emboli. Hence it is that softening, limited to the *cortical grey matter* and subjacent medulla, may and does frequently occur while the *basal ganglia* remain intact.

The lesions which invade this region may also be arranged into two classes, according as they are (1.) *destructive*, (2.) *irritative*. But practically, a complete separation of the two cannot always be maintained; for "lesions which result ultimately in total disintegration and paralysis, are not unfrequently associated with irritative or convulsive phenomena, which may be limited or more or less general."

There is now a large body of evidence which shows—(1.) that destructive lesions of this *motor area* invariably cause paralysis of voluntary motion; (2.) that there is a differentiation in this part of the *cortex* of distinct centres corresponding in situation to those experimentally established in the brain of the monkey, destructive lesions of which cause limited or dissociated paralysis of their respective movements or muscular combinations. But, on the other hand, it is not possible in all cases of purely *cortical paralysis* to demonstrate lesions in these centres; and, at the same time, there is no evidence, no case on record of the occurrence of a destructive lesion in the motor area *not* associated with motor paralysis. A cyst or tumor may appear to lie in the cortex of the motor zone; but if the grey matter is not injured, motion will remain, as such cysts or tumors push aside without destroying the tissue.

The nature of the experimental evidence is of this kind in the case of monkeys. The brain is exposed, and Ferrier then applied that which is universally recognised as an excitant of nerves and nerve centres—namely, electricity to a certain spot, which was followed by a definite movement. All other conditions remaining the same this spot is destroyed by the cautery (a complete destroyer of animal tissues); and immediately there is paralysis of the muscles formerly thrown into action by the electrical stimulus (Ferrier, *Localisation of Cerebral Disease*, p. 40).

Destructive lesions of the *motor area* are thus either, (*a.*) general, or (*b.*)

partial. The general lesions in the monkeys are extensive, represented by general disorganisation of the cortical area, and paralysis of voluntary motion (without affecting sensation) on the opposite side of the body, possessing all those features which characterise ordinary cerebral paralysis—namely, hemiplegia, which, though at first absolute, gradually subsides into a condition in which there is complete paralysis of all the most volitional movements, while associated, alternating, or bilateral movements are more or less spared. Hence the hand is more paralysed than the arm; the arm more than the leg; and the lower facial movements more than the upper; while the muscles of the trunk are scarcely, if at all, affected. A limited region (in the middle of the ascending frontal convolution), when stimulated, caused supination and flexion of the forearm; and when the area was completely destroyed the result was paralysis of motion, limited to this particular movement.

These are examples of general and partial lesions of the cortex induced by experiment, and under circumstances free from complications. But lesions induced by disease are not so simple. Apart from traumatic lesions cases of cortical disease in that part of the human brain corresponding to the like motor-area in the monkey, show that similar lesions in the human brain produce paralysis of voluntary motion on the opposite side of the body—a hemiplegia like that resulting from destructive lesion of the *corpus striatum*, or, more particularly, of the anterior part of the internal capsule. This paralysis is frequently associated with rigidity or convulsive spasms in the paralysed parts, particularly in the early stage; and if destruction of the cortical substance be complete, the paralysis is of permanent duration, and sooner or later it is followed by late rigidity, and secondary sclerosis of the motor tracts. This degeneration is traceable in the medullary fibres, crus cerebri, pons, and pyramid of the medulla oblongata on the side of the lesion; and thence, mainly on the opposite side of the spinal cord, in the posterior part of the lateral column; while a corresponding band of secondary degeneration frequently exists on the internal aspect of the anterior column of the same side as the lesion (TURCK, CHARCOT, FLECHSIG, quoted by FERRIER, l. c., p. 45).

These are exactly the tracts which have been shown to be the continuation of the pyramidal strands; and the fact of the direct continuity of this degeneration with that commencing under the cortex proves, in accordance with Waller's and Türck's researches, the direct motor signification of the cortical regions to which these fibres are traced; and Türck's observations on the secondary affections of the cord seem to show that when disease destroys a certain portion of the nervous centre, the strands, filaments, or cords of nerve-substance which proceed from that centre, or arrive there, subsequently degenerate, having ceased to receive or convey an impulse. This degeneration takes place in the same direction in which these strands or filaments convey impressions; thus, in the centripetal fasciculi (posterior columns) the secondary affection occurs always in the centripetal direction; while in the centrifugal fasciculi (anterior columns) it shows itself in the centrifugal direction; and in the mixed fasciculi (lateral columns) it shows in both directions. Thus it happens that secondary affections of filaments or strands of fibres in the spinal cord may result from a lesion in one of the hemispheres of the brain, generally in about five weeks after the primary injury. This observation may be regarded as a rule; and to some extent it may also explain cases of so-called "*paralysis musculaire progressive*" which have been described by Meryon, Cruveilhier, Aran, Valentiner, and Roberts; although the nervous lesion has only been demonstrated in some of the cases.

It has also been shown by experiments on dogs, that secondary degeneration of the medullary fibres of the centrum ovale occurs after destruction of the cortex; and that secondary degeneration in the spinal cord occurs after removal of the limb centres. Ferrier quotes a case in which a caseous degeneration involved the *ascending frontal*—the bases of the three *frontal convolutions*, the *ascending parietal*, and *postero-parietal* lobule, and corresponding internal aspect of this region, or paracentral lobule,—the phenomena were lateral convulsions of the opposite side, followed by complete hemiplegia, without loss of sensation.

The cases all tend to show that, from purely cortical lesions in the motor tract so defined, permanent paralysis may result—the ganglia being intact; and that in consequence of such lesions, secondary degeneration takes place in the motor tracts, with its accompaniment, late rigidity.

II. *Nature of the Affection.*—The nature of the intrinsic diseases of the nervous system may be distinguished by the following clinical phenomena.

A. *Acute*, but *non-febrile*, to distinguish them from the nervous symptoms which attend and are peculiar to the febrile state of many of the (General Diseases, Sections A and B) diseases already noticed. They are of such a kind as are marked by—(1.) Diminution or lose of functional activity (apoplectic and paralytic diseases). The group of symptoms called "*apoplectic*" are loss of consciousness and of voluntary motion, as if by a sudden "*stroke*." (2.) Increase or excess of functional activity, such as of sensibility (*neuralgia*), of mobility (*convulsions, spasms*), ideation (*delirium*); exalted, perverted, or suspended functional activity, such as in *chorea, hysteria, catalepsy, epilepsy*, some diseases of the intellect.

B. *Chronic* diseases, the character of chronicity depending not only on the time such diseases last, but also on the severity of their course.

Such chronic diseases are marked by—1. Excessive functional activity, as by *neuralgia, hallucination, chorea, hypochondriasis*. 2. Impairment, diminution, or loss of functional activity; for example, *anæsthesia, paralysis, dementia, epilepsy*. 3. Combinations of these conditions, such as—(1.) Loss of mobility, with increased sensibility, as in *paralysis with pain;* (2.) loss of mental, with increased motor activity, as in *coma* with *spasms;* (3.) loss of sensibility, with increased mobility, as in *anæsthesia* with *reflex spasms.*

It is of great importance also to recognise clinically the differences between the symptoms of—(1.) Loss of function; and, (2.) irritation.

The most frequent cause of local paralysis or loss of function is an irritation in certain parts of the nervous centres, or in the trunk or periphery of nerves. Such circumstances may produce loss of function (*paralysis*) in very different parts in different cases, according to the particular fibres on which the irritation has acted. Thus, there are now numerous cases, capable of clinical recognition, where paralysis of the upper and lower limbs, and of the face, as well as of contractions and rigidity, which are traceable to the irritative influence of reflex action.

Symptoms of irritation may be arranged into two groups, according as the irritation acts—(*a.*) In the central parts, as in convalescence from such serious illnesses as *enteric fever*—overtaxing the mental powers, as in the case of weak children; or, (*b.*) in the peripheral parts of nerve-fibres, as in the neuralgic headache of dyspeptic children during the second dentition, when the symptoms frequently resemble those of the first stage of *tubercular meningitis.* Convulsions are not rarely due to Bright's disease, to the irritation of ascarides; and they may also occur concomitantly with the second dentition, just as they occur in the first.

Dr. Brown-Séquard is of opinion that phenomena of irritation are sufficient to account for those diseases of the encephalon in which actions of arrest are exercised on different regions by regions neighbouring to the foci of irritation. Hence he is opposed to the doctrines of Ferrier and of Charcot; and generally, the essence of his views is, that symptoms of paralysis, anæsthesia, aphasia, and the like, are due to irritation, and not to cessation in the function of various parts of the brain; for irritation of parts around the portion destroyed by disease causes certain sensations, not that the parts destroyed cause them, but because an irritation starting from the place around influences cells, some near, some at a considerable distance from the locality of the lesion. He therefore believes that certain functions of the brain, instead of being localised in clusters of cells, are, on the contrary, spread over the greater (if not the entire) part of the brain. He considers that this theory explains a very large number of cases of disease which otherwise it would be impossible to understand.

III. *Anatomical Morbid Condition.*—Although some of the diseases of the nervous system are marked by excessive severity of symptoms during life, such as *tetanus, epilepsy, chorea, hysteria, neuralgia,* and the like, yet no characteristic or constant structural change can be detected in the nervous centres after death, either as a consequence or as a cause of such diseases; and although the belief is daily extending, that no morbid conditions of function can exist without some correspondent change in the organs, yet, so long as we have no means of appreciating such changes, the diseases now noticed must be regarded as "neuroses," "dynamic" or "functional" diseases.

But there are many other diseases of the nervous system which are attended by some physical changes in the organ, expressed by undoubted symptoms during life, and which leave evidence of their existence after death. For example, very different apparent vascularity is discoverable after death in the nervous masses and texture; and there are two very common classes of nervous symptoms during life which evidently depend upon the variable amount of blood in the brain. These symptoms are referable to *active arterial hyperœmia,* and are distinguished by the well-known phrase of "determination of blood to the head;" or they are referable to *passive venous hyperœmia,* commonly called "congestion." The question has been much discussed and experimented on as to whether more than a fixed proportion of blood can find its way into the brain; but all considerations of the subject lead to the conclusion that the quantity of blood within the cranium is extremely variable at different times and under different circumstances; and (as clearly stated by Dr. Sieveking) there is a peculiar property belonging to the white matter of the brain which has a strong bearing on the question—namely, the great elasticity of the medullary tissue, so much so that the resiliency afforded by this property is a sufficient counterpoise to the rigid structures which envelop the brain, and which do not, as is erroneously supposed by some, remove the intracranial contents entirely from the influence of atmospheric pressure. That pressure is exerted on a large surface, composed of columns or tubes of blood in innumerable small curved vessels, which maintain, through the scalp and diploe of the skull, a direct communication with the blood within the cranium; and which is thus directly influenced by atmospheric pressure, while every anatomical arrangement of the part within the cavity of the skull illustrates provisions made to counterbalance the varying interchange of bulk between the solid and fluid contents of that cavity. Among these may be noticed the ventricular and subarachnoid spaces, with their varying amount of contained serosity, as furnishing most prominent evidence of a provision to accommodate the varying amount of fluids within the cranium. (See page 53, *ante*).

Morbid states of the brain are also due to a poisoned state, or to a state of *hyperpyrexia* of the blood, although such a condition cannot be proved in all cases; such, for example, as occurs in many of the general diseases already noticed, as *Typhus, Variola, Rheumatism;* and from the action of *Alcohol, Opium*, and *Urea*. It is often of importance to determine whether urea circulating in the blood is contaminating the brain and impairing its functions. The detection of urea in the brain after death, and its discovery in the blood during life, may be done as follows:—

"1. *In the Serum*.—Take the serum from a good-sized blister, and evaporate it to dryness over a water-bath. The residue is to be extracted with alcohol, which is a ready solvent of urea. This alcoholic extract

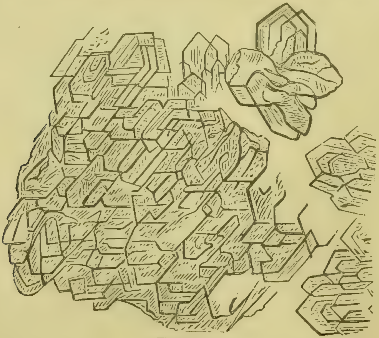
Fig. 15.*

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* Nitrate of Urea (after BEALE). No II. Urinary Deposits, Plate III.

is then to be evaporated to dryness, and a little water added, so as to make a syrupy mass, which should be plunged into a freezing mixture, and a few drops of pure nitric acid added to it. If urea be present, the characteristic crystals of nitrate of urea are soon found in the solution, and may be recognised either by the naked eye or by the microscope (Fig. 15).

"2. *In the Substance of the Brain.*—Take about three-fourths of a whole brain, and cut it up into small pieces. Then treat it with four successive portions of boiling distilled water, each portion, consisting of about ten ounces, being allowed to stand six or eight hours before the next is added. The brain while thus macerating should be frequently stirred and mashed about with a glass rod. The washings, after being poured off, are to be mixed together and filtered. The filtered aqueous extract so obtained must be evaporated to dryness over a water-bath, and the dry residue, after being powdered, is to be again treated with four successive portions of boiling distilled water, observing the same precautions as before. The washings, after being mixed together as before, are to be filtered, and the clear solution evaporated to dryness over a water-bath; and after being thoroughly dried in a hot-water oven, the residue obtained in this manner should be finely powdered, and the powder boiled in five successive portions of ether. The ethereal extract so obtained should be evaporated to dryness at a low temperature, and then treated with a little tepid water, and allowed to get quite cold. It is then to be filtered through paper previously moistened with water, and the clear solution again evaporated to dryness at a low temperature, when a small quantity of the extract procured in this way (which would contain all the urea present in the brain operated upon) is to be placed on a glass slide, treated with a drop of strong nitric acid, covered with a bit of thin glass, and allowed to stand a little time, and then examined under the microscope. A few crystals will then be seen, having all the characters of those of nitrate of urea."

IV. *Records of Cases.*—For the complete study of nervous disorders, there are some points of prime practical importance to be considered and recorded—namely, *age, diathesis* or *constitution and temperament;* the nature of *the secretions and excretions,* especially that of the *urine;* and also, the results of observations by the *ophthalmoscope* and *æsthesiometer.*

SECTION II.—FUNCTIONAL DISORDERS AND SUBSTANTIVE DISEASES OF THE BRAIN AND NERVOUS SYSTEM.

In describing these Substantive Diseases, it is necessary also to take into consideration certain Special Symptoms and Important Phenomena significant of Nervous Diseases, which are dependent on the Situation of the Part affected, and on the Degree or Mode of its Involvement.—These are especially—(1.) Pain generally, dysæsthesia, hyperæsthesia, neuralgia, anæsthesia, headache, and spinache; (2.) vertigo or dizziness; (3.) convulsions and spasms; (4.) motor paralysis; (5.) influence of nervous diseases over processes of nutrition; (6.) progressively ascending, descending, and collateral lesions; (7.) peripheral, central, and reflex sources of disease, with their consequences; (8.) impairment and loss of power of speech (aphasia, aphemia, amnesia); (9.) intellectual and emotional disturbances.

(1.) *Pain* "expresses an ultimate fact of human consciousness—a primary experience of the human mind resolvable into nothing more general or more fundamental than itself" (Mr. ALEXANDER BAIN). It cannot be defined. It is a sensation which cannot be measured. We must depend upon the sufferer of the pain for all information about its amount and its quality. The words and language which express pain have no standard by which their use may be regulated. The existence of pain is mainly to be interpreted by the gestures, postures, and looks which any living creature exhibits; by cries, by the pathos or tone of the voice; by the expression and changes of the countenance, and by its effect on various organs and functions (Dr. J. K. SPENDER, *Therapeutic Means for the Relief of Pain:* Prize Essay, for which the Medical Society of London awarded the Fothergillian Gold Medal, 1874). The cause of pain is to be looked for in the impairment of nerves and of nerve centres. Physical changes are, no doubt, at the root of all pain. In perfect health anything causing pain is absent and unknown. The physiological calm is complete; and the first disturbance of the calm may be such initial tremblings of nerves as are called "tingling," "pins and needles." "By steps so gradual," writes Dr. Spender, "as to be almost imperceptible, each tiny impression of pain becomes sharper and prolonged; . . . and much depends upon the size and relative anatomy of a suffering nerve. Any sensation which is unusual, vehement, and agonising, is rightly called Pain. Going on further, we come to a stage at which a nerve can no longer mediate sensations; it is no longer able to be the carrier of either pain or pleasure. It is said to be '*numb*,' and to be the cause of '*numbness*.' It is not dead but physiologically defective. . . . Technically, all sensations which deviate from the normal, and are identified as pain, are classified as *Dysæsthesia*"—a term under which may be included "a large number of abnormal sensations, referable to the ordinary sensory nerves, to the nerves of special sense, and to the sympathetic system; or, at all events, to the afferent nerves connected with the visceral organs" (Dr. BRISTOWE, l. c., p. 939). The abolition of sensation is *pro tanto* called "*Anæsthesia*." There are also several grades of sensorial aberration, such as sensations "which hover between pain and not-pain; for example, *fierce itching*, implying a highly excited state of nerve peripheries, as in the irritation of prurigo and scabies; the sensation of '*aching*,' which fluctuates between the expression of fatigue and real pain, with an invariable tendency to abate if a wearied part be allowed to rest." There are also other sensations which may be enumerated under the head of dysæsthesia, such as *burning*, *cutting*, *crushing*, *constriction*, and other kinds of pain; *stabbing* and *shooting;* also, *numbness*, *coldness*, *tingling*, *formication*, all of which are perverted sensations referable to the skin. Spasms of muscle is also a frequent cause of pain; but a spasm may be so mild and unobtrusive as not to be associated with any particular sensation. *Tonic* convulsions or cramps are always associated with pain; but *clonic* convulsions, such as occur in chorea, are not attended by pains. *Hiccough* is a short convulsion; but the diaphragmatic spasm is not severe nor prolonged enough to compress nerves. The convulsions of asthma and of hooping-cough are more prolonged; but the pain which accompanies them is merged in the larger distress of the difficulty of breathing—a direct effect of the convulsive cough. The pain which attends spasms of some internal ducts (ducts from gall-bladder and ureters), and also of the uterus in child-birth, or extrusion of uterine tumors, is compounded of violent muscular contraction and the irritation of the body which provokes it (Dr. J. K. SPENDER, l. c.).

Most people know what Pain is by their own perceptive experience (Dr. P. M. LATHAM). It has been classified under the following varieties: (*a.*) *Inflammatory pain*, caused by or forming a part of the local disease described as "*inflammation*" (page 75, Vol. I.); (*b.*) *Irritative pain*, depending on nervous irritation, and having sometimes a local origin; (*c.*) *Reflex or sympathetic pain*, as the pain in the knee from hip-joint disease; the supra-orbital neuralgia and dyspepsia associated with caries teeth; the infra-mammary pain in ovarian disease; and the orchitis in gonorrhœa;* (*d.*) *Eccentric*, not originating in the painful part, but in the brain or nerve centres; as in hysteria, and the local forms of it, such as hysterical affections of the joints (Dr. HEADLAND, Fothergillian Prize Essay on Actions of Medicines, 1852).

In every variety of Pain, however, there is some molecular disturbance of the nerve which carries the idea of pain to the sensorial centre. It is thus that *Neuralgia* has become a substantive disease; and all recent inquiry tends to show that so far as it can be associated with physical change, that change is in the direction of "a lowering and degeneration of tissue." Those are the most acutely agonising neuralgias which occur under circumstances of impaired nutrition incident to the period of bodily decay; and there are strong reasons for the belief that there is especial impairment of the nutrition of the central end of the painful nerves—their central nuclei. A parallel to this variety of pain is only to be found in cases of organic tumors exercising continuous pressure on the branches or trunk of a nerve. Hence Pain always involves a depreciation of a function, and implies a perturbation of nerve force, the susceptibility to which is great in proportion to the physical imperfection of nerve tissue, until this imperfection reaches the extent of cutting off nervous communication altogether (paralysis).

Of the correlation which exists between *Delirium*, *Spasm*, and *Pain*, Dr. Spender also gives an excellent exposition. He agrees with the late Dr. Anstie, that the condition of a patient at the time of the first attack of neuralgia is one of debility, general or special. The sufferer is either decidedly anæmic, or he has undergone some exhausting illness or fatigue. The skin supplied by the nerves about to become painful is often experienced by the patient to have been first anæsthetic; and next, that sensation is blunted in the same parts during the intervals of pain. Hence the doctrine is justified which teaches that,—all causes that impair strength are prone to generate hyper-excitability—perturbation of nerve force—which may show itself in the sensory, or motor, or intellectual functions (Dr. HANDFIELD JONES, *Brit. Med. Journ.*, July 23, 1870). This doctrine is illustrated by the theory that Neuralgia, or Pain, Spasm, and Delirium are subjectively identical, though very different in their objective phenomena; and the same remedy may control (according to the circumstances in which it is administered) the Spasm of hooping-cough, the Pain of neuralgia, and the Delirium or "Wanderings" of alcoholism or of acute mania. An analysis of these three conditions presents itself as follows:—(*a.*) Derangement of sensory force, shown first by dysæsthesia or pain, then by anæsthesia or numbness; (*b.*) derangement of motor force, shown first by spasm, then by paralysis; (*c.*) derangement of intellectual force, shown first by delirium, then by coma. Thus, while the *spasm* of muscle, the *pain* of nerve, and the *delirium* of brain, are held to be correlative phenomena; the *paralysis* of muscle, the *numbness* of nerve, and the *coma* of brain, are similarly correlated. Dr. Spender also shows the ready interchangeableness of some of these phenomena (l. c., p. 11.) Thus "augmented or perverted sensibility has the same relation to the sensory part of the nervous system as spasms and convulsions hold to the motor; and the two conditions are not unfrequently associated."

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* Objection has, with justice, been taken to the explanation that such pain is due to reflex action (Dr. DAVID HART, *Practitioner*, July, 1878, p. 349). For a reflex action afferent and efferent fibres are necessary. The former are of necessity sensory; the latter may be motor, vaso-motor, vaso-inhibitory, cardio-inhibitory, or secretory. They are never sensory; for a sensory nerve is always afferent, and there can be no reflexion without descending fibres; and, it is difficult to understand how descending currents can drive ascending ones *down* sensory fibres. Dr. Hart gives good reasons for the hypothesis that hyperæmia of nerve centres, by an extension of irritation from one centre to an adjacent one, may help to account for this "metastasis of pain," in place of the reflex theory, which is untenable.

Hyperæsthesia does not necessarily imply pain, but is merely an exalted sensibility— "a condition in which the various organs of sense are more readily affected than they should be by impressions which are made upon them; or in which the sensorium is more appreciative than natural of the impressions which are conveyed to it from the organs of sense. But, practically, such exalted sensibility is scarcely if ever distinct from painful sensibility. The hyperæsthetic eye cannot bear bright light; the hyperæsthetic ear is affected painfully by powerful, high, or discordant sounds; the hyperæsthetic skin shrinks from the slightest pressure, or from mere contact. The condition is not uncommon, and is frequently observed in hysteria, sometimes in the early period of febrile disorders, occasionally in inflammatory and other affections of the central nervous organs. It is a common feature in a hemi-paraplegia, when the paralysed side is not only still sensitive, but its sensibility often becomes painfully acute. Inflammatory affections involving the skin are often also attended by hyperæsthesia" (Dr. BRISTOWE, l. c., p. 933).

With regard to the cause of pain being referable to hyperæmia or atrophy of the nerve structures, neither theory can be at present scientifically verified; but proofs seem to accumulate on the weak or atrophic side. The influence of therapeutic agents also points in this direction.

The terminology of pain is peculiar. It is often attributed to certain viscera or parts being painful, instead of to the nerves which enter or supply them. But the meaning of any part or structure being painful is this:— "the nerve threads which are bound up with the structure are involved in the causes which bring about the pain, and tell that tale of pain to the sensory centre."

The Principal Anatomical Seats of Pain.—The motor and sensory nerves, after the union of their roots, are commonly bound up together, although the separation of each individual nerve-fibre is anatomically complete. At one time it was taught by some that motor-nerves might (under some circumstances) be the seat or vehicle of pain; but there is no valid reason for believing this. All pathological and physiological evidence points to pain as "a modification or error of sensation, *conditionated* only by sensory nerves. The peripheral extremities of nerves ramify all over the skin, and the faintest injury which breaks the cutis is perceived by these nerves, and conveyed to the sensorium. A bruise, a prick, a scratch, and a cut cause sensations peculiar to each, so as to be instinctively recognised by the sufferer. Wounds of greater size and importance, scalds and burns, and the lesions caused by sprains, dislocations, and fractures, affect a number of sensory nerves, and produce complex sensations ranging from uneasiness to the acutest pain. The tracts of mucous membrane are less sensitive, but are specially resentful of the injurious influences of heat and cold; and serous membranes (when injured) may give rise to the sharpest agonies that can afflict the human body. Consequently, wherever there are sensory nerves there may be pain; and certain tracts can often be marked out as specially the source or seat of pain, and we can identify those tracts with the anatomical position of certain nerves" (SPENDER, pp. 19, 20). To pain in such tracts the name of Neuralgia has been given—often an indication of "central change, often also the cause of trophic and motor symptoms, which complicate sensory aberrations."

Pain, or Neuralgia proper, may be anatomically classified as—A. Superficial, B. Visceral (SPENDER, l. c.).

A. *Superficial Neuralgias* are again subdivided as follow:—(1.) *Trifacial Neuralgia, Tic Douloureux,* or *Neuralgia* of the trigeminus, which always exhibits itself with especial violence in certain foci (as first accurately defined by Valleix). These are always at points where the nerve becomes more superficial, either in turning out of a bony canal or in penetrating fasciæ. *Ophthalmic neuralgia* is the most common; and the most frequent foci of its pain are the supraorbital and parietal parts; also *brow-ague, hemicrania, megrim* or *migraine.* (2.) *Cervico-occipital Neuralgia,* affecting the first four pairs of spinal nerves, the great occipital being the most important. (3.) *Cervico-brachial,* which includes pain occurring in nerves originating from the brachial plexus, and affecting chiefly the shoulder, forearm, and hand: troublesome and severe, owing to the number of painful foci. The most common seat of brachial neuralgia is the ulnar nerve. (4.) *Dorso-intercostal Neuralgia,* affecting certain dorsal nerves, which have three points of division, with three sets of superficial branches, which make their way towards the surface near these points, and where the foci of pain are found. (5.) *Dorso-lumbar Neuralgia* is not common; but there are five foci to its pain, which is most often observed in the vertebral region over the *crista ilii.* (6.) *Crural Neuralgia,* almost always a secondary seat of pain to that form of neuralgia which primarily shows itself in the external pudic branch from the plexus. (7.) *Sciatic Neuralgia,* the most frequent, and therefore the most important. All the varieties have one or more foci of severest pain—those in the leg and foot being generally the most troublesome. Anæsthesia, convulsive movements of muscles, and paralysis, are not uncommon complications of sciatica.

B. *Visceral Neuralgias* are those in which other varieties of dysæsthesia are manifested. They are subdivided as follow (SPENDER, l. c., p. 22) :—(1.) *Cephalalgia*, or true brain neuralgia, to be identified as "headache," apart from pain of superficial nerves. Dysæsthesia of the organs of special sense may be indicated by the appearance of subjective phenomena referable to the organs of special sense; for example,—of the eye, by the appearance of sparks, or flames, or dark or luminous objects; or of objects in definite form, sometimes endowed with motion and assuming the visible attributes of life;—of the ear, by the perception of sounds, such as humming, buzzing, singing, the ringing of bells, violent explosions, even words and actual conversation;—of the nose, by the perception of odours;—of the taste, by the perception of flavours, for the development of which no extrinsic cause exists (BRISTOWE, l. c., p. 934). But all of these phenomena point (according to the present state of cerebral pathology) to lesions in those centres of the nervous system where the nerves connected with those special senses take their origin in the nuclear elements of those centres. (2.) *Cardiac Neuralgia*, or the agony of *Angina pectoris* and of mitral valve disease of the heart. (3.) The agony of distressing thirst and of craving for food. (4.) Indescribable sensations referable to different parts of the body, of which patients complain; or which constitute many of the varieties of the epileptic aura—all of which also point to definite damaged centres. (5.) *Gastric Neuralgia*, as in stomachache, heartburn, and gastrodynia, or "hyperæsthesia of the vagus nerve" (ROMBERG). (6.) *Enteric Neuralgia*—enteralgia or colic—characterised by cutting, tearing, or griping pains, sometimes accompanied with retraction of the abdominal walls and spasm of the abdominal muscles, and sometimes with distension of the whole cavity. Enteric and gastric neuralgias may and do often coexist. Lead colic is a special form of enteric neuralgia. (7.) *Hepatic Neuralgia* is characterised by the absence of pyrexia, while the pain is often more severe than in acute hepatitis, and is usually accompanied by tenderness of the hypogastrium and hypochondrium. (8.) *Renal Neuralgia*, the pain of which is not easy to distinguish from that caused by the passage of urinary calculi. (9.) *Neuralgia of the Bladder and Urethra* is a disorder which may be defined. (10.) *Neuralgia of the Testicle, and also of the Ovaries*, are by no means uncommon. (11.) *Uterine Neuralgia*, unconnected apparently with organic disease, has largely engaged the attention of obstetricians. It is now attributed, in most cases, to displacement of the uterus. *Menstrual colic* is also a name given to painful disorders of menstruation (EULENBERG). (12.) Laryngeal and Pharyngeal Neuralgia has been described by Dr. Anstie, but its identity is not quite clear.

Definitions of Certain Varieties of Pain (SPENDER, l. c., p. 24) :—(1.) *Myalgia*, or "pain produced in a muscle obliged to work when its structure is imperfectly nourished or impaired by disease," as in lumbago or stiff neck. (2.) *Spinal irritation* expresses "all those conditions in which, without any mental affection and without any single nerve being definitely affected, there are sensations varying between cutaneous tenderness and acute pain approaching neuralgia in character, together with fixed tenderness of certain vertebræ on deep pressure." Hysterical hyperæsthesia (often scarcely dysæsthesia) may be here mentioned. (3.) *Hypochondriasis*, a term given to phenomena which are manifested in a scattered form, affecting especially the spinal sensory centres, associated with characteristic psychical peculiarities. (4.) The pain of *Locomotor Ataxy* is characterised by intense darting sensations, chiefly in the limbs, certain nerve centres being diseased. (5.) Severe pain is part of the history of cerebral abscess and cerebral tumor. (6.) Pains of alcoholism are thus far characteristic, that they do not follow the course of a recognisable single nerve, but are usually present in more than one limb, and generally in both halves of the body at the same time. Here, as in locomotor ataxy, there is undoubted implication of nerve centres in disease. (7.) The pain of syphilis (see Vol. I., p. 912) is characteristic, terribly severe, and extensive, both in the earlier and later stages. (8.) The pain of rheumatism and gout (see Vol. I., pp. 843, 865) attacks many regions of the body, and so does that which complicates many of the constitutional and implanted or enthetic specific diseases. (9.) Spasms of internal ducts occasion pain of the sharpest kind. (10.) *Various morbid processes* cause pain, partly by reason of their position, and partly by the pathological changes produced by them. Inflammation and malignant diseases are characteristic agents in this respect. The ulceration of cancers often exposes extensive surfaces of most sensitive nerves. (11.) *Reflex irritation* may also cause pain. (12.) Neuralgia has also been classified according to the time of life at which it occurs; such as that which characterises—(*a.*) the period of bodily development (*growing pains*); (*b.*) the middle period of life; (*c.*) of general vital decay.

As a substantive disease the following account is given of ailments in which pain is a prominent symptom :—

NEURALGIA.

LATIN EQ., *Neuralgia;* FRENCH EQ., *Néuralgie;* GERMAN EQ., *Neuralgia;* ITALIAN EQ., *Neuralgia.*

Definition.—*Excruciating pains occurring in the course of nerves, and in their area of distribution, usually of a shooting character, which flash with momentary intensity along the fibres of the affected nerve, occurring in paroxysms composed of momentary shocks following one another in rapid succession* (BRISTOWE); *or returning with renewed violence in a part after periods of temporary remission, due to some unknown morbid state of the nerves of sensation or their centres, a symptom of some local lesion, or more commonly of a general affection or cachexia.*

Pathology.—All neuralgias are symptomatic either of an organic lesion, of which the neuralgic pain is a reflex expression; or, the pain is due to a more or less grave organic lesion, involving, compressing, or otherwise irritating various branches of nerves, and so giving rise to direct pain, as may arise from contusions, wounds, and impactions of foreign bodies; or, pressure, as takes place when bony channels, through which certain nerves pass, become contracted from any cause; or, when nerves are compressed by tumors or other adventitious means; or, on the implication of nerves in disease, as in the lesions of rheumatic or other varieties of inflammation; or, in syphilitic growths; or, in carcinomatous and other tumors. In some cases neuralgia is *symptomatic* of various cachexias—*e.g.*, of *hysteria*, of *chlorosis*, of *lead-poisoning*, of *anæmia*, of *malaria*, of *rheumatism*, of *syphilis;* or it is a *reflex* induction from an acute inflammation, as from a *caries tooth*, a *necrosed bone*, a *tumor*, or a *phlegmon.* The form of the neuralgia from these several sources may vary, but *pain* of the characteristic kind described in the definition is alike common to all. In most cases marked peripheral lesions are the starting-points of neuralgia, as in cases of decayed teeth, of necrosed bones, of tumors developed in the vicinity or in the substance of nerve-trunks, or of inflammation including nerves within its area (TROUSSEAU). In a considerable number of cases (often the most severe and incurable) no cause whatever—local or constitutional—can be discovered. Megrim or sick headache, and possibly also *tic douloureux*, are hereditary forms of neuralgia (BRISTOWE).

The pain of neuralgia is distinct in character, and involves the minimum of organic change in the part affected, while the whole circumstances of the affection point to lowered vitality as the antecedent cause of the pain. "The general bodily health is always at a low point when the attacks occur, and the nerves of the part are habitually in circumstances which must tend to lower their functional activity." Circumstantial evidence of this consists in the passive flow of tears; the hair, for example, of the eyebrow becoming decidedly grey at a point exactly opposite the *supra-orbital nerve* affected—so many indications of *defective nervous energy.* Pain under such circumstances is generally the direct consequence of a further depression of an already feeble vitality in the nerves (ANSTIE, *op. cit.*, p. 84). The lesion or local condition causing neuralgia may exist in the course of the implicated nerve, or may be seated in the spinal cord or brain; or may occupy some remote part, from which it acts indirectly.

The principal varieties of neuralgia have been mentioned at page 103, *ante.*

Symptoms.—The symptoms are similar in kind, whichever nerve is affected, modified only by the position, connections, and distribution of the nerve. Any sensory nerve may suffer:—*visceral* or *cutaneous.* Neuralgias of visceral parts are experienced in the heart, stomach, kidneys, uterus and ovaries, testicles, and mammary glands. The cutaneous neuralgias have been already named. Two forms of pain are to be distinguished, namely—one continuous, increased by pressure, confined to circumscribed spots or points in the course of the nerve; usually not a very severe pain, but a very annoying and persistent source of irritation,—aching, boring, burning, crushing, cutting, darting, stabbing, or tingling. Another form occurs in paroxysms made up of a number of sudden lightning-like shocks, spreading *from a point* along the course of a nerve. This kind of pain is terrible, and almost unbearable. It is sometimes also continuous or occurring in exacerbations passing upwards and sometimes downwards from a point; and the pain is felt to be deep-seated rather than superficial. Several varieties of neuralgia require more special notice.

(*a.*) *Facial neuralgia*, or *tic douloureux*, is described by Trousseau as an epileptiform neuralgia. It is the most severe and the most typical form of the affection. The several branches of the *trifacial* nerve may be attacked separately or conjointly on one side; most commonly, however, only one branch is affected, less frequently two; and the case must be severe in which the three branches, or the whole side of the face, is affected. Nevertheless, it sometimes so happens, extending even over the summit of the head, and over the temporal region, by the deep branch of the fifth pair, which emerges to the surface anterior to the external meatus. It may be also associated with a similar affection of the occipital branches at the same time. Next to the sciatic nerve, no nerve is so often the seat of pain as the *trigeminus*—a greater liability to neuralgia arising from two circumstances, namely—(*a.*) The passage of its branches through narrow canals, or openings in bones, where they are readily compressed (HYRTL); and, (*b.*) from the distributions of the nerve over a large cutaneous surface more exposed to cold and to changes of weather than any other part of the body.

An attack of facial neuralgia is sometimes sudden; but, more generally, it is preceded by a dull aching pain at the points where the nerve issues from the cranium, or becomes superficial. There are especially three points of pain in facial neuralgia, namely—the *supraorbital foramen; the anterior opening of the suborbital canal; the mental foramen.* These points lie nearly in a vertical straight line. If the neuralgia be limited to the *first* branch of the fifth pair, the pain spreads in the branches of the supra-orbital, affecting especially the forehead, eyebrows, and upper eyelids, occasionally the eye itself. After this threatening symptom has lasted a few hours or a few days, the patient is seized with a violent darting or shooting pain in the course of the nerve, returning at intervals—phenomena characteristic of the disease. The paroxysm is short, lasting only a few seconds or a few minutes; but the pain is perhaps the most severe that the human frame is capable of enduring. Some patients have compared it to an electric shock of great intensity; and others to the intensity and violence of a fulminating powder. Dr. Pemberton (a noted physician of times long gone by) is said to have stamped out the bottom of his carriage during the paroxysms of facial neuralgia; and Valleix mentions a physician who was induced, by excessive agony, to make deep incisions into his face, and then to apply the actual cautery to the wound; but his pain not being mitigated by these methods, he several times attempted suicide. But the disease does not tend directly or of itself to shorten life. Even in mild cases the patient often, on the instant of attack, becomes fixed like a statue, fearing to move a muscle or a limb, lest he should aggravate the pain, or reproduce the seizure. This is a condition common to many nervous affections attended by excruciating pain, such as *angina pectoris.* In cases of ordinary intensity the effect is so completely limited to the nerve that the skin is not discoloured, while the organs immediately in connection with it are little affected—the eye, perhaps, being only watery, the nose hot, and the teeth aching. In severer cases, however, and where the disease affects the nerve generally, or the whole face and scalp, the condition of the patient is most lamentable. The mouth is spasmodically drawn, as in palsy, so that the saliva flows over the chin and neck, or, in cases where the second branch of the *trigeminus* is affected, the teeth chatter by the clonic spasms which sometimes attend the disease, and the pain is usually most severe in the parts supplied by the infra-orbital, namely—the lower eyelid, alveoli, upper lips, and teeth of the upper jaw. The saliva is increased in quantity and altered in quality. The patient is afraid to brush his teeth, lest the paroxysm should return, and the whole of the teeth of the lower jaw become so encrusted with tartar, as to form one solid mass, indicating at the same time a depraved state of the digestive organs, as well as of the saliva. The eye and eyelid are likewise frequently convulsed, the conjunctiva injected, from which tears freely flow, especially on remission of the paroxysms, and the nose discharges a muciform secretion. The twigs of the lachrymal branch of the *fifth nerve* going to the lachrymal gland and conjunctiva, account for this increased secretion of tears and redness of conjunctiva. To touch even the hair of the head produces pain, and sometimes the affected nerve may be traced by a red line marking its course. The recurrence of the paroxysm is uncertain; in slight cases it may return only once in a few weeks, or in a few days; but in some severe cases it will return every quarter of an hour, every five minutes, or every minute, and even every few seconds, night and day. In a few cases the paroxysms occur periodically, and at stated intervals. In general, however, the times of recurrence are uncertain: sometimes the patient is attacked with great violence many times a day for many days or weeks together, so that the disease is almost continuous; at other times it intermits for a week, a month, six months, or a year. Neuralgia affects nearly as often the right as the left side of the face, and sometimes both sides; and may be set up by any movement of the part, by pressure, by sudden shock, or by a draught of cold air.

Neuralgia is rare in the course of the third branch of the *fifth pair* of nerves, especially of the *auriculo-temporal* and *lingual branches.* When it does occur it generally affects the *inferior alveolar*, especially the *mental*, after it escapes from the *mental foramen*, and pain is experienced along the gums and teeth of the lower jaw, the chin, and lower lip, accompanied sometimes with salivation.

When all the branches generally of the fifth cranial nerve are affected, the most painful points are at the exit of the ophthalmic, of the superior and of the inferior maxillary branches; next to these the frontal, and next the parietal, and lastly the occipital, although its origin is independent of that of the *trigeminal;* and whether the *trigeminal* was affected by itself, or the *occipital* nerve as well, Trousseau has always observed that "pressure made on the spinous processes of the first two cervical vertebræ always caused pain, and in a certain proportion of cases immediately brought on shooting pain in the diseased nerves."

(*b.*) *Sciatica is a neuralgic affection of the sensory nerves of the sciatic plexus, composed of the fourth and fifth lumbar and first and second sacral nerves.*

Of this Dr. Anstie describes three varieties—(*a.*) That which is associated with a strongly-marked nervous temperament, and is usually excited by bodily fatigue or mental distress; (*b.*) that which occurs to persons who have been subjected to excessive muscular exertion, or who have been much exposed to damp cold—a variety which is generally characterised by obstinacy and intractability; (*c.*) the third variety is that connected with the cachexia of syphilis, or of rheumatism or gout (hip-gout), and generally caused by inflammation of the tissue round the nerve.

It is so often associated with *rheumatism*, that rheumatism is really the most frequent cause of sciatica; and with *gout*, so as to constitute a special form of these diseases—namely, *rheumatism of the hip* (*hip-rheumatism*) and *gout of the hip* (*hip-gout*). Hence, catching cold is a frequent cause of *sciatica.* The pain of sciatica may also result from caries of the vertebræ, or tumor or vascular hyperæmia, with varicose dilatations of vessels in the intervertebral foramina, through which the nerves pass. The presence of enlarged glands in the pelvis, or of pelvic tumors, especially ovarian cysts, and fæcal masses in the sigmoid flexure of the colon, may also be the source of *sciatic neuralgia.* The disease is most frequent between twenty and sixty years of age, and is more common in males, and those most exposed to changes of temperature, than in females and those who are living in ease and comfort.

The most frequent seats of pain in sciatica are along the *posterior cutaneous nerve* of the thigh, where the posterior and outer part of the skin of the leg becomes painful; the *superficial* branch of the fibular nerve, where the pain is over the outer and anterior surface of the leg, and the dorsum of the foot; the *communicans tibialis,* where the pain is in the outer side of the ankle and the foot.

The most frequent points of pain are behind the trochanters, some parts of the thigh, about the knee-joint, just below the head of the fibula, just above the outer ankle, the ankle-joint generally, and dorsum of the foot (VALLEIX).

The disease rarely begins suddenly, but develops itself gradually, and slowly becomes severe. The pain is constantly increasing and deeply seated, especially near the ischiatic tuberosity. Tension of the fascia usually makes the pain worse, and the leg is generally flexed, whether in bed or out of it; and, from not using the limb, it may sensibly diminish in bulk in chronic cases.

Usually *sciatica* is an obstinate disease, lasting for months, or even years, and liable to relapse.

(*c.*) *Intercostal neuralgia depends on the morbid excitement of one or several spinal nerves* (*sixth to the ninth*), *especially those which pass along the upper intercostal spaces towards the sternum, and along the lower spaces to the epigastrium.*

It is very common, and is met with more frequently in women than in men, especially in the sixth, seventh, and eighth intercostal nerves, and more commonly on the left than the right side. It is not unusual after recovery from pleurisy, and may also accompany pulmonary tubercles. It is usually also associated with hysteria and anæmia, especially in women weakened by *hemorrhagia* or *leucorrhœa.*

The following points are especially noted as commonly most painful, namely—

The vertebral point in the posterior part of the intercostal space somewhat outward from the spinous process, and on the level of the point of exit of the nerve from the intervertebral foramen.

The lateral point lying in the middle of the intercostal space, corresponding *to the point of division of the intercostal nerve, and from which the nerves pass to the skin.*

A third point is near the sternum, between the costal cartilages in the upper intercostal nerves; in the lower ones it is in the epigastric region, somewhat outwards from the middle line (VALLEIX).

These points are generally very sensitive to pressure (hyperæsthesia). *Hard* pressure sometimes relieves the pain. An attack of intercostal neuralgia sometimes just precedes an eruption of *herpes zoster, zoua* or *shingles;* and in the intervals of the sharper pangs of pain, numbness, coolness, and formication are occasionally felt (WALSHE).

(*d.*) *Crural neuralgia occurs where the sensory filaments of the lumbar plexus going to the thigh and leg are affected.*

The pain is felt along the *anterior* and *inner* surface of the thigh, leg, ankle, and dorsum of the foot, and in the great and second toes. It thus differs from *sciatica*, which is generally along the *outer* and *posterior* surface.

(*e.*) *Lumbo-abdominal neuralgia affects the cutaneous nerves of the lumbar plexus, going to the lower part of the back, the nates, the anterior abdominal wall, and the genitals.* The seats of pain are commonly,—to the outside of the first lumbar vertebra, just above the middle of the crest of the ilium; to the inner side of the anterior superior spine, and the termination of the nerves in the *mons veneris, vulva*, or *scrotum*, towards the *nates* and the genitals generally.

(*f.*) *Cervico-occipital neuralgia arises from excitement of the sensory nerves, originating from the first four cervical nerves, and affects the occiput, neck, and nape of neck.*

(*g.*) *Cervico-brachial neuralgia is located among the sensory twigs of the brachial plexus, composed of the lower four cervical and the first dorsal nerves.*

When the nerves of the brachial plexus are affected, pressure made over the spinous processes of the last cervical vertebræ give pain; so also in cases of intercostal, lumbar, or sciatic neuralgia, similar pressure over corresponding regions would also produce pain. Trousseau, therefore, makes the general statement, that "in neuralgia the spinous processes of the vertebræ are tender on pressure at a spot nearly corresponding to the point of exit of the nerve from the intervertebral foramen, and that the pain pretty frequently extends a little farther up along the vertebral column." Thus neuralgia reveals itself by acute pain when pressure is made over the spinous processes which correspond to the origin or point of exit of the implicated nerves.

(*h.*) *Mastodynia, or "irritable breast"* (of COOPER), *is a neuralgia of the intercostal nerves, or anterior supra-clavicular nerves, going to the mammary gland.*

It affects women about the period of puberty, up to the thirtieth year of life. The gland is sensitive to the slightest touch at one or more points; and severe pain occasionally shoots out towards the shoulder, axilla, or hips, and is worse shortly before the menstrual period. At the height of the pain vomiting may occur. The disease causes great anxiety besides the pain; for the patients generally believe they are to have a "cancer of the breast," especially if *neuromata* form in the gland. It may last for months or years without any perceptible change or improvement.

It thus appears that in all these varieties of neuralgia there are certain characteristic symptoms, and especially—

(*a.*) *Cutaneous hyperæsthesia* at the points of exit of the nerve-trunks; most marked in cases of *intercostal, lumbar,* and *crural neuralgia.* In such cases a slight scratch of the skin, or gently rubbing the skin with a blunt end of a pencil, will cause pain of a burning or pricking kind; and in many cases the track of the implicated nerve may be followed by the tip of the finger as far as its cutaneous distribution. More rarely the reverse of this condition supervenes—namely, *anæsthesia.* It is most apt to attend neuralgia apparently of a rheumatic origin, or which is due to a slight lesion of the cord. In such cases, when the *hyperæsthesia* has lasted a long time, it is followed by *anæsthesia,* and sometimes by paralysis. In *herpes zoster* this is apt to occur; also in *sciatic neuralgia.*

(*b.*) Certain *superficial tender spots* are also characteristic of neuralgia. It is generally at the point of exit of nerves from an osseous foramen that pain is most particularly felt. Thus, in neuralgia of the fifth pair, it is over the *supraorbital notch,* where the ophthalmic branch becomes superficial; over the *infraorbital foramen,* which gives passage to the superior maxillary branch; and over the *mental foramen,* through which emerges the inferior maxillary division of the fifth pair. But when *supraorbital neuralgia* is intense, the point over the nasal branch is also extremely tender on pressure over the point of exit of this small nerve; and pressure over the frontal eminence also causes acute pain; and another tender point is over the *zygomatic process* in front of the ear (TROUSSEAU).

The particular form of cachexia has also a remarkable influence on the seat of neuralgia. In *chlorosis, neuralgia* is apt to affect several regions, but notably the *trigeminal nerves* and nerves of the *solar plexus.* In cases of *anæmia* from *uterine hæmorrhages* or *leucorrhœa,* the neuralgia is mainly *gastric* and *intestinal.* In *malaria,* the *supraorbital branch of the trigeminus* is mostly implicated. In *rheumatic cachexia* it is generally the *occipital* and *sciatic nerves;* and neuralgias of rheumatic origin are generally multiple in their manifestation, and frequently alternate with articular pains (TROUSSEAU).

The exceptional cases, where pain is relieved by pressure, show that the conditions causing the pain have their site to the central side of the place pressed upon. Such cases may be relieved by division or removal of a portion of the nerve-trunk at the part pressed upon.

Prognosis.—The duration of neuralgias is uncertain; they often become chronic. In some cases neuralgia terminates after a few paroxysms; in others it lasts from one to six months; and in some cases it embitters existence during the whole period of a long life. It seldom disappears suddenly, but oscillates with a decreasing intensity, the intervals between the paroxysms gradually becoming lengthened, till at last the disease subsides. Complete cure is by no means frequent, except where the cause is *rheumatic* or *malarial.*

Causes.—The remote causes of neuralgia have been already indicated. Extremes of heat or cold, or sudden changes from the one to the other, often stand in the relation of cause to this disease. It is a frequent result of impaired general health, from malaria, syphilis, gout or rheumatism. Thus women, after profuse menorrhagia, or after childbirth, or persons recovering from fever or other severe diseases, are apt to suffer from neuralgic affections. Persons who have attempted to poison themselves with arsenic suffer agonising pains along the course of the nerves of the limbs during convalescence. Blows, or wounds, or the pressure of aneurismal or other tumors, sometimes seated in the nerve itself; irritation from foreign bodies, such as bullets or gunshot material, which have become encapsulated near a nerve; the compressing of contracting cicatrices; pressure on nerve trunks by *aneurisms, exostosis, cancers, neuromata;* varicosities and over-distension of veins surrounding nerves as they pass through long passages like the intervertebral foramina, are also causes of neuralgia. This last I have verified by dissection in a case of severe coccygeal neuralgia, in which the veins surrounding the nerves passing through the intervertebral spaces were extremely varicose and irregularly dilated. It is also common with intercostal and trigeminal nerve neuralgia (NIEMEYER), and rheumatic neuralgia is probably due to such hyperæmia and œdema of the *neurilemma.* A peculiar *neuralgia* often precedes attacks of *herpes*, as of the *intercostal nerves* in cases of *herpes zoster* or shingles; of the nerves over the gluteal region and femoral region, in cases of *herpes preputialis;* and of the side of the face, in cases of *herpes labialis,* or of the skin on other parts of the face or forehead.

Neuralgia is more common in men than in women, and in women it occurs rather more frequently before thirty than afterwards, especially in those whose menstruation is irregular either as to time or quantity. Among the poor and laborious classes neuralgic complaints are numerous; and, roughly speaking, they mainly present three varieties:—(1.) *Neuralgia of the head and face;* (2.) *Neuralgia intercostalis;* (3.) *Sciatica.* The great majority of the patients are anæmic, and in a fair sprinkling of cases the anæmia is obviously connected with malaria (ANSTIE, *op. cit.*, p. 86).

Diagnosis.—The disease to which *neuralgia* bears most resemblance is *rheumatism;* but it is distinguished from it by the paroxysmal nature of the attack, and by the absence of all swelling. There are certain points of the face which, upon being pressed in the interval of the paroxysm, give pain, and so far furnish phenomena which may aid in a differential diagnosis. Other diagnostic features are of importance to be noticed, such as, if pain exists—(1.) Where the nerve emerges from the bone, as at the supra-orbitary, infra-orbitary, and mental foramina, in trifacial neuralgia; (2.) where the nerve passing through the muscle reaches the skin; (3.) where the nerve terminates in the skin; (4.) where the nerve becomes very superficial, as the peroneal nerves; and lastly, (5.) where the pain affects the spinous processes corresponding to the exit of the implicated nerves. The pain of inflammation must also be distinguished from the pains of neuralgia, which are usually of a shooting paroxysmal character, flashing with momentary intensity along the fibres of the affected nerve, in paroxysms of momentary shocks following each other in rapid succession. The pain of inflammation is constant and increasing up to a maximum.

Treatment.—When the cause of the neuralgia can be discovered, its removal is the obvious procedure to follow. There can be no question that the disease has often subsided under the use of various remedies; but the tendency in neuralgia to a spontaneous intermission is so great, that it is doubtful whether in any case medicine can be said to have cured it. Opiates are unquestionably serviceable in mitigating the sufferings of the patient, and perhaps in influencing the disease, but not to the extent generally supposed. *Belladonna*, both internally and as a plaster, may sometimes relieve the pain. *Stramonium* and *opium* have a similar temporary effect. *Chlorodyne* is also to be recommended; and the tinctures of *gelseminum* and of *guarana.*

The remedies of most value are the diffusable stimulants, such as *sal-volatile, hot tea, quinine, chloral,* and *croton-chloral,* in full doses, and *alcohol* in small doses, *blistering,* the application of *ammoniacal fluid* to the skin immediately over the painful nerves, and the endermic application of *morphia.* These remedies act by increasing the supply of blood to the painful nerve, and, *pro tanto,* heightening its vital energy (ANSTIE, *op. cit.*, p. 84). The most speedy way of obtaining a temporary relief is certainly the application of a local stimulant, and more especially of some volatile agent, such as *mustard,* or, still better, *chloroform* diluted with *seven* parts of some simple liniment, such as *belladonna* (ANSTIE). A rapidly acting blistering fluid is still more effective. But the more profoundly the general health has been affected, and especially the greater the degree of anæmia, the more necessary is it to join with the use of stimulants (both local, such as above mentioned, and general, such as the *carbonate* and *muriate* of *ammonia,* taken in *five* and *ten* grain doses respectively), a treatment directed to improving the condition of the blood by "food tonics," such as *cod-liver oil, arsenic,* or *steel,* or a combination of some of them, joined with the use of local stimulation by means of frictions with *dilute chloroform,* and the manipulations of the scientific "*shampooer*" (ANSTIE, *op. cit.*, p. 87). Dr. Radcliffe reports favourably of the *hypophosphite of soda,* and believes that the *phosphorus* of this salt acts directly as a food to the nervous tissue. The subcutaneous injection of small doses ($\frac{1}{6}$ to $\frac{1}{4}$ of a grain) of *morphia* in solution will give temporary relief, and is especially useful to those patients who are obliged to go through an ordinary day of labour (ANSTIE, *op. cit.*, p. 87).

When these or other general remedies have proved insufficient, recourse has been had to local remedies. The most efficient of these applications is the alkaloid *aconitina,* rubbed upon the pained part in the form of an ointment, in the proportion of one or two grains to one drachm of lard. A more convenient and efficient application is the "*anodyne amyl colloid,*" prepared by Messrs. Ferris & Co., of Bristol, from *hydride of amyl, aconitina, veratria,* and *ethereal collodion. Morphia* similarly used, and blisters, have often exercised a beneficial effect upon the disease. Holding the head over steam, and the warm bath, are sometimes beneficial in cases of facial neuralgia. The belladonna plaster is also a favourite application. When the neuralgia is superficial, compresses steeped in a solution of *atropia* have a good effect. For this remedy Trousseau recommends the following formula:—

℞. *Atropiæ sulphatis,* gr. v.; *Aquæ distillatæ,* ℥iii.; *solve.* Renew the compresses several times in the twenty-four hours. Continue them for at least an hour each time, and cover them with oilskin, to prevent evaporation.

When general and local applications are unsuccessful, the cause may be sought for in a diseased tooth or stump, and in a *very few* instances an exostosis of the stump has been discovered, removed, and the disease cured. Sometimes, however, even when the patient submits to have every tooth in his head drawn, no relief or benefit may result. The division of a nerve is sometimes had recourse to; but even this operation is very uncertain. Where the causes of neuralgia cannot be removed, modes of treatment which greatly modify the change of tissue and the nutrition of parts are often beneficial. Chief among these is the use of electricity; and Niemeyer gives the results of his experience in the following propositions:—

"1. In treating neuralgia with the induced current, it is best to employ the metallic electrodes known as the electric brush. While one electrode, containing a moistened sponge, is held in one of the patient's hands, or against any part of his body, we stroke the brush along the course of the affected nerve; if there are any specially painful points, we allow the brush to remain over them rather longer (*electric moxæ*).

"2. Many cases of neuralgia, which had been previously treated without benefit by the most varied remedies, were completely and permanently cured in from twelve to twenty applications, or even sooner. In other cases no benefit or cure was effected.

"3. The first sitting shows whether the neuralgia can be cured by the induced current. We can only expect a cure where the pain is decidedly relieved, or entirely disappears, immediately after the first electrisation, even if it should only be for a short time. If this temporary result do not take place, the continuation of the treatment will also prove ineffectual.

"The application of the induced current as above directed is very painful. The current employed ought not to be of greater intensity than the patient can easily bear; the applications should also be of short duration and frequently repeated. It is only after the patient has actually experienced benefit that he suffers it with patience."

The constant current is far more effective than the Faradic in neuralgia. Some cases that have been treated without result by the Faradic current have been cured by the constant, but never by the reverse. Niemeyer places both poles along the affected nerve, and, without attending to the course of the current, holds the zinc pole on the most painful part and on those parts where the nerve approaches nearest to the surface, as at the *supra-* or *infra-orbital foramen*, or at the *zygomatico-facial foramen*, or at the *sciatic notch*. If it be possible to get the nerve between the poles, as in the cheek or nose, he introduces one pole into the mouth or nose to the point whence the pains radiate, while the other is placed at the corresponding point on the skin. "At first the application of the constant current is not particularly painful; but an unpleasant, burning, piercing pain soon commences, and gradually increases; and where the number of electric elements is large, it may become unbearable.

"The changes induced in the skin at the point of application of the constant current, if the electrodes be applied for a length of time, are far greater than those caused by the induced current. They not only consist in a lively erythema, a decided swelling of the skin, and an increase of the subjacent tissue; but papules and blebs arise on the skin, particularly at the positive pole. If the action continues long, the surface of the elevations sloughs off. These changes take place not only at the point of application, but occur equally, or at least similarly, in the deeper parts, as is shown not only by the increase in volume of the subcutaneous tissue and muscles, but also by the following experiments (ERB), which show a great deal about the mode of action of the constant current in neuralgia and other neuroses, as well as in some diseases of the muscles and joints: If we cross the forearms, placing the volar surfaces in contact, and apply the electrodes to their dorsal surfaces, there will be reddening not only of the parts to which the electrodes are applied, but of the corresponding points on the anterior surfaces. Generally, relief immediately follows the application of the constant current, just as it does that of the induced current; but occasionally the pain is at first increased, and that should not always induce us to stop the treatment. There seems to me no doubt that the curative action of the constant current, in most cases of neuralgia, is to be explained by the modification of the circulation, endosmosis, or change of tissue in the diseased nerve, its neurilemma, or the parts around. This 'catalytic action' may result from the chemical disintegration induced through the nerves, or the attraction of the constituents of the nutrient fluids toward the pole, or it may occur in some other way" (NIEMEYER).

Narcotics, in the form of hypodermic injections of solutions of morphia, are of great value, and it is immaterial whether the injection is made near the seat of pain, or at a remote distance from it. "Subcutaneous injections of *morphia* constitute the only decided and effectual, and truly invaluable, method of treating neuralgia" (VON PITHA). The hypodermic syringe and the morphia solution are now almost as indispensable accompaniments of the physician as the stethoscope and thermometer. Its use, however, is attended with similar bad results as the taking of opium in any other form. The dose must be gradually increased, and the remedy cannot easily be given up. If it is omitted the patient feels dull, weak, uncomfortable, nervous, and trembles from head to foot, as after an alcoholic debauch. As a palliative it is invaluable, to the extent of *one-sixth* to *one-third* of a grain of *morphine,* injected hypodermically. But in slight cases, and in sensitive persons, and in the young, very much smaller doses may relieve the pain—*e.g.,* the twentieth to the twelfth of a grain. In severe and persistent cases the dose has to be increased; and it is very desirable not to have recourse to such hypodermic injections oftener than twice daily. It is necessary also to employ the remedy with proper caution, with reference to the patient's individuality or idiosyncrasy. The hypodermic treatment with morphia must be commenced tentatively in small doses. The first dose of a morphia injection for an unknown individual must not be more than one-twelfth of a grain; and in very sensitive and delicate women and young people not more than one-twentieth, or even yet less. If the patient is the subject of that peculiar idiosyncrasy of extreme sensitiveness to opium, symptoms of poisoning may supervene. Thus, there may be syncope, spasm of the heart, or an asthmatic paroxysm, the skin being covered with a cold sweat. Fresh air should at once be admitted through opened windows, while the patient's face is diligently fanned. An assistant should also bathe the face and temples, as well as the bared chest, with vinegar, and these parts are to be sprinkled at the same time with cold water. A strong decoction of coffee is to be administered as an enema, or, when possible, given by the mouth; and, in extreme cases, artificial respiration must be resorted to. In most cases these means will succeed, providing that the dose has not been too large. When the vomiting and disposition to syncope persist without yielding to the usual measures, a careful and interrupted inhalation of chloroform may be useful. If there is a constant disposition to nausea and sickness after the hypodermic use of morphia, it may be counteracted by adding from a quarter to a third of the tincture of belladonna to the morphia solution, then the extract, and, finally, minute quantities (about one-hundredth of a grain) of the *sulphate of atropia.* The extract answers best (VON PITHA).

The injection should be projected into the subcutaneous tissue, and not into the skin itself; and this may be secured by pinching up a fold of skin, care being taken not to make a double perforation in it. Well known or visible vessels or nerves are to be avoided. The places most eligible for the injections are—the external parts of the arm and thigh, the back, and the integument of the abdomen and nates. The face and neck should be avoided; and the painful regions ought not to be punctured.

"With respect to the preparation to be employed, it may be stated that the *acetate* and *hydrochlorate* may be used indifferently. Only in cases in which very large doses are given, as above a grain, need the *acetate* be preferred, on account of its greater solubility. It will form a clear solution in six parts of water, whereas the *hydrochlorate* requires twenty parts. Hot water will dissolve it in ten parts, but, on cooling, half of the alkaloid is precipitated; so that if a concentrated solution is wanted, it has to be prepared only just before using it. On the other hand, the *acetate* requires great care in keeping it in a cool place and shielded from the light. The solution should always consist of *morphia* and *distilled water*, without any addition whatever. The favourite addition of glycerine has the disadvantage of causing white flocculi to be precipitated from the originally clear liquid. Belladonna, however, may be added, for the reasons already stated, with great advantage; and although the solution is rendered somewhat turbid, its efficacy is not diminished. It is very desirable that similar combinations with different other substances, and especially other narcotic alkaloids, should be thoroughly investigated" (VON PITHA).

Frictions of the skin with *veratrine ointment,* in the proportion of gr. iv. to gr. x. of *veratrine* to ℥i. of *lard;* or with *aconite ointment,* in the proportion of *one grain of aconite* to one drachm of *lard,* have also been used with advantage. The application of cold water sometimes, but only exceptionally, does good, especially if the pain is attended by heat—applied as a compress, or as a fine douche. Warmth by means of a hot iron, a stream of warm water, or as a warm cataplasm, may also afford relief. Richardson's spray producer has also been of use; and also acupuncture as recommended by Teale, especially in the neuralgia of joints persisting after rheumatism or spasm. As internal remedies *chloral* and *bromide of potassium* combined are of great service.

The treatment of sciatica consists in following out, as far as possible, the removal of the causes and circumstances already noticed as producing the disease. In the rheumatic form, the warm baths are most useful, especially the systematic treatment at Neuenahr, Wildbad, Wiesbaden, Teplitz, and Bath. *Iodide of potassium,* in large doses, and *sulphur,* especially in the form of the *Chelsea pensioner,* appear to do most good. Of other specific remedies Niemeyer mentions the induced, and still more, the constant current of electricity as rarely failing to do good. *Oil of turpentine,* as an electuary, is also recommended in the following—

℞. *Ol. Terebinth.* ʒi.; *Mell.* ℥i., of which a tablespoonful is to be taken twice a day. This is very highly spoken of by Romberg.

With regard to the treatment of mastodynia or irritable beast,—a plaster worn on the breast, if composed of anodyne elements, may soothe the pain and prevent the gland being touched by the patient, and it as well may give support to the breast. Cooper recommends the *soap plaster* with *extract of belladonna,* and the following pills :—

℞ *Ext. Conii, Ext. Papaver, a a* gr. ii.; *Ext. Stramonii,* gr. $\frac{1}{4}$ to $\frac{1}{2}$, are mentioned by Romberg as useful.

CEPHALALGIA.

Headache, cephalalgia, or *pain in the head,* with or without giddiness, although frequently attending diseases of the nervous system, are yet very uncertain signs of lesion in the brain or head, because they are associated with so many disorders, such as gastric derangement, constipation, and dyspepsia. But the conditions attend active cerebral congestion, cerebral meningitis, encephalitis, the specific febrile diseases, syphilitic periostitis, and intracranial tumors. Headache is therefore rather a symptom than a disease. Some headaches are limited to one-half the forehead or to one eye, the region of pain corresponding to the area of distribution of the first branch of the fifth cranial nerve; some occupy both sides of the forehead, some affect the vertex, some the occipital region; some mainly occupy the temples, the pain changing or shooting to and fro from one side to the other; some are confined to the neighbourhood of the ear, or in the form of an earache. In some cases the pain seems to be generally diffused all over the head. It varies in character, and is variously described as aching, shooting, throbbing, or like a weight upon the top of the head, or a constriction round it. Intolerance of light and sound, visual spectra, tinnitus aurium, vertigo, nausea, sickness, sleepiness or wakefulness, delirium and incoherency of talk, wholly independent of brain disease, are phenomena which are frequently associated with headaches. The causes of headaches are not always easy to discern; but pressure on the frontal branches of the fifth pair, rheumatic neuralgia of the forehead from exposure to cold air, often inducing a sense of drowsiness, as in railway travelling with the face to the engine, may induce a headache. Frontal headache is more generally referable to abdominal affections; vertical headache to cerebral disturbance; occipital pains to disorders of the circulation, and more especially to anæmia (Dr. Hughlings Jackson). But pain due to cerebral disease, especially in the case of cerebral tumors, may be referred to all parts of the head; and this pain may exactly simulate those which are of less serious origin. It may be slight or intense, continuous or paroxysmal; and the most intense pain (usually very limited as to its seat) is that which is induced by pressure of intracranial tumors or abscesses upon sensory nerves (Dr. BRISTOWE). The sensitiveness of the dura mater is now a well-established fact, and there are now also many considerations which tend to show that the pia mater, though apparently insensitive, may become the seat of pain in conditions of disease. That those diseases are associated with intense headache in which there is abnormal increase of the intracranial pressure, and thereby undue tension of the cerebral membranes, is amply justified by clinical facts. Acute pain may accompany brain disease of an inflammatory nature, in which the inflammatory action does not implicate the dura mater, and before the tension of the membranes can have been appreciably increased by the products of inflammation. In such cases the pia mater must be regarded as the seat of pain. In cases of cerebellar disease, or disease in the posterior fossa of the skull, the situation of pain in the head most frequently corresponds to the position of the lesion; but this is less constantly the case in affections of the cerebral hemispheres. Dr. Ferrier and Dr. Alexander Robertson have each called attention to the value of percussion of the skull in cerebral disease, even when the patient makes no spontaneous complaint of pain in the head, as an aid in fixing the locality of cerebral lesions, more especially those of the cortex (*Glasgow Med. Journ.*, l. c., and *Brain*, Jan., 1879). A real nervous headache is one that starts *ab initio* in the brain: the sensory tract of the encephalon becomes irritated and over-excited, and pain results.

The doctrines regarding the nature of headache have been very fully elucidated by several recent writers, especially by Drs. Sieveking, Edward Liveing, P. W. Latham, Day, Symonds, Anstie, and others. It is of the greatest importance, in the first instance, to ascertain the immediate exciting cause of the headache, especially with reference to—(1.) An increased afflux of blood; (2.) a diminished amount of blood; or, (3.) with reference to the existence of a specific or constitutional disease. Any of these states may explain its existence, and must determine the course of treatment to be adopted for its relief. To determine, in doubtful cases, whether headache is associated with repletion or emptiness of the intracranial vessels, dry-cupping is found to yield valuable assistance. Applied to the nape of the neck, it will, in the one case, afford more or less immediate relief; whereas, if emptiness of the intracranial vessels is the immediate cause of the pain, the effect of the operation will increase the pain, will tend to produce prostration, and may even induce syncope. In the former instance, the headache will be benefited by diminishing the contents of the vessels; in the latter case, it will be benefited by increasing the amount of blood in them. The ratio in which cephalalgia occurs as a symptom in organic affections of the intracranial contents, determined by *post-mortem* inspection, shows that it is a symptom of less frequent occurrence than might have been anticipated. Dr. Abercombie's and Andral's cases yielded to the analysis made of them by Dr. Sieveking the result "that it seemed an even chance whether the intracranial disease was or was not accompanied by headache."

The causes of cephalalgia may be arranged into three classes:—(1.) Those directly affecting the head; (2.) those associated with the chylopoetic viscera and the functions of digestion and nutrition; (3.) those taking their origin in connection with the sexual functions. Among the causes of cephalalgia connected with the state of the cranial contents, the principal are mental and intellectual excitement carried beyond the limits of healthy stimulation, in addition to those already mentioned.

In private practice a form of headache is frequently met with which is variously called:—

"*Nervous headache*," "*Sick headache*" or "*Bilious headache*," "*Brow-ague*," "*Megrim*," "*Migraine*," "*Hemicrania*." It affects both males and females, and especially males of studious and sedentary habits, up to a certain age. It is a noteworthy variety of trigeminal neuralgia—a combination of neuralgic symptoms with headache occurring in paroxysms, sometimes limited to one side of the head and brow. The sick headache of *migraine* is not to be confounded with the sickness and headache which attend a bilious attack, consequent on indigestion after eating too freely of a good dinner. Such a headache, after a debauch, or after too much wine or alcoholics generally, is common enough; also the headache in particular idiosyncrasies from eating certain articles of food, and which probably has a gouty origin (WILKS). The true sick headache of *migraine* is a purely nervous affection, and occurs generally in the most temperate livers, and is thus often totally misapprehended by those who only think of headache and sickness as symptoms of stomach disorder. It appears in a large proportion of cases to be a hereditary affection. It then not uncommonly commences during the period of the second dentition, from which period of life and up to thirty years of age it usually first declares itself. It rarely commences after thirty; it tends to subside with advance of years, and is not common after fifty. This form of headache is preceded, for a variable period, by certain disorders of sensation, the most striking of which is a transient disturbance of vision. In some persons the malady stops short here, and is not followed by headache; in others, the headache appears to be developed without any premonitory symptoms, until careful inquiry reveals the contrary. The complaint, then, has two stages:—1. The stage of disordered sensation; 2. the stage of headache, nausea, chilliness, and absolute misery. There are thus different ways in which the stage of disordered sensations commence—viz., either by a glimmering near the outside corner of the field of vision, or by a glimmering near the middle of the field of view, or by objects at or near this point becoming obliterated. In some persons the phenomena are accompanied with colour; in others this is not the case, the outline only being somewhat luminous. In others, again, in slight attacks there is no colour, but in the more marked ones colours are seen not only at the outline, but generally vibrating over the field of vision; and in still more decided attacks numerous stars are seen floating in all directions. Another very important point about these attacks is, that in perhaps from one-fourth to one-third of the cases during this glimmering stage, there is tingling in some portion of the body—the part in that state is commonly described as being *asleep*.

In a young female seen by Dr. P. W. Latham, the tingling affected one arm and the side of the tongue; and, curiously enough, both her sister and her father were affected in precisely the same way. The tingling was on the *same side* as that on which the glimmering in the eye began. In another case, the patient complained of a feeling of pricking and scratching on that side of the face corresponding with the glimmering. In others, speech or hearing may be affected in such a way that the usual command of speech is lost, aphasia of the most typical description existing for a time; and memory fails so much that what is said is not remembered, and talking may become incoherent. In slight attacks one eye only seems affected. Drowsiness is very common as an early indication of the attack. In some cases it comes on during its progress, culminating in a more or less prolonged semi-comatose condition; but much more frequently it is the natural ending of the attack. Double vision sometimes occurs. In some cases certain portions of the retina become insensible; and, if the central spots of the eye be involved, the patient (in other respects feeling well) notices that he cannot see the ink of the pen with which he is writing, or the letters he is forming; or, that while distinguishing all other parts of the body, he cannot see the face of the person he is looking at. Sometimes he observes a tremulous, vibratory, or rotatory movement in some part of the field of vision. This disturbance of vision is often associated with a feeling of chilliness, coldness of hands and feet; it may last from five to thirty minutes or longer, and then be succeeded by the stage of headache, which shows itself as follows:—"When the vibratory movement is at its height, a little aching is felt in the head, on the side *opposite* to that on which the glimmering first appeared: it is slight at first, but gradually increases in intensity. Some persons have said that the sensation was as though a point in the temple were being bored with a gimlet, and the gimlet slowly increasing in size. The pain gradually spreads from this point, which may be covered with the finger, and pressure upon which affords relief, first over one side of the head, and then, but not always, extends to the other. As the headache increases, the ocular disturbance declines, nausea is felt; this increases with the headache; there are retching and vomiting, the latter sometimes, though rarely, giving relief; the head throbs; the slightest movement increases the pain; and any attempt to move from the recumbent posture increases the gastric uneasiness; the mouth feels clammy; the eyeballs ache, and are tender on pressure, one more so than the other; the pupils are rather contracted, and generally unequally so; the patient lies apparently more dead than alive; his face pale, and the head hot. After a varying number of hours he is somewhat relieved by troubled sleep; he wakes up next morning free perhaps from headache, but he is listless, his brain is weary, and he feels as if he had undergone a hard mental struggle. There may be now an interval of a few days, weeks, or years, before the disorder again shows itself.

"This kind of headache varies much in character, degree, and duration. In some persons the pain is not localised in any particular spot, but seems generally diffused over the head; others have not noticed that there is more pain on one side of the head than on the other, or that the aching radiates from one painful spot, until their attention has been directed to the fact, and then they distinctly recognise it; others, again, have neither vomiting nor nausea; and lastly, the duration of the headache may be very short, or may not extend over more than two or three hours, or it may be entirely absent. The disorder may even stop short at the vibratory stage, the vision be restored, and no further inconvenience felt. On the other hand, the headache may be, and in many individuals always is, developed without the ocular disturbance; but other sensations are substituted for it. The patient has a feeling of chilliness; the feet are cold, and there is mental depression; a dread of impending evil; he is restless and uneasy; 'cannot quite tell,' as he says, 'what he would be at;' he has what is expressively called the 'fidgets.' This condition may continue half an hour or more, and then the slight boring, piercing pain is felt in the head with which the aching begins, and the disorder runs its course, as in the forms previously mentioned. In other cases, this feeling of depression or uneasiness lasts for several hours, the patient goes to bed, and in the early morning wakes with the headache fully developed" (P. W. LATHAM, *Brit. Med. Journ.*, March 23, 1872).

The conditions under which this disorder shows itself are as follow:—The great majority of persons who suffer from this form of headache are at the time more or less anæmic; there is a general want of tone—a relaxed condition of the muscular and the arterial systems, especially of the latter; the pulse being rather small and soft, often decidedly slow, but much accelerated on slight exertion or excitement. Even in those who are not anæmic and appear robust, there is still this want of tone about the pulse. The sufferers possess what is called the nervous temperament; their brains are excitable, their senses acute, and their imaginations free. The attacks are often induced by prolonged mental work, protracted mental excitement, or any intense strain on the feelings, such as grief, anxiety, passion, and general emotional disturbance. Bodily fatigue, late hours, loss of sleep, the depression which follows over-excitement, a debauch, are all predisposing causes; and it is curious that the attack is not generally developed during the paroxysms of mental excitement, but afterwards, when the excitement has passed off, and the mental strain is somewhat lessened. The influence of malaria has also been assigned as a cause. Again, in females the attacks are more frequent at or after the catamenial period. Rapidly varied impressions produced upon the senses, such as are produced by glaring lights, rapid succession of objects presented to the eye, loud or discordant noises—all of which are to be found on the crowded platforms of railway stations on the arrival and departure of trains—also strong odours and offensive smells, are frequent cases of this headache. It is to be observed that all these causes, and causes like to them, are of a depressing nature; exhausting the powers, and, therefore, lowering the tone of the system; putting it out of tune, disturbing the harmony in the functions, and, at the same time, exalting the susceptibility of the nervous system. Dr. Latham believes that its primary cause is some affection of the vaso-motor nerves: for there is *contraction* of the vessels of the brain, and so a diminished supply of blood, produced by excited action of the sympathetic; and the exhaustion of the sympathetic following on this excitement causes *dilatation* of the vessels and the headache. To the anæmia so produced, the defects of vision and the other early phenomena are due. Dr. Liveing, on the other hand, maintains that there must still be some more antecedent cause to which the vaso-motor affection must itself be due. He regards the optic thalami, and all those parts which lie between these bodies and the roots of the vagi, as being affected. Thus the disease would have a close pathological relation with epilepsy, although it is not found that they pass the one into the other.

The attack of sick headache is often sudden; or it may come on during the night, the patient waking from time to time from a disturbed sleep with the consciousness of a weight or heaviness in the head. When it is time to get up the attack has become so fully developed that he feels incapable of exertion, and desires to be left undisturbed. Or the attack may come on immediately after rising, having awoke with a headache. Or it may come on at various periods of the day without any obvious exciting cause.

Dr. Wilks (himself a sufferer from this ailment) thus describes an attack of sick-headache:—"One rises in the morning more dead than alive, perfectly unable to swallow the smallest particle of food, and often perhaps actually sick; the head throbs, and the pain is increased by the slightest movement; speaking or doing is a burden beyond bearing; one prays to be left alone in the utmost quiet, so that he may, if possible, sleep. To other persons the sufferer looks extremely ill, very pale, dark around the eyes, and with contracted pupil. To himself his head feels hot, and the application of cold is most refreshing. The clamminess in the mouth, the nausea, and general gastric disturbance, are secondary, and have no connection with any improper meal, and thus is in no way relieved by the too frequent and ignorantly administered purgative."

It occurs in both sexes, but more often in women than in men. In women the attacks are common just before the menstrual period, or during its course. In other cases mental excitement has to do with the attack. The headache is probably due to excitement of the sensory filaments from the trigeminus to the *dura mater*, or to the *sympathetic* filaments accompanying the vessels. It increases rapidly soon after waking in the morning, with chilliness, loss of appetite, and sliminess of mouth, and at last the headache is so intense as to be almost unbearable. The eyes are extremely sensitive to light, and the ears to noise; so that the darkest rooms and the most retired are anxiously sought for. Absolute seclusion is desired. The pulse is usually abnormally slow, and there may be some nausea, and vomiting may occur, of a bitter greenish fluid.

The duration of the attack is from twelve to twenty-four hours; but it may be prolonged by successive relapses for several days. On the other hand, there are short attacks of an hour or two only. It generally subsides by slow degrees—sometimes after food is taken, and sometimes after vomiting. The patient does not generally get well till he has passed a night in bed, and has enjoyed a profound and refreshing sleep, from which he generally awakes as well as usual. Perspiration may have been profuse during this period, and a large quantity of urine may be passed.

The attacks vary from a simple to a complex character. The affection may take the form of a violent paroxysmal headache, either hemicranial or bilateral; an attack of nausea, vomiting, and prostration, very like that of sea-sickness; or a transient disorder of vision, such as half blindness. There are also cases in which all these phenomena occur in succession; also, others of a more complex kind, in which are superadded a numbness and tingling, generally having a centripetal progress towards the head and neck from the fingers, or fingers and toes of one side. There is also not unfrequently a transient form of aphasia, or difficulty of utterance; and occasionally a confusion of ideas or failure of memory of a similarly transient character.

Megrim is very frequently an unilateral affection; and in Dr. Latham's experience the aphasic cases have been exclusively those in which the other phenomena—more particularly the numbness and tingling—have been confined to the right side or bilateral.

A tolerably constant and definite order and succession amongst the leading symptoms are also maintained—the visual phenomena being usually initial, always taking precedence of the pain, while the vomiting is the last in the series.

The malady is essentially one of the nervous system, for the most part hereditary. It is above all others a "family complaint." It is strictly paroxysmal as to the nature of the seizures and freedoms of the intervals. The attacks exhibit an approach to periodicity in their returns in most instances; but with considerable variations in the length of the intervals in different cases—the same interval, however, being approximately maintained in the same individual—weekly, monthly, or bi-monthly periods being common. After a severe seizure an immunity seems to be secured to the patient for a certain period; and the aggravation of the next paroxysm, if a double interval of immunity should have elapsed. The exciting causes seem to be mainly operative only at or near the times of spontaneous recurrence. Such are various forms of peripheral irritation, and certain states of the system; more particularly gastric irritation; powerful impressions on the sensorium, as by the combined sights, sounds, and general excitement of public spectacles and assemblies; ovarian irritation and catamenial periods; overtaxing the sight on small objects, or straining after accommodation by the asthenopic or hypermetropic eyes; strong odours; and so forth; and, perhaps more certainly influential than any of them, mental emotion and excessive brain-work (LATHAM, l. c.) "Whatever produces a strong impression on the nervous system of one predisposed will cause an attack, and it may thus be induced in a hundred different ways. Consequently the sufferers from this complaint often make it the whole business of life to avoid moving a single step out of the even tenor of their way, so as to prevent, as far as possible, its occurrence. The visit to the theatre, the concert-room, or the dinner-party, is always followed by headache, for the excitement, the altered temperature or vitiated air, are all equal to its production; but even less than these is sufficient, for any strong impression on the special nerves will produce it, as a loud noise, an hour's visit to a picture-gallery, looking through the microscope, odours of various kinds, as of spring flowers, and even the tasting of some substances; also exposure of the body to the sun or a strong wind; moreover, various moral causes and worry are sure to be followed by the familiar headache" (WILKS).

All these features of the malady warrant its classification amongst those of the great natural family of Neuroses, represented by such affections as spasmodic asthma, epilepsy, angina pectoris, paroxysmal insanity, and the like. None of these have a better claim to that fellowship, or exhibit a more typically neurosal character, than megrim. The last link of confirmation is shown by the fact that attacks of megrim are sometimes replaced in the same individual by other neurosal seizures—as, for example, epilepsy or mania transitoria—although the rule is, that the same type of paroxysm is maintained throughout life. On the other hand, such transformation in the course of hereditary transmission is sufficiently common: thus, a parent suffers from typical migraine, one of his children from the same malady, another from epilepsy, and a third, perhaps, from chorea (LATHAM, *Brit. Med. Journ.*, April 6, 1872).

Having regard also to the almost exclusively sensory character of the phenomena and the natural sequence they exhibit, and that nothing but a disturbing cause situated behind the optic chiasma could explain the symmetrical affection of the visual field in each eye, so nothing but a like cause operating in the neighbourhood of their common origin along the sensory tract at the base of the encephalon could explain the successive implication of nerves so remote in their distribution as those of sight, the sentient nerves of the extremities, the trigeminus, and the pneumogastric; and bearing also in mind that the cause must be of a kind to explain both the interruption of the ordinary transmission of objective impressions and their replacement by subjective ones, as well as the strictly paroxysmal and culminating character of the symptoms, no hypothesis seemed to Dr. Latham so well adapted to explain the phenomena as that of a nerve-storm traversing more or less of the sensory tract from the optic thalami to the ganglia of the vagus, or else radiating in the same tract from a focus in the neighbourhood of the quadrigeminal bodies. The analogy of epilepsy, which is supposed to have its point of departure in a tendency to explosive discharge in the motor tract of the medulla oblongata, lends support to this view (LATHAM, l. c.)

The disturbance of vision seems mainly due, in the first instance, to defective supply of blood to one side of the brain from the contraction of the cerebral arteries, probably of the middle cerebral. And when the cause may exist in loss of tone of the cerebro-spinal system from overwork, the brain is no longer able to inhibit the action of the sympathetic, so that a slight cause serves to excite the action of that nerve. It affects more particularly the cerebral artery of the left side, causing glimmering on the right side of the field of vision.

"The relationship of the disease with epilepsy, as first suggested by Dr. Edward Liveing is happily a very distant one. The contraction of the cerebral arteries, the tingling in some portion of the body and twitchings of the muscles, which are sometimes associated with it, suggest the resemblance; whereas it differs widely from that terrible disorder in that it never threatens life, it is never associated with unconsciousness, and I have never heard of its passing into epilepsy. On the contrary, with advancing age the attacks, as a rule, become much less frequent. They cease generally after fifty or sixty, and in women not uncommonly at the change of life" (LATHAM, l. c.)

Treatment.—(1.) *Of Sick-headache.*—Looking to the true cause of sick-headache as one which lies deep in the patient's idiosyncrasy, and as an affection which is developed by a hundred different sources of excitement, the advice to sufferers is to give as much tone as they can to their nerves by adopting all those methods which experience has shown to be good, and then avoid, as far as is practicable, all those causes which are known to excite an attack. Purgation has no good result. The only remedies which are of any avail are those which act on the nervous system, such as *hot tea* and *coffee;* or, after the stomach is quieter, and the more urgent symptoms have passed off, a little *wine* or *ammonia.* If the headache take more the form of hemicrania, then other remedies are occasionally useful, as the local application of the *bisulphide of carbon,* or *galvanism,* and internally the *bromide of potassium.* This is the only drug which Dr. Wilks has really seen to be serviceable. Whilst the nausea exists and the worst symptoms prevail, even this remedy is of no avail. As regards tea and coffee, which often relieve, it is possible that these and other stimulants, taken in excess, render the nervous system more susceptible to the attacks.

In the treatment of the paroxysm nothing is so efficacious as complete rest in the recumbent posture, with warmth, in a perfectly quiet and darkened room. A cup of very strong tea or coffee has also given relief. A comparatively new drug—*guarana*—has also been recommended. A full dose of *brandy,* or of *ammonia,* has also sometimes dispelled the headache; so has continued sipping of a solution of *carbonate of potash* or of *soda.* Pressure upon the carotid artery of the affected side sometimes also relieves the pain, but for a time only. Evaporating lotions, *belladonna,* or *aconite* ointment may also ward off or subdue the headache; but, as a rule, time is the most efficient element, with warmth to the feet and to the body, and the induction of sleep.

Adopting the theory of Dr. Latham, what are the indications for treatment with which it supplies us—(*a.*) During the stage of disturbed sensation; (*b.*) during the stage of headache; and, (*c.*) during the intervals between the attacks? The following are the results of his experience:—

(*a.*) *During the Stage of Disturbed Sensation.*—It is found that the longer this stage lasts, the greater will be the headache; and therefore it is desirable to shorten it as much as possible. If the condition depend upon deficient supply of blood to a part, such means must be adopted as shall assist and increase the flow of blood to the part; and this can be done in some measure by posture and stimulants. Directly the glimmering appears, the patient should lie down with the head as low as possible, and if the glimmering be on the right or left of the field of vision, he should lie on the *opposite* side. Let him take at once one ounce of *brandy,* either "*neat*" or in half a bottle of soda-water. Champagne would be preferable, being more diffusible; but its administration would often involve a little delay. If alcoholic stimulants be objected to, or if it be not advisable to recommend them, then a teaspoonful of *sal-volatile* in water may be prescribed instead. If the patient be chilly or his feet cold, the couch should be drawn before the fire, a hot bottle applied to the feet, and his body covered over with a warm blanket or eider-down quilt. By these means the heart is enabled to drive the blood with greater force to the brain, and the duration of the vibratory movement is thereby materially lessened. After it has passed off, the patient should lie still for a time, so that the glimmering may not return. This injunction will only be necessary when the headache is slight; if it be severe, attended with much nausea or vomiting, the patient will be little disposed or able to leave the recumbent position. If, instead of the disturbance of vision preceding the headache, there be a feeling of depression or irritability (fidgets), the administration of such cerebro-spinal stimulants as *henbane, valerian, assafœtida, spirit of chloroform,* or *ether*, will often cut short the attack; ten or fifteen drops of the tincture of *henbane*, with the same quantity of *spirit of chloroform*, will soothe the nervous irritability in the slighter forms, and may be repeated in three or four hours, if necessary. If there be great mental depression, then *valerian* or *assafœtida* should be tried. Stillé says,—"Nothing is more astonishing in the operation of remedies than the promptness and certainty with which a dose of *valerian* or *assafœtida* dispels the gloomy visions of the hypochondriac, calms the hurry and agitation of nervous excitement, allays commencing spasms, and diffuses a soothing calm over the whole being of one who but an hour before was a prey to a thousand morbid sensations and thick-coming fancies of danger, wrong, or loss." Dr. Latham gives the preference to *valerian*, and prescribes from half a drachm to a drachm of the *ammoniated tincture.* Or it may be combined, as in the following formula, with bromide of soda: *ammoniated tincture of valerian* ℥iv. with ʒvi. *bromide of sodium,* in teaspoonful doses every hour. The *assafœtida* may be given in the form of the *spiritus ammoniæ fœtidus* of the Pharmacopœia, also in half drachm or drachm doses. As a rule, alcoholic stimulants are not advisable in this stage of the headache. A small quantity will cause flushing, heaviness, slight confusion of thought, &c., without relieving the depression; and though the severe headache may be averted, alcoholic stimulants do not answer so well as the remedies previously mentioned.

(*b.*) *During the Stage of Headache.*—If the headache be slight and the patient soon able to sit up, there is little to be done: a cup of coffee or tea, cheerful conversation, a walk, drive, or ride, may often help to remove the pain. If, however, the headache and nausea be severe, then the administration of further remedies is called for. The patient should keep perfectly still and quiet, with the room darkened; for every sound or sight causes pain, and the slightest movement is sufficient to produce gastric uneasiness. Sometimes free evacuation of the contents of the stomach, especially if it contain undigested food, is followed by relief. Dr. Fothergill says,—"An emetic and some warm water soon wash off the offending matter, and remove these disorders," which may be very well where there is any offending matter to wash off, but it is not very often that this is the case; the nausea frequently continues long after the contents of the stomach have been discharged; an inverted action of the duodenum is set up; the bile appears in the fluids excreted; the patient believes that all his troubles are due to "its overflow;" "it 's all liver," he says, and it is sometimes difficult to persuade him to the contrary. Generally, then, you should try to relieve and check the vomiting. Iced *soda-water*, with or without two or three drops of *dilute hydrocyanic acid,* or *spirit of chloroform; cold tea;* the effervescing *citrate of potash,* with *hydrocyanic acid,* may often afford marked relief. The headache may be lessened by applying cloths dipped in cold water, or evaporating lotions, to the head; if the extremities be cold and the headache severe, a warm stimulating foot-bath can be tried so soon as the nausea will allow the patient to sit up. If the attacks occur in the early part of the day, as soon as the pain has subsided it is generally better for the patient to sit up, or move about, or take exercise in the open air. During the attack the appetite is diminished, the idea even of food provoking disgust. Still, after the nausea has passed away and the headache has continued a few hours, a plate of hot soup or some easily digested food will often have a good effect in equalising the cerebral circulation. A remedy which may very often be given with advantage if the headache be severe, is *bromide of potassium,* in doses of five, ten, or fifteen grains, to which thirty or forty minims of *sal-volatile* may, in some cases, be added with advantage; and if the nausea still continue, these may be given in combination with the effervescing *citrate of potash.* A *saline purgative* at the commencement of an attack is sometimes an effectual remedy; but, as a rule, the use of purgatives is objectionable. So far, these measures are only palliative.

(*c.*) *During the Intervals between the Attacks.*—First of all, the exciting cause is to be discovered and removed. Hours of study or work must be abridged; excessive bodily fatigue, loss of rest, everything, in fact, must be avoided which the sufferers know from individual experience will act as exciting causes. Where the attacks are associated with excessive mental work, they should be regarded as danger-signals, showing necessity for relaxation. The tone of the bodily and nervous systems must be improved by proper medicinal and hygienic means; and the chief remedies are *steel, strychnine, phosphorus,* and *cod-liver oil.* The success, however, following these remedies depends a great deal upon the way in which they are administered. For a day or two after the attack the stomach and bowels may possibly be disordered, and not in a fit state to tolerate such remedies. This must first be corrected. The simple vegetable bitters, such as *gentian,* with small doses of *henbane* and some aromatic, may be of service; and, if necessary, one or two grains of *blue pill,* with four or five of *compound rhubarb pill,* may be given at night. We may then try *steel.* In anæmic cases it may be advisable to stimulate the action of the heart, as by the following :—

℞. *Ammoniæ muriatis,* ʒ ss., *Tinct. actææ racemosæ aquæ, aa* ʒ iii. Two fluid drachms for a dose after meals in a wineglass of water.

If there be despondency with depression of spirits, *phosphorus* to the extent of $\frac{1}{100}$ to $\frac{1}{50}$ of a grain, combined with *nux vomica* or *carbonate of iron,* or both, taken with each meal, will improve the tone of the patient. If the attacks have been very frequent, or if there be any scrofulous tendency, the *iodide of iron* may be given in the following form :—

℞ *Ferri et ammon. citrat.,* gr. v.; *potassii iodidi,* gr. ij.; *aquæ,* ℥j.; and, according to circumstances, fifteen to twenty minims of *tincture of henbane,* or twenty or thirty minims of *aromatic spirit of ammonia* may be added.

If the stomach be at all irritable, this medicine may be given in the effervescing form, adding to each dose twenty grains of *bicarbonate of* *potash,* and directing it to be taken with a tablespoonful of *lemon-juice,* or a corresponding amount of *citric acid:* the dose to be taken twice a day, about eleven and four. The effervescing form may be soon left off, and then to each dose may be added five minims of *liquor strychniæ* (omitting the *henbane* and *sal-volatile,* and continuing the *iodide of potassium* according as it seems to be indicated or not). In other cases, the *citrate of iron* and *ammonia* with *strychnine* may be given at the beginning, sometimes combined with *infusion of calumba.* The iron is indicated by the greater or less anæmia of the patient; but the *strychnine* is a very important remedial agent in the disorder. In small doses it acts as a simple tonic, increasing the appetite and improving the digestion; it dilates the vessels, and thus increasing the supply of blood, it augments the activity of the spinal cord (HARLEY). It promotes the capillary circulation, and therefore its use is advisable for persons troubled with cold hands and feet (ANSTIE); and if it fulfil these conditions, it is clearly indicated in this disorder. *Cod-liver oil* also often acts very beneficially. "It has been found by experiment that great exertion and prolonged labour can be endured without fatigue when starchy and fatty foods are alone eaten; . . . and there is reason to think that cod-liver oil is more easily absorbed than other similar substances" (RINGER). "It improves the digestive process, increases the proportion of red corpuscles in the blood, and invigorates the whole nutritive function" (WOOD); and it particularly sustains the energy of the brain during prolonged mental exertion. A gentleman in the foremost rank at the bar told Dr. Latham that whenever he was engaged in a jury-trial which was likely to tax his energies to a greater degree than usual, the thing which best sustained him was a good dose of cod-liver oil taken in the morning before going into court; and others engaged in mental work have confirmed this view. Besides its other properties, cod-liver oil is a nutrient and tonic in its action on the cerebrospinal nervous system. As a remedy for these nervous headaches, Dr. Latham prescribes it once a day, beginning with a small teaspoonful immediately after breakfast, and gradually increasing the quantity to a tablespoonful, but not beyond, unless in exceptional cases.

The action of the bowels must be regulated, but not by strong purgatives. Five grains of the *Socotrine aloes pill,* given at night, are generally sufficient. If the bowels be habitually constipated, then no remedy seems to answer so well as the *aloes and iron pill.* Five grains given twice a day, half an hour before meals, will act freely; and in a few days the dose will have to be diminished, for its effect is augmented instead of being lessened by continual administration, especially when *strychnine* is given at the same time. The natural waters of Friedrichshall or Marienbad may, in many instances, be of service, given as laxatives.

In 1872 Dr. Samuel Wilks, Physician to Guy's Hospital, directed the attention of the profession to *guarana* as a remedy for sick-headache, and at the same time asked for the experience of those who already had some acquaintance with the drug. His own knowledge of it dated about 1870, when, after the appearance of a lecture of his upon sick-headache, he received a letter from Mr. Helmcken, of British Columbia, inclosing two powders, which he recommended with much confidence as able to cure the complaint. "Upon my first headache after the receipt of Mr. Helmcken's letter, I took the powder, but with only doubtful effect. I therefore did no more than casually mention the medicine to my friends, but did not recommend it. A few weeks ago, after the appearance of a second communication of mine in the journal upon the same complaint, I received a letter from Dr. Wood, of Montreal, in which he also recommended *guarana* as a remedy for headache, and gave a history of his own personal sufferings and the relief which he had obtained. He says,—'By taking one of these powders and remaining quiet when I have felt premonitory symptoms by a beginning of pain always in the right temple (headache on the other side, or in any other part of the head, I never mind), I have carried off the attack; and, with the first box, absolutely put it off for two months—something which had never occurred in my life before.' Upon so good an authority, I determined to try the remedy in a more systematic manner, and requested my neighbour, Mr. Hooper, the chemist, to procure me a packet of the powders. These I have recommended to several patients and friends; and the result is so encouraging, that I have hastened to suggest their trial to my professional brethren. One lady speaks most enthusiastically of their power, as she has now, on two separate occasions, had her headache arrested by their use. The drug has long been known, for mention is made of it in English and French pharmacologies, but appears never to have come into general use. It consists of the seeds of a tree growing in Brazil, called *Paullinia sorbilis;* and these, according to Johnstone, in his *Chemistry of Common Life*, are used as we do cocoa. The seeds are ground into powder, and contain an alkaloid which is said to be identical with that found in tea and coffee. The medicine is manufactured by Grimault & Co., No. 7 Rue de la Feuillade, Paris" (WILKS, *Brit. Med. Journ.*, April 20, 1872).

The general outcome of experience regarding the treatment of sick-headache seems to be that,—

(1.) *Bromide of potassium, Indian hemp, Guarana*, or *Paullinia powder*, are remedies which have afforded relief. With reference to *bromide of potassium*, it is so valuable a medicine in many cases of sick-headache that it can scarcely be superseded by a better remedy. The patient may have a splitting headache, fatigued and worried after a hard day's work. After fifteen or twenty grains of *bromide of potassium*, he presently goes off to sleep in his easy chair, and wakes in an hour well (WILKS). It is a medicine which Dr. Wilks always employs first, having seen such eminent advantages follow its use. "I have known (he writes) many patients declare, that the bromide was the first medicine they had taken in their lives which had had the slightest effect in relieving their headache." As to *Cannabis Indica*, it seems best adapted to the interval between the attacks, in doses of a few drops of the tincture, when headaches are recurrent. It is best given in ten-minim doses, three times a day, continued for weeks or months (WILKS, JOHN MURRAY). As to *guarana powder*, it seems most of all useful with females in arresting headache, so that it must be taken early when the headache is approaching; and if headache is not arrested, a second dose should be taken in fifteen, twenty, or thirty minutes after the first. If the medicine is of use, the headache will have gone in about an hour. The dose is 15 grains of the powder; and as it is somewhat astringent, it might be combined as an electuary with some laxative, such as the *senna electuary* (HOLLIS).

(2.) Certain classes of cases ought to be distinguished from each other. There are two principal types:—the congestive, met with in dark or plethoric people, in those with constipated bowels and suppressed secretions, and those addicted to alcoholic beverages, or leading a monotonous sedentary life; and that arising from exhaustion, whether from want of sleep or want of food, or great bodily or mental fatigue, strong emotions, &c.—this may be nervous, or asthenic, or anæmic in many cases. This latter form is very common in slight, delicate, and tuberculous females, and sometimes in the male sex. The treatment of these two forms must, of course, differ widely. An emetic or a purge in the one case will do what only rest, food, stimulants, and such medicines as *quinine*, *iron*, *opium*, *hyoscyamus*, and *belladonna*, may afford in the other case (Dr. BATHURST WOODMAN). Rest and quiet, especially sleep, and abstinence from food, give relief and cure by a natural process of restoration. Generally, no relief is obtained till after a night of sound sleep—*i. e.*, "one night through the sheets"—awaking next morning free from pain, but generally more or less depressed. If depression continue, *phosphorus* with *nux vomica* in the form of sugar-coated or pearl-coated pills may be given to the extent of one-fiftieth of a grain of *phosphorus* and one-eighth of a grain of extract of *nux vomica* three times a day with food. Such despondency and depression are apt to occur in those who are overworked mentally; or who are harassed by business; or who suffer great mental anxiety. If there be sleeplessness, the following pill may be of use:—

℞ *Camphoræ pulveris*, gr. xx.; *Ext. Cannabis Indica*, gr. xii.; *Ext. Hyoscyam.*, gr. xxiv., *misce bene.* Divide in pill xii. *signa*, one at night, to be repeated in two hours, if necessary, to procure sleep.

As to local applications, a large sinapism over the stomach, followed by a warm linseed meal poultice, has sometimes been of service. If pressure relieves, the head may be bound round with a handkerchief. When the head throbs and is hot instinct seems to suggest cold and pressure to the part; and one of the best external appliances on which we can rely to procure relief is the wet bandage tied tightly round the head. The method must be instinctive, for it is universal, and has been used from all time. Shakespeare is often quoted to illustrate the morbid states of the body as well as the passions of the mind, and he also testifies to this ancient practice. For example, in the scene between Hubert and Arthur in *King John*, the latter, when petitioning for the preservation of his eyes, says—

> "When your head did but ache
> I knit my handkerchief about your brows."

And in *Othello* we have not only the remedy for headache given, but the cause. The former was the handkerchief about which the chief interest of the play centred.

> "*Desdemona*, Why do you speak so faintly?
> Are you not well?
> *Othello*, I have a pain upon my forehead here.
> *Desdemona*, Faith, that's with watching: 'twill away again.
> Let me but bind it hard, within this hour
> It will be well."

Sick-headache is not to be cured by gastro-hepatic remedies. It is a purely nervous affection, due to hereditary predisposition, and excited by causes innumerable which act on a susceptible nervous system. There is, then, no cure in the proper sense of the term, for this would imply a change in the patient's nature; and for the attacks themselves, when severe, the only relief which can be reckoned upon is to be found in a wet bandage round the head, profound quiet, and, if possible, sleep (Dr. BATHURST WOODMAN).

Considerable relief may be obtained from the use of cold applications, in the form of wet rags, ice in a bladder, or a little of the ether-spray; but in not a few cases, after all, the best treatment during the attack is absolute rest and quiet, in a dark room, with warmth to feet and body, total abstinence, except perhaps from fluids (hot and alkaline) in small quantities at a time; to go to bed at once, and refrain from the use of any remedies.

In a lecture on the therapeutics of headache, delivered at Bellevue Hospital Medical College, Dr. A. A. Smith suggests certain combinations of remedies which he has found of service in certain forms of headache:—(1.) *Malarial Headache*, where the pain is most frequently located in the sub-occipital or frontal regions, or on either side—hemicrania. If distinctly periodical, give ten or fifteen grains of *quinine* two or three hours before the expected attack. It may be necessary to push the *quinine* in divided doses until cinchonism is produced, and kept up for several days. Then gradually diminish the dose. If the pain still continues to recur, resort to *arsenic* and *belladonna*, five-drop doses each of *Fowler's solution* and *belladonna tincture*, after meals, increasing the Fowler's one drop each day, until *œdema arsenicalis* is produced. This will seldom fail to give relief.

(2.) In *Headache dependent on Gout:*—

℞ *Vini colchici seminum*, ʒiij.; *Lithii bromidi*, ℥ss.; *Syr. zingiberis*, ℥ss.; *Aq. cinnamonii*, q. s. ad ℥vj.; M. Sig. ℥ss. in a tumbler of Vichy water every four hours.

(3.) *Rheumatic Headache* is characterised usually by tenderness of the scalp, which is increased on pressure or motion. Use the mild Faradic current on the scalp, and internally the following:—

℞ *Potassii iodidi; Ammoniæ muriatis*, āā ʒjss.; *Infusi humuli*, ℥vj.; M. Sig. ℥ss. four times a day in a wineglass of water.

In cases which have not yielded to the above treatment, *bromide of ammonium* in twenty-grain doses every two hours may be effectual.

(4.) *Uræmic Headache* is of great importance as a symptom of serious disease. The pain in the head may be the first evidence that there exists renal disease, and that we have to deal with uræmic headache. The judicious plan of treatment in such cases is to call into action one or all of the three great emunctories of the body, the kidneys, the intestines, and the skin. To make the kidneys act, apply dry cups over the region of them, and give internally the following:—

℞ *Potassæ acetatis*, ʒvj.; *Infusi digitalis*, ℥vj.; M. Sig. ℥ss. every third hour.

The infusion should be made from fresh English leaves. Give this until the kidneys act freely, if you can make them do it within twenty-four hours. If the kidneys do not act freely, and the headache be not relieved within twenty-four hours, give a saline cathartic. A treatment almost domestic, and often very effectual, is to put an ounce of *cream of tartar* in a quart of water, and have the patient drink this in eight or ten hours. It acts both as a diuretic and cathartic. Do not use hydragogue cathartics unless convulsions be threatened. Some prefer elimination by the skin. This can be done by diaphoretics and the hot, moist, or dry air bath. Diuretics, cathartics, and diaphoretics are only to be used where there is deficient quantity of urinary secretion. There are other causes of headache in Bright's disease which occur independently of the presence of an abnormal amount of urea in the blood, and which yet are dependent on the results of the kidney-disease. These causes may be anæmia, neuralgia, œdema of the brain itself, serous effusion into the ventricles; and, in acute Bright's disease, cerebral congestion. Under the last condition, if the headache be very severe and convulsions threaten, blood may be taken if the patient's condition will admit of it, from twelve to twenty ounces, if necessary to relieve distressing symptoms. The best way to take it is by means of wet cups over the region of the kidneys. If the headache be dependent on serous effusion into the ventricles, or on cerebral œdema, improve the vitiated condition of the blood, and stimulate the heart and kidneys by *acetate of potash* and *infusion of digitalis*. There is apt to be general anasarca with this effusion and œdema.

(5.) *The Headache of Acute Alcoholism*, or inebriety. The first indication is to remove the alcohol from the intestinal canal. For this, give of *rhubarb* and *magnesia calcined* each a half drachm, and then the following:—

℞ *Spiritus ammoniæ aromat.*, ʒij.; *Tincturæ camph.*, ʒjss.; *Tincturæ hyoscyami*, ʒijss.; *Spiritus lavandulæ comp.*, q. s. ad ℥ij.; M. Sig. ʒj. every hour until the headache is relieved, and then give *capsicum* gr. ij, and *quinine* gr. iij, before each meal for several days. If there be sleeplessness give ℞ *Sodii bromidi*, ℥ss.; *Chloral. hydrat.*, ʒijss.; *Syrupi aurantii cort.*, ℥ss.; *Aquæ*, ℥iijss. M. Sig. ℥ss. at night; repeat in two hours if necessary to produce sleep.

(6.) *Dyspeptic Headache.*—If there be indigestible food in the stomach, and it have been there some time, give an emetic, as mustard and warm water, or *sulphate of zinc*, gr. xv. If there is evidence of indigestible food in the alimentary canal beyond the stomach, give gr. xx. of *rhubarb* and *magnesia* each, to remove it from the bowels. If the headache be frontal, and the pain be located immediately over the eyes, give *dilute* *nitro-muriatic acid* in ten-drop doses, well-diluted, after meals. If the pain be located about the roots of the hair, give an alkali before meals, as gr. xv. of *bicarbonate of soda* or *magnesia*. The dyspeptic headache oftentimes is not confined to these regions, but spreads over the entire head. In such cases combine an acid with an alkali, and add to these *nux vomica*, as in the following prescription :—

℞ *Sodæ bicarbonatis*, ʒijss.; *Acidi nitro-mur. diluti*, ʒij.; *Tinct. nucis vomicæ*, ʒjss.; *Syr. aurant. cort.*, ʒvj.; *Aquæ*, q. s. ad ℥vj. M. Sig. ℥ss. after meals in a wineglass of water.

If there be gastric pain, a mild counter-irritant, as a mustard plaster to the epigastrium, will often relieve the pain in the head as well as the pain in the stomach. If flatulence be a troublesome symptom, give the following :—

℞ *Bismuthi subcarbonatis*, ʒijss.; *Tinct. nucis vomicæ*, ʒjss.; *Tinct. cardamomi comp.; Spiritus lavandulæ comp.* āā q. s. ad ʒiv. M. Sig. ʒij. before meals in a wineglass of water.

If there be constipation, the following pill may be given, one in the morning :—

℞ *Aloes pulv.*, ʒss.; *Ext. nucis vomicæ*, gr. v.; *Ext. belladonnæ*, gr. iv.; *M. Div. in pilulas*, xv.

In some forms of headache associated with stomach indigestion, small doses, often repeated, of *tincture of nux vomica* may be effectual. A single drop every fifteen minutes, and continue this two or three hours if necessary. In other cases, where the headache comes on soon after a meal, and seems to depend on delayed stomach digestion, large doses of *pepsin* are effectual. Give a half drachm of *saccharated pepsin* in a wineglass of sherry wine, three times a day, and let it be taken during meals.

(7.) *Congestive Headache.*—Cerebral congestion as a cause of headache may be divided into two varieties, *active* and *passive*. These demand almost directly opposite plans of treatment. In the *active variety* the patient should be kept in a darkened room, perfectly quiet, and have cold and evaporating lotions applied to the head. A saline cathartic may be given; and the following prescription :—

℞ *Sodii bromidi*, ʒijss.; *Fl. ext. ergot.*, ʒijss.; *Syr. zingib.*, ℥ss.; *Aq. aurant. flor.* q. s. ad ℥iv. M. Sig. ℥ss. every two hours.

If the skin be hot and dry, and the pulse full and rapid, give two drops of *Fleming's tincture of aconite root* every two hours, until the heart's action is sensibly diminished. Sometimes also a hot mustard foot-bath will give relief. The *passive congestive variety* demands a different mode of treatment. It is often found associated with cardiac disease, and most frequently where there is predominant dilatation. Hypertrophy gives rise to the active variety. Improve the condition of the blood by the use of *iron*, *quinine, bitter tonics, alcoholic stimulants,* good food, and stimulate the heart's action by the use of the following:—

℞ *Tinct. digitalis,* ʒiij.; *Spirit. amm. aromat.,* ʒvj.; *Spirit. lavandulæ comp.; Syr. simp.,* aa q. s. ad ℥iij. M. Sig. ʒj. every four hours.

(8.) *Anæmic Headache.*—Cerebral anæmia produces a headache which is often mistaken for the passive cerebral congestive form. It is associated with general anæmia, nervous exhaustion, and may occur in heart disease, in consequence of enfeebled heart power, such as is met with in enlargement with dilatation, fatty degeneration, and myocarditis. Improve the general condition of the patient, and stimulate the heart's action as recommended in the passive cerebral congestive variety. *Nitrate of amyl* will relieve the immediate headache. Let the patient inhale three to five drops of it on a piece of cotton, placed within one nostril, while the other is held closed. When associated with nervous exhaustion, employ the following remedies:—

℞ *Strych. sulph.,* gr. ss.; *Tinct. ferri chloridi,* ʒij.; *Glycerinæ,* ℥ss.; *Infusi gentian.,* q. s. ad ℥vj. M. Sig. ℥ss. after meals, in a wineglass of water.

Alcoholic stimulants are beneficial in headache dependent on cerebral anæmia. Champagne as a remedy, and not as a beverage, may be given to those who suffer from nervous exhaustion; or a tablespoonful of brandy, diluted with water, after each meal.

(9.) *Cerebral tumors* give rise to headache, often severe. In all such cases, give *iodide of potassium;* for it cannot be safely said that in any given case the tumor does not depend on syphilis, and by administering the remedy, the patient gets the benefit of the doubt.

There is reason to believe, too, that patients with cerebral tumors, dependent on other and unknown causes, are benefited by the use of *iodide of potassium.* If there be much sleeplessness, use the *bromide* and *chloral mixture* (p. 132, *ante*). *Ergot* has also been used. It is usually given in large doses, beginning with ʒi. of the *fluid extract* three times a day, and gradually increasing the dose to half an ounce (*New York Med. Record,* and *London Med. Record,* Sept. 15, 1876).

ANÆSTHESIA.

LATIN EQ., *Anæsthesia;* FRENCH EQ., *Anesthésie;* GERMAN EQ., *Anæsthesie;* ITALIAN EQ., *Anestesia.*

Definition.—*Anæsthesia implies an impairment, diminution, or complete loss of sensitiveness to external impressions over various regions of the body.*

Pathology.—Two kinds of sensibility have been shown to exist in the skin and mucous membrane round the natural orifices: the one is *tactile sensibility,* by which *form, temperature, consistence,* and such like qualities of a body are distinguished by touch; the other kind of sensibility appreciates the sense of pain. The impairment, diminution, or loss of the first has been distinguished by the term *anæsthesia;* of the latter by the term *analgesia.* The skin may also lose the sense of pain and still retain the power of touch. The finger may then be felt when rubbed over the skin, but the skin is insensible to pricking or pinching. In extreme cases the skin and subjacent parts may be cut, burnt, or otherwise injured without the patient's knowledge; but when common tactile sensibility is lost, the sense of pain is also lost, except in some instances of centric reflex paralysis, and likewise the appreciation of heat and cold (BEAU).

Anæsthesia most commonly occurs as an immediate antecedent of motor palsy, or coincident with it, and also exists in various parts of the body, independent of paralysis of motion. It varies in extent from a tingling, pricking formication, or mere numbness of the parts, to a complete loss of sensation. The patient feels as if the affected part were protected or covered by some thick soft texture. The hands feel as if clothed with gloves, or the feet experience the sensation as if walking on felt, cotton, wool, or other yielding soft substance. The cutaneous nerves are those most frequently affected, and from this cause the disease most usually advances to the integuments of a portion of the trunk, or of an arm, or a leg, or some portion of the extremities, and also the whole face or parts of the face.

The pathology of anæsthesia may be comprehended from the following considerations:—The function of sensation depends upon the concurrence of three sets of organs, anatomically continuous, yet to a certain extent physiologically distinct—(1.) The nerve-extremities, which receive an impression; (2.) the afferent nerve-fibres, which carry the impression to the centre; (3.) the centres, which receive the impression and transform it into a sensation. Thus the three factors of sensation are, the sensitive nervous extremities, the sensory conducting filaments, and the sentient portions of the nervous centres. If the integrity of any one of these is affected, there must result diminution or loss of sensation. If perception in the sensorium is destroyed, the nerves may transmit the impression to the sentient centres, but there is no sensation. This cause of anæsthesia occurs in cerebral hemorrhage, softening, and tumors; or the function of the organ may be temporarily in abeyance, from the effect of certain agents, as chloroform or narcotics. Anæsthesia is also produced by interruption of the continuity of the nerve-trunk, by section, or compression, or atrophy, or tissue-degeneration. A third mode is, whilst both the nerve-centres and the nerve-trunks are sound, the sentient nerve-extremities have undergone some morbid change, and the impression is not received, as in insensibility of the skin from the application of intense cold, blindness from disease of the retina, loss of the sense of smell during coryza, and certain cutaneous eruptions. It sometimes happens as the result of reflex action in visceral disorders, and after mechanical injuries (BROWN-SEQUARD, S. W. MITCHELL). This pathogeny of anæsthesia being admitted, can we determine clinically the seat of the morbid condition, and refer it to a given lesion of the brain or medulla, or of the nerves themselves? Is the anæsthesia central or peripheral?—(1.) *Anæsthesia resulting from cerebral disease* is usually of large extent, and may involve the whole body, as in the general paralytic condition which is associated with a special form of insanity, and in which there is general impairment of sensibility. Generally it is unilateral, and on the side opposite to the brain lesion. Such hemi-anæsthesia may arise from disease of one of the cerebral hemispheres, or the ganglia or crus immediately connected with it; and it rarely occurs independently of hemi-paralysis. It is commonly the consequence of disease involving the optic thalamus and white substance in its immediate vicinity, or the subjacent tegmentum. Such hemi-anæsthesia may be absolute, the patient feeling nothing. More frequently it is incomplete, the patient retaining more or less sensation in certain parts. When disease involves the pons or medulla oblongata, some degree of anæsthesia is apt to be associated with motor paralysis (*bulbar* anæsthesia), and all the sensory nerves taking origin in these parts are, like the motor nerves, apt to be implicated (Bristowe). Voluntary motion is also lessened or abolished, the reflex movements are commonly intact, intelligence is disordered, and the nutrition of the paralysed parts impaired. (2.) *Anæsthesia consecutive to spinal disorder* is mostly less general than that caused by disease of the brain; it is, as a rule, bilateral and symmetrical, but when unilateral it is on the same side as the affected column of the medulla (though Dr. Brown-Sèquard admits crossed spinal paralysis). Disease of one lateral half of the spinal cord, interrupting the longitudinal continuity of the fibres, causes anæsthesia of the opposite side of the body. Voluntary motion is lessened or destroyed; reflex functions may be preserved or absent, according as the medulla is or is not sound below the site of the lesion; nutrition of the affected parts is often greatly impaired. It is rarely met with apart from paralysis. (3.) *Anæsthesia depending on some lesion of a sensory nerve or its nucleus* is often very limited, its site corresponding exactly to the distribution of the nerve. Voluntary motion is intact if a nerve exclusively sensory is involved, or if it be a compound nerve the paralysis is limited to a single group of muscles. Reflex movements are constantly wanting where the anæsthesia is complete, and the state of the nutrition of the affected part is variable, but often good. It occupies the same side of the body as that on which the lesion exists. The anæsthesia which forms the upper boundary of the paralysed region in cases of unilateral injury or disease of the cord is a typical example of this condition (Bristowe).

Peripheral is distinguished by Niemeyer from *central anæsthesia* mainly by the following points:—(1.) Extension of the anæsthesia and its complication, or non-complication, with motor palsy. If the anæsthesia be confined to one side, and be combined with hemiplegia, it is probably cerebral. If, however, it extend over the lower half of the body, and be combined with paraplegia, it is probably spinal. The co-existence of palsy of the face with facial anæsthesia indicates a central cause of disease, while anæsthesia with normal motion indicates peripheral origin. (2.) Reflex symptoms may be present in *central anæsthesia*, but they are entirely absent in *peripheral anæsthesia*.

One of the most important forms in which *sensory nerve* anæsthesia comes under the notice of the physician is that known as Facial Anæsthesia. It consists in an impaired, deficient, or entire loss of sensibility in the parts supplied by the fifth pair of nerves.

The following phenomena require to be considered in the diagnosis of this form of anæsthesia:—(1.) The more the anæsthesia is confined to single filaments, the more peripheral is the seat of lesion. (2.) If the loss of sensation affects a portion of the facial surface, together with the corresponding cavity of the face, the disease may be presumed to involve the sensory fibres of the fifth pair before they separate, or one of the main divisions after its passage through its cranial foramen. (3.) When the entire sensific tract of the fifth nerve has lost its sensation, associated with derangements of the nutritive functions in the affected parts, the Gasserian ganglion, or the nerves in its immediate vicinity, may be the seat of the disease. (4.) If the fifth nerve is complicated with disturbed functions of adjoining cerebral nerves, it may be presumed that the lesion is situated at the base of the brain (ROMBERG). The symptoms may develop themselves gradually, or may come on suddenly, and are not unfrequently preceded by neuralgia; and, owing to the differences in the seat of lesion, what may seem to be a permanent symptom in many is absent or less marked in others. It is frequently complicated with facial palsy of the *portio dura* of the seventh nerve. In a well-marked case the symptoms are:—loss of tactile sensibility of the parts supplied by the nerve—namely, the integuments of the cheek and side of the head, the eyelids, conjunctiva, tongue, Schneiderian membrane—accompanied by loss of taste on the side of the tongue which is affected; frequently by loss of smell and hearing, and by inflammation of the eye, terminating in ulceration of the cornea, and by no means uncommonly in total disorganisation of the globe. Paralysis and wasting of the deep muscles of mastication are also sometimes associated. The real nature of the affection may escape notice if the attention is directed solely to any one prominent symptom. The disease makes its onset in two or three ways. It is often sudden, preceded by a slight perversion of sensibility, or by a tingling sensation, frequent attacks of headache, and pains in the occipital region and side of the face. Dimness of vision is so prominent a symptom in some cases that loss of sensibility may not at first attract attention. Of twenty-four cases, vision was affected in fifteen, hearing in nine, and smell in six (Dr. J. B. COWAN, in *Glasgow Med. Journal*, No. 2, July, 1853).

There are two important features in facial anæsthesia worthy of special notice—namely, 1. That the parts do not waste as in muscular palsy; 2. that though the parts are insensible to touch, they still remain sensible to changes of temperature, and to pain arising from inflammation in the nerve itself.

Facial anæsthesia may result from disease—(1.) Of the cerebrum, where the fifth nerve takes its origin; (2.) of the nerve within the cranium; (3.) of the nerve after it has emerged from the cranium; and according to the seat the symptoms vary. The disease may be a consequence of some injury, such as the extraction of a tooth, as frequently happens to the submental branch, which conveys sensation to half the lower lip.

Dissections show that condensation, atrophy, softening, and the pressure of tumors are the chief morbid conditions out of which facial anæsthesia springs. So varied are the sources whence the anæsthesia results, that its treatment can only be palliative, and directed towards mitigation of the symptoms. Spontaneous cures are said not to be unfrequent. The local applications, such as blisters, leeching, and cupping, are said to have been sometimes useful, combined with the administration of purgatives and the continuous or interrupted current of electricity.

Other forms of anæsthesia are those of the special senses; such as, loss of sight from *amaurosis*, nervous deafness (*paracusis*), loss of smell (*anosmia*), and loss of taste (*aguestia*). *Cutaneous anæsthesia*, as has been shown, may be centric or peripheral, and may vary from slight numbness to complete abolition of sensation, with no sense of pain or appreciation of heat (*thermo-anæsthesia*), and be bilateral, or unilateral, or limited to a single spot of some extent; sometimes its degree varies in different parts of the body. It is frequently associated with anæsthesia of the mucous membrane (generally the conjunctival), and with muscular anæsthesia. Besides organic lesions of the brain, medulla, and nerve-trunks, it occurs in functional disorders of the centres, as hysteria, chorea, and occasionally in insanity (AXENFELD); in visceral disease (the reflex form); after excessive fatigue (SANDRAS); after the local application of cold and certain irritating substances (ROMBERG); from the effects of certain toxic articles, as lead, arsenic, bromide of potassium, sulphuret of carbon, nitrous oxide gas, sulphuric ether, chloroform; in the course of and after septic diseases, and in asphyxia (FAURE). *Muscular anæsthesia* has been defined to be a diminution or loss of the muscular sense. There is abolition of muscular consciousness; the patient, if his eyes are withdrawn from the limb, is unable to tell its position, whether it is flexed or extended, abducted or adducted; and he may try to extend a limb which is already stretched out. There is no appreciation of the amount of force required to meet or overcome any opposing force; the weight of the limbs is not felt; and the degree of density of various bodies cannot be estimated. The perception of muscular contraction is totally or measurably gone. Muscular anæsthesia, when fully developed, is even more unequivocally shown by the absence of sensibility when the muscles are pinched, squeezed, or shampooed, or an electric current passed through them, or a sharp instrument pushed into them. It may be unaccompanied by the loss of the function of motility, shown by the ability to execute all necessary movements when the eye is on the limb, and by the amount of muscular vigour that can at need be exhibited, and by the feeling of lightness and the absence of all sense of fatigue in the extremities, so opposite to the heaviness and weariness of muscular paralysis; or there may be motor palsy, as in hysterical paralysis; or it may be accompanied by insufficiency in the power of co-ordinating voluntary movements. It frequently coexists with cutaneous anæsthesia. Laundry speaks of numbness and formication as precursory phenomena, but they probably really belong to the primary disorder of the nervous centres. It is more fixed than cutaneous anæsthesia, and rarely presents, even in hysteria, those fitful mutations of site so common in the latter. The prognosis depends upon the presence of centric lesions (BEAU, BROWN-SÉQUARD, SZOKALSY, LAUNDRY, BELLION, VULPIAN, YELLOWLY, BRACH, DUCHENNE (de Boulogne), BOURDON, AXENFELD, quoted by CLYMER).

Dr. Bristowe directs attention to the following points in the pathology of anæsthesia:—(1.) Persons suffering from anæsthesia generally experience subjective sensations referable to the anæsthetic regions, and not unfrequently complain of neuralgic and other pains in them. (2.) Sometimes when sensation is greatly impaired, the patient does not take cognisance of impressions made on the affected part until after the lapse of a few seconds, or it may be as much as half a minute. The impression appears to be delayed in its transmission to the sensorium. (3.) Under similar circumstances such patients often have a peculiar difficulty in distinguishing between the characters of different impressions. (4.) Inasmuch as it is through the sensory or afferent nerves that reflex motor phenomena are excited, it is obvious that if the disease causing anæsthesia exist in the course of the nerve or in its nucleus, no irritation of its extremity can evoke reflex action; while, if it be due to spinal disease, the probability is, that irritation of the extremities of the anæsthetic nerves given off below the seat of disease will evoke muscular action in the corresponding muscles. Similar phenomena, but of a more complicated character and of a higher order seem not unfrequently to be produced through the agency of the nerves of sight and hearing (Bristowe, l. c., p. 931).

Sensibility admits of varying degrees, being different at different parts of the cutaneous surface, as proved by the now well-known experiments of Weber, whose mode of experimenting may be adopted as a practical means of diagnosis in paralysis of sensation.

The physical diagnosis of perversion of the sense of touch and power of contactile discrimination is based on the fact, that "if two points of a hard substance touch an unmoved cutaneous surface, they can only be perceived separately when the distance between them exceeds a certain limit." The minimum of distance thus established varies in different parts of the skin. A pair of compasses, the points of which are armed with suitable pieces of cork, may be used to find out the shortest distance at which the points are recognised as separate. A shorter distance than this will give rise to an indistinct impression of a long drawn point; and, finally, on approximating the points still more closely, the perception of the two points becomes completely the perception of one point only. Dr. Sieveking devised an instrument of this kind suitable for such physical diagnosis of contactile sensibility—the *Æsthesiometer* (Fig. 16). Applied

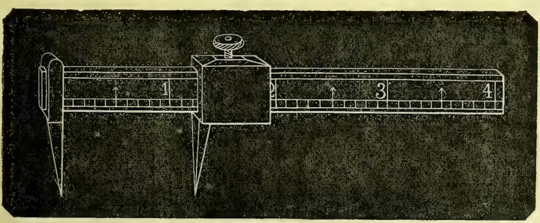

Fig. 16.[*]

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[*] Æsthesiometer of Dr. Sieveking.

in paralysis to ascertain the amount and extent of sensational impairment, it affords a means of diagnosis between actual *paralysis of sensation* and mere *subjective anæsthesia,* in which the tactile powers are unaltered; and of determining whether a case of impaired sensation is progressing for better or for worse (SIEVEKING, *Brit. and For. Med.-Chir. Review,* Vol. XXI., p. 280, 1858).

Instruments on a similar principle have been devised by Drs. Brown-Sèquard and John Ogle; but the best instrument is said to be that of Dr. Jaccoud, made by Colin and Robert, of Paris, about nine centimetres long, with a movable scale in the form of an arc of a circle, the whole folding up so as to be easily carried in the pocket. In the diagnosis of nervous diseases this instrument is as indispensable as is the stethoscope for the diagnosis of diseases of the chest. It not only measures the degree of tactile sensibility, and ascertains its perversion, but it aids in making out the precise site and nature of the disorder, particularly if the lesion be at the base of the brain, and in the spinal cord.

As it is necessary to know the normal distance limits of distinct sensation, Weber's results may be stated as a standard for comparison, premising that a delicate skin and an active mind admit of shorter distances than are here given (VALENTIN'S *Physiology,* p. 493):—

"(1.) The point of the tongue has the most delicate sense of touch. Here the minimum distance is ·0433 inch. The skin of the middle of the back gives a minimum distance of 2·13 to 2·68 inches, and is the region where touch is dullest. Hence the extremes may differ from fifty to sixty-fold. (2.) Assuming that the average for the tongue is equal to 1, the distance for the terminal phalanx of the index finger is 1·2, and for that of each of the remaining fingers 1·8. At the thumb-side of the first and second phalanges, it is 3·3; and on the dorsal surface of the last phalanx, 4·4. (3.) The red part of the lips gives 3·1, and the white 4·6. This difference is chiefly due to the unequal thickness of their coverings, and perhaps to their nervous relations. The remainder of the face has a still duller sense of touch. On the outer surface of the eyelids it is 7·9; on the skin of the cheeks, 9·4 to 10·9; and in the inferior frontal region 12·4. (4.) The tactile sensibility of the foot is in every respect inferior to that of the hand. For example, the volar side of the terminal phalanx of the thumb gives 1·5; and that of the great toe, 6·7. The dorsal surface of the hand gives from 4·4 to 14·4; and that of the foot, 12·2 to 25·9. (5.) The extremities of the limbs, such as the hand and foot, have a more delicate sense of touch than their middle segments, such as the forearm and leg; while these again are more sensible than the segments connected with the trunk, such as the thigh and upper arm. The two latter belong to those parts which do not possess a high development of tactile capacity. The vicinity of the elbow and the knee-joint is more sensitive, being easily excited to pain. (6.) The face has a more accurate sense of touch than the crown of the head or the neck. The dorsal surface of the trunk is inferior to the abdominal in this respect. (7.) The friction of some parts of the skin gives rise to peculiar feelings of tickling, or to voluptuous sensations. But such parts do not necessarily rank high in the scale of tactile sensibility. Thus the axilla gives 26·9, and the foreskin 10·6, as the minimum of distance. (8.) The tactile sensibility is capable of being increased by habit to an extraordinary degree.

In this way some blind persons are able to recognise different colours by inappreciable differences in their grain. The Bengalese spinning women can distinguish the threads of the cocoon with a tactile delicacy which is almost incredible. And persons devoid of arms may educate the sensibility of the toes, until it corresponds with that of the fingers of an ordinary individual. (9.) In judging of the delicacy of touch, we usually take the minimum distance at which two points can be recognised as the unit from which to start. This fact explains a peculiar illusion, to which attention was first drawn by Weber. When we draw the protected points of the compasses downwards from the cheek to the lips, it seems as if the distance between them gradually increased in consequence of our thus proceeding from a less sensible part to one which is more so."

The following are the rules laid down by Dr. Brown-Sèquard for the use of the *Æsthesiometer*:—

(1.) Fix the two points of the instrument at the distance which is normal to the part of the skin to be inquired into. (2.) Cover the points of the instrument by pieces of cork, as the slightest *prick producing pain* interferes with the perception of tactile sensation. (3.) The points must be applied simultaneously. If the patient feels only one point when both points touch the skin at the same time, the two points must be gradually separated from each other, and re-applied to the part until both points are felt. This arrangement will indicate the extent of anæsthesia. If, on the other hand, the two points are each distinctly felt at the normal distance limit, they are to be gradually brought nearer to each other till one point only is felt. This arrangement indicates the extent of hyperæsthesia. (4.) The patient ought not to be allowed *to see* whether one or both points of the instrument are applied, nor should he be made cognizant of the object of the experiment. (5.) The instrument should first be applied upon a healthy portion of skin, so as to ascertain how far he can discriminate the sensation of one or of two points. (6.) If the patient *knows* that both points are in contact he may *imagine* he feels both, no matter how short the distance may be between the points. In such cases one point only should be applied; and after he has asserted that he feels both, let him see that one only has been used, otherwise preconceived notions will destroy the value of the experiment. (7.) In anæsthesia of considerable extent, the two points of the instrument may be applied one after the other after an interval of forty or fifty seconds, and yet give the sensation of only one point: this is due to the slowness of the transmission of the impression; and the æsthesiometer in this way may mark the rapidity of the transmission of tactile impressions. (8.) When the degree of anæsthesia is great, the two points of the *æsthesiometer* are felt only as one, no matter how great the distance between them, provided that they are applied upon the same longitudinal line. Brown-Sèquard has known one point to be put upon the wrist and the other upon the forearm, and yet there was the sensation of one point only. (9.) Cutaneous hyperæsthesia may be of such an amount that, however close the two points may be, both continue to be felt. In a case of *chronic spinal meningitis,* tactile sensibility was so much heightened that the patient felt both points applied to the thigh at a distance of one millimetre apart; whilst, in health, there should have been a space of from five to six millimetres between them for both to have been distinctly felt. (10.) To avoid the mistake of finding either *anæsthesia*, or *hyperæsthesia*, when there is really neither, it should be borne in mind that *strychnia* increases sensibility, whilst *belladonna* lessens it. If, then, it is necessary to find out the exact state of the cutaneous sensibility of any one part of the body in patients using either of these drugs, the state of tactile sensibility in the healthy parts should be first explored, and the actual normal type of the individual under the influence of these drugs be ascertained. (11.) In certain cases when two points of an *æsthesiometer* are applied there is a distinct sensation of three points; or two points may be felt when one only is applied. In the instances in which this perversion of contactile discrimination has been met with, there has always been inflammation or congestion, sometimes the result of an intracranial tumor at the base of the brain, and especially in one of the cerebral peduncles, or in one of the lateral halves of the annular protuberance. The sites of this phenomenon are the face, neck, and the hand, but particularly the face; for in nine cases in which it was observed, in six it was limited to the face. When the distance between the two points of the instrument was two centimetres, two, or sometimes only one, point was felt; but at two and a half, or three, and even at four centimetres, there was a distinct sensation of three points touching the skin.

(2.) *Vertigo, Giddiness, or Dizziness.*—*Vertigo* is a sensation as if every thing were turning round one, or as if one were going to fall. *Giddiness* implies a confused sensation of swimming or of whirling in the head; or of unsteadiness of equilibrium, with a tendency to totter or to fall. *Dizziness* implies a more modified form of these conditions, with which are associated more or less hallucinations, consisting in a vivid representation of movement, either of the body of the patient, or of surrounding objects which are actually at rest. "Giddiness is objectively a motor symptom; and the 'sensation' it is attended by is a state of consciousness accompanying the outgoing current" (HUGHLINGS JACKSON) "Generally in man the tendency to movement is arrested by an effort of the will *per se*, or by grasping some object for support. The sensation of its production remains, and is projected outwards into the objects of the material world" (Dr. R. REYNOLDS). Things are said to go "round and round," or to oscillate before the eyes, so much so that either a sense of movement and an actual movement—subjective vertigo—or a motor tendency to turn and actual rotation or falling, are aspects of the same process, which varies from a sensation of disturbed equilibrium of the body such as to throw the sufferer to the ground; or, which may merely result in a sense of corresponding movement, or of mere instability. It is the motor tendency to turn which is felt as subjective vertigo. The chief part of the feeling of vertigo is that of movement in the individual or in external objects. It is the sensation of an "incipient motor process," is shown by the fact that the sensation in the slightest attacks of giddiness is always in harmony with the movements in the severe attacks. If the patient feel as if he were moving, it is *towards* the side to which he does move in the severe paroxysms. If the sensation be merely one of movement in external objects, it is always *from* the side towards which the patient falls—*i.e.*, it is the apparent movement of objects (say to the left) which would result from an actual movement of the head or eyes to the right. The practical significance of this is, that the character of the vertigo is an important aid in the diagnosis of its cause; and we are able to determine its character as accurately from such a subjective sensation as if we saw the patient reel. Hence the importance of ascertaining in every case of giddiness the precise character of the patient's sensation (Dr. W. R. GOWERS). A slighter chronic sensation of disturbed equilibrium may take the form of a vague sense of instability, which is expressed by the word "dizziness" or "giddiness"—a condition in frequent relation to the gastric functions. The least stomach disturbance at once excites the feeling.

The most common causes of giddiness are—labyrinthine or auditory diseases; diseases or disturbances of the brain, as in the slighter attacks of epilepsy; also in the oscillating movements of disseminated sclerosis, as described by Charcot; in paralysis agitans, and in injury to some parts of the brain, such as the crura cerebri, and tumors affecting the neighbouring parts. Affections of the cerebellum are attended with vertigo, as also some affections of the eye (such as those involving paralysis of one or more of the recti muscles), the ear, and spinal nervous system, and gastric disturbance or dyspepsia. It may also be referred to disturbance of the cerebral circulation; and in this connection may be associated with syncope, anæmia, and loss of blood on the one hand; and with cerebral congestion, or inflammation, on the other. It may also occur from an unhealthy condition of the blood, or of the presence in it of poisonous matter, such as alcohol, which, at a certain stage of intoxication, produces characteristic vertigo, so that even when lying down with the eyes shut "everything seems to be going round and round," or the bed on which the inebriate patient lies seems to float about and round the room, sometimes at one level and sometimes at another. Vertigo also accompanies the specific fevers, either at their commencement, or with the setting in of delirium. The recognition of the causes of vertigo must be based on the other symptoms with which it is associated; and in connection with this symptom, the following substantive disease is described from the writings of Drs. W. R. Gowers, Ferrier, and others:—

LABYRINTHINE VERTIGO, OR MENIÈRE'S DISEASE—*Syn.*, AUDITORY VERTIGO.

Definition.—*A condition characterised by sudden attacks of vertigo, paroxysmal in their occurrence, depending on a morbid state of the organ of hearing, and especially with lesions of the semicircular canals.*

Pathology.—The evidence on which the pathology of this disease is based is of three kinds:—(1.) The frequent association of paroxysmal vertigo with defect or disturbance of the function of the internal ear or auditory nerve. (2.) On some pathological facts which point to the existence in those cases of a morbid state of the semcircular canals. (3.) On the well-known experimental evidence of the connection between the functions of the semicircular canals and the maintenance of the equilibrium of the body, the theory of their function being that they give information of the position of the body to a co-ordinating centre which presides over the equilibrium of the body, and which experimental physiology has located in the cerebellum. The differentiation of this labyrinthine vertigo as a special or substantive affection may be cited as the result of physiological experiment in the elucidation of disease. "It was the remarkable experiments of Flourens on the semicircular canals of pigeons and rabbits, which led Menière, in the year 1861, to associate certain symptoms which he had frequent opportunities of observing (in the Deaf and Dumb Institution of which he was Director) with disease of the labyrinth in man, a view which was thoroughly justified by *post-mortem* examination." Flourens found that strange disturbances of equilibrium followed section of the membranous canals of the labyrinth, the character of the phenomena varying with the seat of lesion. These experiments have since been repeated by many investigators, and with various interpretations and explanations of the results. Some attribute the results to the irritation of mechanical injury, and therefore of the character of reflexly excited movements (BROWN-SEQUARD, VULPIAN). Goltz, on the other hand, regards the semicircular canals as the origin of impressions which regulate the equilibrium of the head, and with it of the whole body; and therefore, an explanation of the phenomena is to be sought for in the direction of loss or disharmony of certain impressions by which the functions of equilibration and co-ordination are regulated. It is thus "assumed that certain impressions on certain organs of sense are directly correlated and co-ordinated with certain adaptive and responsive actions—phenomena which may rise into consciousness, and which, under certain abnormal conditions, excite painful consciousness." It has been also shown that "for a so-called sensori-motor action the integrity of three factors is necessary, namely,—1. Certain afferent or sensory apparatus; 2. a co-ordinating centre; 3. efferent or motor tracts, which connect the centre with the muscular apparatus specially concerned in each action. A lesion or perversion of either of these three factors causes perturbation of the whole mechanism, which shows itself in reeling, staggering, or rotation" (FERRIER, *West Riding Lunatic Asylum Medical Reports*, Vol. V., p. 27). It is the disturbance of some one of these factors which constitutes the essential feature of Menière's disease.

"The sensory impressions on which equilibration depends are mainly of three kinds—viz., tactile, visual, and labyrinthine. On the harmony or consensus of these three normal equilibration depends, and the perversion or interference with either group causes perturbation of equilibrium, or induces a disharmony which manifests itself in consciousness as vertigo. When tactile impressions are abolished, as by section of cutaneous nerves, or by lesions of sensory tracts, equilibration becomes difficult or impossible. In some degree the absence of these may be compensated for by visual impressions; but when the visual impressions are also withdrawn, the faculty of equilibration is entirely annihilated. In the presence of tactile impressions visual are not so necessary, but visual impressions render equilibration more precise; and disturbances of visual impressions, such as by sudden lesions of the eye or abnormal positions of the eyeball, as result from paralysis of certain ocular muscles, cause manifest perturbations of equilibrium, and give rise to vertigo. Nothing, however, compensates for entire loss of labyrinthine impressions. Visual and tactile impressions may continue normal, but never replace those coming from the labyrinth; hence pigeons with their semicircular canals destroyed on both sides never recover the faculty of equilibration. It is, however, to be noted that the loss of labyrinthine impressions on one side only does not cause permanent affection of equilibration, the system, as it were, accustoming itself to rely on one set alone. This is a fact of no small importance in reference to the pathology of Menière's disease. The nerve which conveys labyrinthine impressions is not that concerned in the transmission of aerial vibrations. This was shown by Flourens, and has been confirmed by Goltz. These observers found that after destruction of the semicircular canals, animals continued to react even more acutely than before to sounds. Destruction of the cochlea causes deafness, but not disturbance of equilibrium. Hence the nerve supplying the labyrinth, though included in the auditory nerve, is not the nerve concerned in the reception or transmission of sound or aerial vibrations. The nerve which distributes itself to the ampullæ of the membranous canals is the vestibular division of the auditory nerve, which likewise supplies the utricle and saccule. This division of the auditory nerve is the path of transmission of the labyrinthine impressions concerned in the function of equilibration.

"The membranous canals are supplied with nerves only at their ampullary dilatations. These canals being filled with fluid, it is easy to see that variation in the position of the head will cause different degrees of tension or pressure on the respective ampullæ, and hence impressions may be generated in this manner on the ampullary nerves apart from any impression originating from without.

"If we suppose that the head is perfectly still, then the labyrinthine impressions are in a state of statical equilibrium. A movement of the head to the one side or the other will alter these conditions. A sudden deviation of the head will cause simultaneously a plus and minus variation in opposite ampullæ, variations which call forth adapted movements of equilibration. Hence we can understand that the balance will be overthrown if one of the forces is withdrawn or abnormally altered. The perturbation of equilibrium will be in the direction of the unopposed or predominant force" (FERRIER, l. c.)

Contrasting the conditions of negation and irritation of the semicircular canals, Dr. Ferrier, from the researches of Flourens, writes that it would seem the negation of the horizontal canal on one side causes the body to be thrown or spun towards the same side, and that negation of the superior canals causes the body to be thrown forwards, and a like negation of the posterior canals causes the body to be thrown backwards. We should, therefore, expect that if the lesion were an irritative one the phenomena would be reversed; and that, therefore, irritation of the left horizontal canal should cause a feeling of rotation to the right side, and *vice versa* in the case of irritation of the right; and similarly, a feeling of being hurled forwards or backwards, according as the irritation affected the posterior or superior canals respectively.

Next, in regard to the feeling of giddiness or vertigo: disturbances of equilibrium do not necessarily create a feeling of vertigo; and (as Hughlings Jackson justly remarks) it is erroneous to say that the feeling of vertigo is the cause of the reeling. Vertigo is rather the subjective side of perturbed or incongruent sensori-motor co-ordination. In Menière's disease it is chiefly the incongruence between labyrinthine and visual impressions which causes the feeling of giddiness. When the body is rotated round on a revolving disc, there is no feeling of giddiness so long as the eyes are shut, as Crum-Brown has shown. If the eyes be opened, however, either during the primary rotation or during the sense of complementary rotation after the disc has ceased to revolve, a feeling of giddiness at once comes on. External objects seem to be revolving also, and this causes a feeling of insecurity or giddiness; and giddiness is also felt if external objects turn round, though the individual does not. Labyrinthine impressions are correlated with ocular movements in the co-ordinating centres, and Flourens' experiments show that the oscillation of the eyeballs occurred when the semicircular canals were injured. This is a fact of considerable importance in reference to the mechanism of central co-ordination, and the condition of the eyeballs in attacks of Menière's disease would be well worthy of study (Dr. H. SUTHERLAND, *Medical Record*, Feb. 15, 1876, p. 69).

We have thus apparently within ourselves a complex self-adjusting mechanism whereby, when the body is at rest, a statical equilibrium is maintained; and in which, by movements of the head and body, impressions are generated by respective variations in tension in opposed ampullæ, which serve not only to indicate the direction of movements, but by the symmetrical plus and minus variations excite the appropriate bodily movements for maintaining the balance, each inclination in one direction being the stimulus to compensatory action in the other (FERRIER, l. c.)

But it is not necessary (as Hughlings Jackson observes) that there should be direct lesion of the semicircular canals in order to produce vertigo and reeling; for indirect affections of the membranous labyrinth, such as result from pressure on the vestibule by foreign bodies, or by variations of pressure on the membranous labyrinth, such as may be produced by a simple injection of water into the external ear, as for the dislodgement of cerumen, especially in cases where there is a loss of substance of the tympanum, or where it is ruptured (Dr. WEBER LIEL); or by the products of inflammatory affections of the meatus—each and all of these conditions may cause vertigo and reeling. These are all *irritative* lesions, as contradistinguished from *destructive* lesions; and it is of great importance to note the *direction* in which the patient reels in such cases. Dr. Ferrier is of opinion that it will always be towards the side opposite the merely irritative lesion.

Symptoms.—Those most characteristic are:—"That the patient is suddenly seized with vertigo, and a feeling of nausea or positive sickness, with great constitutional depression and faintness. Usually the giddiness comes on simultaneously with ringing or buzzing in one or, it may be, both ears; and generally in the ear principally affected more or less complete deafness is found to exist, which ultimately becomes absolute." Charcot calls special attention to the fact that the vertiginous symptoms and the buzzing in the ear go hand-in-hand; and, that the two disappear together—a condition generally coinciding with absolute deafness—indicating that not merely the canals, but also the cochlea have been invaded by the lesion, which now becomes negative rather than positive in its character.

The sense of rotation or actual rotation to the right side is caused by irritation of the left horizontal ampulla. Rotation forwards round a horizontal axis coincides with irritation of the posterior canal; and rotation backwards coincides with irritation of the superior canal. Conversely, irritation of these different structures (such as is supposed to be the case in Menière's disease) causes the feeling of rotation to the right, backwards or forwards, according as the lesion affects the left horizontal, posterior, or superior canal respectively (Fig. 17).

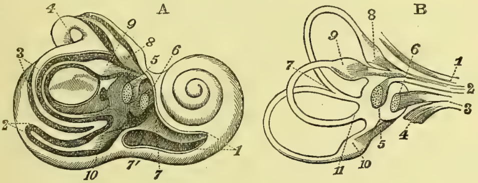

Fig 17.*

"In the normal condition the balance is maintained by the fact that any irritation which causes a tendency to rotation in one direction excites the antagonistic muscles of the body to compensatory action. Hence, for example, the tendency to backward rotation caused by irritation of the ampullæ of the superior canals, excites reflexly the muscles which tend to pull the body forwards, and so the balance is recovered. In abnormal irritation of one set, however, the compensatory action is insufficient, and so the balance is overthrown. The left superior and right inferior antagonise the right superior and left inferior; and the left horizontal antagonises the corresponding canal on the right. Now, if one set is removed, the tendency will be to fall in the direction of the action of the unopposed canals. Hence, removal of the posterior canals will cause a tendency to fall backwards in the direction of the influence excited by the unopposed superior vertical canals. This tendency may, however, be antagonised (inhibited) by voluntary effort in the absence of the reflex mechanism, and so the individual may be able to maintain a condition of stationary equilibrium. This stationary equilibrium, however, is overthrown by any disturbance which draws away the attention, or when the individual attempts other voluntary efforts. This is exactly what is seen in Flourens' experiments—the disorder of equilibrium being always most marked when the animal was disturbed or attempted to move from its place" (FERRIER, l. c., p. 35).

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* The Interior of the Right Labyrinth with its Membranous Canals and Nerves (Breschet). A. The outer wall of the bony labyrinth is removed so as to display the membranous parts within. 1. Commencement of the spiral tube of the cochlea. 2. Posterior semicircular canal, partly opened. 3. External or horizontal canal. 4. Superior canal. 5. Utriculus. 6. Sacculus. 7. Lamina spiralis. 7′. Scali tympani. 8. Ampulla of the superior membranous canal. 9. Ampulla of the horizontal. 10. Ampulla of the posterior semicircular canal.—B. Membranous labyrinth and nerves detached. 1. Facial nerve in the internal auditory meatus. 2. Anterior division of the auditory nerve giving branches to 5, 8, and 9, the utricle and the ampullæ of the superior and horizontal canals. 3. Posterior division of the auditory nerve, giving branches to the sacculus (6), and posterior ampulla (10), and cochlea (4). 7. United part of the superior and posterior canals. 11. Posterior extremity of the horizontal canal.

The next most prominent symptom is the nausea or sickness, with its accompanying systemic depression. This is in accordance with the remarkable relation which subsists between vertigo and visceral disturbances, a connectionship which maybe based (HUGHLINGS JACKSON) on the intimate relation between the auditory and vagus nuclei in the medulla oblongata, as demonstrated by Lockhart Clarke. Ferrier is in favour of the existence of a still higher co-ordination of visceral with auditory and other impressions than that of the medullary nuclei. He is in favour of the supposition "that visceral impressions form one of the factors in that consensus which regulate equilibration and co-ordination; and, that therefore their afferent nerves are co-ordinated with the tactile, optic, and labyrinthine nerves in the encephalic centres of equilibration and co-ordination." In certain visceral disturbances we have a distinct form of vertigo—*vertigo a stomacho læso* of Trousseau. But visceral disturbances, whether causing vertigo or not, usually excite nausea and vomiting; and so conversely, disturbances of the mechanism of equilibration, either from central disease or from propagation of irritation affecting the semicircular canals, may manifest themselves as reeling or staggering on the motor side; and, as vomiting or nausea on the visceral side.

The last symptom to be noted is the local condition of the ear as regards hearing. The deafness is shown to be of labyrinthine origin by the insensibility both to the aërial and skull vibrations of the tuning-fork. In the case recorded by Dr. Ferrier there was, however, a peculiarity in this respect, that "the patient retained sensitiveness to the aërial vibrations of the tuning-fork, even more acutely than before on the left side than on right." (The disease was in the left ear.) Not only so, but the power of discrimination of musical sounds seemed sharper on the affected ear than on the other. The patient himself was of opinion that the left (the affected) ear was the more acute. On the other hand, there was absolute insensibility on the left side to the vibrations of the tuning-fork placed on the bridge of the nose or on any part of the skull. In one or two other cases in Dr. Ferrier's experience, there was complete insensibility, both to aërial and skull vibrations, on the affected side. He considers his present experience (if confirmed) justifies the conclusion "that the semicircular canals are, as regards hearing, specially concerned in the reception and transmission of skull vibrations in contradistinction to aërial vibration."

The derangement of hearing with which labyrinthine vertigo is associated may be of two kinds. "Sometimes there is evidence of undue excitation of the auditory nerve, noises in the ears, permanent or only at the moment of the paroxysm. More frequently, indeed almost universally, there is evidence of defective sensitiveness. This defect may be conspicuous or obscure; may range from a considerable degree of deafness to a slight defect in audition, which it requires much care to ascertain. The knowledge that the defect may be limited to the perception of the sound conducted through the bones of the skull, is an important addition to our means of diagnosis. The fact was pointed out by the late Mr. Hinton (*Guy's Hosp. Reports*, 1873), and by Mr. Dalby (*Lectures on Diseases of the Ear*, 1873), and cases in which it was noted have been published by Dr. Ferrier, Dr. Duffin, and others. This loss of what may conveniently be termed *perosseal audition* is regarded as evidence of an affection of the labyrinth or of the auditory nerve, and may be absolute; a tuning-fork vibrating in contact with any part of the head or with the teeth may be unheard in the affected ear, while it is heard readily if held opposite the meatus; or the loss may be partial, and in that case it may be detected by the method, commonly employed by aural surgeons, of closing the ears while the tuning-fork is sounding in contact with the vertex or some other part of the skull. Closure of the meatus, if the latter be unobstructed, increases the intensity of the sound, if the perception of sounds conducted through the skull be unimpaired,—renders it weaker or inaudible, if there be impairment. But the method is one that requires much care and repeated examination to detect a slight defect in an unintelligent patient. It is important, however, to examine these cases, not only with a tuning-fork, but with a watch. There may exist marked impairment of perosseal audition to the watch, while the tuning-fork is well heard" * (Dr. W. R. Gowers, *Brit. Med. Journal*, March 10, 1877, p. 388).

In a case recorded by Dr. Gowers, the liability to the paroxysms of vertigo seemed to correspond to the periods of imperfect audition; and when tested after an attack, the power of hearing the watch has been always absent. Even a slight degree of impairment of the power of hearing a watch in contact with the skull may be of significance, as when the tuning-fork fails to reveal any abnormality in the function of hearing. A "confusion of sounds," apparently in the head, with headache in the occipital region and behind the ears, may be at first all the phenomena, as in a second case related by Dr. Gowers. But eventually severe attacks of giddiness, with great "confusion of sounds in the left ear," and with the occurrence of quasi-convulsive movements, left no doubt as to the nature of the case.

Diagnosis.—(*a.*) *From Gastric Disorder.*—The vomiting which accompanies the disturbance of equilibrium, and which is to be attributed to the association with the function of the pneumogastric, is the source of much misconception as to the nature of the cases of Menière's disease. A few years ago had the question been asked, what are the commonest causes of paroxysmal vertigo? the invariable answer would have been, "A disordered stomach or a diseased brain." It is probable that, even now, the exceptions to such an answer would be comparatively rare. The answer, at any rate, indicates the conditions from which the diagnosis of auditory-nerve vertigo has to be made (Gowers). "The gastric associations are especially liable to mislead. The occurrence of vomiting in the absence of any other obtrusive cause of the vertigo, is held as proof of a causal derangement of the stomach. Sometimes an attack of vertigo may not cause vomiting; it may merely disturb the gastric functions, just as the motion of a ship does with some persons in whom it does not cause sea-sickness. The dyspepsia which results is regarded as a sufficient cause for the giddiness. But, further, in persons the subjects of auditory vertigo, whose equilibrium nerves and centre are deranged, a primary gastric disturbance seems sometimes to excite a paroxysm of the special vertigo to which they are liable; and the gastric disturbance which thus causes the attack is naturally regarded as its only antecedent." In some cases, as reported by Dr. Gowers, there is a combination of chronic gastric symptoms with auditory-nerve vertigo, which may cause great confusion in the diagnosis. If the gastric symptoms are due to organic disease of the stomach, the diagnosis becomes more easy. The sufferers from this affection, besides their acute attacks, are often liable to a slighter chronic sensation of disturbed equilibrium, the "interparoxysmal reeling" to which Dr. Hughlings Jackson has called attention. It may take the form merely of a vague sense of instability, which is expressed by the word "dizziness." This condition is often in constant relation to the gastric functions. The least stomach-disturbance at once excites the feeling, and most careful dietetic management is necessary, in order to keep the patient free from his annoying sensations. Dr. Gowers relates such a case.

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* The method of testing perosseal audition with the watch which Dr. Gowers has found most useful, is to hold the watch first close to, and then in firm contact with, the zygoma, the parietal eminence, and the mastoid process—the direct passage of the vibrations to the meatus being prevented by placing the hand as a screen between the watch and the ear in the two former positions, and in the latter by drawing forward the pinna. The effect of contact in the normal state is very marked. The watch not in contact is scarcely heard or inaudible, while in contact it is loud. This method of testing is somewhat coarse, but very convenient. The effect of closure of the meatus on the sound of a watch is slighter than on the sound of a tuning-fork; and although in health it is distinct, it is not sufficient to render this test a convenient one.

"The most important point in the diagnosis of auditory from gastric vertigo is the recognition of an impairment of the function of the auditory nerve, shown, first, in deafness, and, secondly, in tinnitus; and the most significant change is the loss of the power of hearing a watch in contact with the skull. The point of next importance is the character of the vertigo. The sensation which results from a primary gastric disturbance is usually *vague*—a confused sense of defective equilibrium; that which results from a labyrinthine affection is *definite—a sense of movement in a certain direction subjective, or referred to other objects.* This statement is not in complete accordance with the descriptions of gastric vertigo which have been given by Trousseau and other writers, who have described vertigo of very definite character as the consequence of gastric disturbance only. But the fact that the patient may be unconscious of a most significant auditory defect, lessens the value of former observations as evidence of the definite character of stomachal vertigo. My own conviction is that, in the vast majority of the cases in which a vertigo of definite and uniform character is apparently excited by gastric disturbance, an auditory defect will be discovered on careful examination. In a small proportion of cases no auditory defect may be found. In some of these cases, there may be disturbance of some other sensory nerve also related to the function of equilibration. But, on the other hand, the sensation which attends a labyrinthine disturbance may be vague in character. Hence, *definiteness in the character of the sensation is of greater value as suggesting a primary affection of the auditory nerve or the* *centre of equilibration, than is the absence of definiteness as excluding such a source of the giddiness.* Lastly, the sequence of the gastric symptoms and vertiginous sensations is of some significance, although this may easily be misunderstood. Vomiting, dyspepsia, or diarrhœa, after an attack of vertigo, is no evidence that dyspepsia is the cause of the trouble. Gastric derangement before the vertigo may be a primary cause of the vertigo, or may merely excite an attack in the presence of a derangement of the equilibrium centre" (GOWERS, l. c.)

(*b.*) *From Cerebral Disease.*—"Vertigo may be a symptom of cerebral disease, and the attacks which result from a labyrinthine affection may be of such a character as to simulate very closely those of cerebral origin. This resemblance depends chiefly on the occasional occurrence of loss of consciousness in the attacks of auditory vertigo, and on their suddenness. Charcot states that in about three-fourths of the cases of disseminated multilocular sclerosis, vertigo is one of the phenomena which mark the invasion of the nerve centres; and the vertigo is generally of the gyratory kind. All objects seem to be whirling round with great rapidity, and the individual himself feels as if revolving on his axis. Threatened with loss of equilibrium, he lays hold of the nearest object he can grasp. In most cases the giddiness returns in fits of short duration. The gait of such a patient is not to be confounded with the uncertainty of gait connected with diplopia, which ceases when the patient keeps one of his eyes closed ('Diseases of Nervous System,' *Syd. Soc.*, 1877, p. 194). Sudden loss of consciousness may be a symptom of two forms of cerebral disease—apoplexy and epilepsy, and from each of these auditory-nerve vertigo has to be distinguished."

(*c.*) *From Apoplexy.*—"The resemblance to apoplexy struck Menière very forcibly, and the affection was accordingly termed by him 'apoplectiform.' Instances are occasionally met with which merit such a designation. But the cases are rare in which the loss of consciousness is so considerable and so lasting as to render the diagnosis a matter of real difficulty. A speedy return of consciousness, coupled with the persistence of vertigo and the absence of any symptoms of local weakness, generally renders the nature of a case sufficiently obvious. The history of preceding attacks will also indicate the nature of the affection. In a first attack, as in one depending on an acute inflammation of the semicircular canals, a diagnosis might be for a time impossible."

(*d.*) *From Epilepsy.*—"The attacks of auditory-nerve vertigo resemble those of epilepsy in several particulars, a resemblance of much significance. A sensation of vertigo often attends the slighter attacks of epilepsy. In these, however, consciousness is usually, but not always, lost, while consciousness is usually preserved during the paroxysms of auditory-nerve vertigo. Severe attacks of the latter may be accompanied by sudden, transient loss of consciousness. The sufferer may even fall, and be unaware that he has fallen. The common features of the two conditions are instructive. No doubt the significance of the feeling of vertigo is in each state the same. In each it depends on an 'incipient motor process' (HUGHLINGS JACKSON). In each case, also, there may be a developed motor process. The violence of the movement, in severe attacks of auditory-nerve vertigo, is remarkable. It is no mere compensatory action to maintain a disturbed equilibrium; the patient is dashed to the ground with force. Such movement must result from the 'discharge' of a motor centre, quite analogous to the discharge which occurs in an epileptic fit. If we locate the centre for equilibration in the cerebellum, we may perhaps regard Menière's disease as a cerebellar epilepsy, reflex in character, determined by an irritation of the semicircular canals, alone or in conjunction with impressions from some other nerves with which the centre is in connection, as those of the pneumogastric nerve. On this view, it is not difficult to understand the loss of consciousness which may accompany the severer seizures, since the connections of the cerebrum and cerebellum are intimate; fibres from almost all parts of the hemispheres being gathered into the superior cerebellar peduncles."

Epilepsy may influence the occurrence of labyrinthine vertigo, and Dr. Gowers relates two cases in each of which there was the co-existence of epilepsy and auditory-nerve vertigo; but much care and hesitation are demanded in diagnosing auditory-nerve vertigo in the presence of epilepsy. The diagnosis from epilepsy must turn, as in the case of gastric disturbance, to some extent on the detection of defective audition, and especially on defect in the power of hearing vibrations conducted through the skull. The definite character of the vertigo is in these cases no criterion. A more important symptom is its persistence between the paroxysms, and its long duration in the paroxysms themselves. Its relation to movement is also important. If it be related to change of posture, and can be produced by certain sudden movements of the head, it is probably due to auditory-nerve disturbance. In the latter, the attacks are succeeded by vomiting much more frequently than are the attacks of *petit mal.* Lastly, there is rarely the objective evidence of momentary loss of consciousness, which is so frequent in minor epilepsy, even in the slightest attack; in auditory-nerve vertigo consciousness is only lost or obscured in severe attacks, in which the giddiness persists for a considerable time and vomiting is frequent (*Brit. Med. Journal,* April 21, 1877, p. 477).

Treatment.—Of practical importance in the treatment of labyrinthine vertigo, and at the same time in illustration of its pathology, it is to be remembered that the tympanum is supplied with arterial blood from the carotid, and its veins discharge into the jugular; that the labyrinth is supplied by the vertebral, and its veins discharge into the superior petrorsal sinus; that the vertebral artery derives many of its vaso-motor nerves from the inferior cervical ganglion; and that these nerves communicate with the brachial plexus. The same ganglion furnishes the inferior cardiac nerve—the principal inhibitory nerve of the heart. Besides these connections a fasciculus is given off from the pneumogastric, near the recurrent laryngeal, to the lower cervical ganglion. We have, therefore, in this ganglion an organ connecting the upper extremities, heart and upper portion of the digestive tract, with the labyrinthine circulation. From any irritation there may consequently arise a diminished inhibition, which will relax the walls of the vertebral artery, and so cause pressure on the endolymph with consequent vertigo (WOAKES, *Chicago Med. Journ. and Exam.,* Nov. 1878).

Hence such vertigo does not necessarily imply previous disease of the ear. Gunshot wounds of the upper extremities are often followed by dizziness (WEIR MITCHELL). *Quinine,* in large doses, and *tobacco* diminish vascular tonus; while *bromine,* the *bromides,* and especially *hydrobromic acid,* stimulate vascular innervation. Hence the antagonistic effect of the latter on the *tinnitus aurium* caused by the former (quoted by Mr. D'ARCY POWER, *London Medical Record,* April 15, 1879, p. 152).

The treatment of labyrinthine vertigo, the result of *destructive* lesions, can be but palliative; but auditory-nerve vertigo, due to *irritative* lesion, may be more or less relieved. Absolute rest is the first essential. The direct treatment of labyrinthine affections is a subject for the special aural surgeon; and it is obvious that when symptoms point to the existence of any disease or irritation in the accessible parts of the ear, special treatment should be directed to those morbid states. When there is evidence of an irritative process, blistering behind the ear sometimes affords very marked relief to the vertigo. Occasionally, there may be evidence of a constitutional condition on which the aural disturbance is dependent. In some cases there may be reason to believe that a gouty change in the membranous labyrinth is the cause of the morbid action (HINTON, GOWERS). Marked relief is afforded under these circumstances by *colchicum* and *potash.* In rare cases the vertigo may depend on a syphilitic inflammation of the labyrinth. Such a case has been described by Moos of Heidelberg. One of the most severe forms of paroxysmal vertigo which Dr. Gowers has seen was in a child, with almost complete deafness and every sign of inherited syphilis. In all syphilitic cases the specific treatment for syphilis is, of course, required.

It has been remarked that certain drugs have a marked influence on the organ or nerve of hearing. The effect of *quinine* induced Charcot to employ it, in full doses, in a case of auditory-nerve vertigo, with some beneficial result. Dr. Gowers has tried it; but he has not found that any marked effect was produced on the vertigo. The influence of *salicylate of soda* upon the equilibrium suggested its use in this disease. It does not remove the giddiness; but in some cases it lessens its intensity, in doses of from five to ten grains three times a day. The most unremitting care in diet and regimen is necessary. Antacids have been found useful in subduing stomachal disturbance, and so arresting an actual attack, and *carbonate of bismuth* has been of use. The *bromides of potassium* and of *ammonium* have been the most beneficial of all medicines in subduing the paroxysms; and the good effects of *bromide of potassium* are increased by *belladonna,* and also by its combination with bark. Other sedatives alone, such as *opium, Indian hemp, gelseminum,* and *hyoscyamus,* are, in the experience of Dr Gowers, without effect. The affection is a very obstinate one; but when the several factors in the individual cases are sought out and corrected, a considerable amount of relief may be afforded (Dr. W. R. GOWERS, l. c.)

(3.) *Convulsions, Spasms, and Eclampsia.*—*Convulsions* are characterised by the occurrence of excessive and more or less extensive muscular movements, taking place independently of the will, the nerve force being transmitted in intermitted paroxysms of longer or shorter duration. They vary in duration and intensity, and are exemplified in the mere flickering of muscular fibres, the tremulousness of cold and of febrile rigors, the subsultus tendinum of the typhoid state, the tremors of paralysis agitans, and those of chronic mercurial poisoning. They thus affect single muscles, or portions of muscles, or groups of muscles—those of the head, neck, trunk, or limbs, or the whole body generally; and their violence may be such as to rupture muscles during their uncontrollable contractions, as in the cramps of cholera. *Spasms* are *involuntary* convulsive actions of less extent, of which there are several varieties:—(*a.*) *Clonic spasms,* in which the contractions are intermittent, in rapidly alternating contractions and relaxations, as in *subsultus tendinum;* (*b.*) *tonic spasms,* or *spastic contractions,* which are continuous, the paroxysms having a lengthened or certain appreciable duration, as in *common cramps,* which occur after fatigue in the muscles of the calf of the leg; also in various muscles in Asiatic cholera and in tetanus, and in strychnia poisoning; in the contractions of the limbs following some forms of paralysis; the turning in of the thumb upon the palm, and the gradual flexing of the arm, or other slow, but strong contractions of muscles, which usher in the convulsive phenomena of the true epileptic fit. They are in some cases, as in tetanus and strychnia, attended with extreme rigidity and hardness of the affected muscles.

Convulsions and *spasms* affect those muscles which are animated by the cerebro-spinal nerves; but there are also *spasms* ascribed to a morbid excess of motion in the unstriped contractile fibres which are animated by ganglionic or sympathetic nerves; for example, spasms of the stomach or heart. Rigor, tremor, spasms, convulsions are all varieties of motor phenomena; but closely allied to inco-ordination, and through this to paralysis.

Eclampsia, Epileptoid or Epileptiform Attacks.—Eclampsia is a name now commonly applied to all those varieties of convulsions which resemble the epileptic convulsions in the sudden loss of perception and voluntary power, with more or less generally distributed spasmodic movements, which are *quasi-tonic* at first, then *clonic,* and appear to impede the function of respiration. Such attacks may last from ten to twenty minutes, followed by some exhaustion and sleep (Reynolds). The proximate cause of the different convulsive movements in epilepsies are caused by irritation or "discharging lesions" of the grey matter of the hemispheres in the region of the *corpus striatum* (Drs. Ferrier and Hughlings Jackson). The affection may be limited, in animals, to one muscle or group of muscles, or may be made to involve all the muscles represented in the cerebral hemispheres, with foaming at the mouth, biting of the tongue, and loss of consciousness. When induced artificially in animals, the affection, as a rule, first invades the muscles most in voluntary use. Dr. Bristowe also observes, "that all those regions which, by their destruction, cause paralysis of certain parts, are necessarily likely, under irritation, to cause motor phenomena in the same parts."

The causes of the convulsions of eclampsia are of more or less accidental occurrence, and are to be sought for either—(1.) in morbid conditions directly affecting the brain, such as injury or disease affecting the grey matter, some specific lesion of the skull, effusion of blood into the brain, obstruction of a cerebral artery, the growth of an intracranial tumor (tubercular, syphilitic, carcinomatous, hydated, or aneurismal); or, (2.) they may occur in connection with some special pathological or physiological process, as from sudden and copious loss of blood, or when the brain is deeply congested, or when certain vegetable and mineral poisons circulate in the blood, as hydrocyanic acid, strychnia, brucia, picrotoxia, lead, absinth; or the retention and circulation of effete matters in the blood from renal disease, or from hyperpyrexial blood. They may occur as the early indication of scarlatina or other infectious disorders in children, in whom they often take the place of the usual rigors, as in the course of the General Diseases of Sections A. and B. Or, (3.) to causes which are reflex or sympathetic, such as digestive derangement, irritation from intestinal worms, dentition, passage of renal or biliary calculi, painful injuries of the skin, such as burns, blisters, sinapisms. Reflex movements, or twitchings, also occur in sequence to the sudden darts of intense pains which attend neuralgias. "If the toe be attacked, the leg is momentarily drawn up by an uncontrollable impulse; if the finger, the arm; if the face, as in ordinary *tic douloureux*, spasmodic twitching of the muscles of the painful region occur. These convulsive movements may vary from mere twitchings of the muscles to spasmodic contractions of considerable force" (BRISTOWE, l. c., 1122).

Fits of eclampsia have much more frequently a fatal issue (either from coma or exhaustion) than any other form of convulsions; and they are much more irregular in their occurrence. If the affection on which they depend is a progressive one, the fits may become more frequent and severe as the disease advances; or they may cease if the cause of the occurrence is removed.

Another form of convulsion is the *choreic*, as seen in *chorea*, which is of a similar nature to the *epileptiform*, or *eclampsia* dependent on momentary and successive discharging lesions of the individual cerebral centres. *Nystagmus*, or oscillation of the eyeballs, is an *epileptiform* affection of the cerebellar occipito-motorial centres. The movements cease during sleep; and are greatly aggravated during voluntary efforts, and under mental excitement. They are characterised by abruptness and irregularity; and are apt to interrupt the progress of any voluntary movement by a series of grotesque contortions, not necessarily limited to the limb which is engaged in making the effort.

The clonic convulsions of epilepsy and eclampsia "are characterised by more or less violent and rapidly repeated alternating movements, by rapidly repeated alternate flexions and extensions of the arms or legs, or jerking of the head and neck, or similar movements of the muscles of the face or of the eyes" (BRISTOWE, l. c., p. 932).

The influence of the emotions as a cause of convulsions by irritation, or in "discharging lesions," must also be acknowledged, such as fright, terror, prolonged grief and anxiety, a sudden impression on the imagination or memory.

Eclampsia, or convulsions, in connection with lesion of the cortex consist sometimes of a limited number of localised muscular spasms; at other times they have all the characters of what is usually called epilepsy. They generally commence in an isolated group of muscles (unilateral), and propagate themselves to the other muscles of the same limb, or to the whole body, before consciousness is lost, which is not a constant phenomenon—the individual can often give a complete account of the course of the attack. Such convulsions were observed by the older physicians; but only lately have their true nature and pathology been elucidated. Odier, Lallemand, and Abercrombie have recorded such cases. Serres, in 1824–26, relates the case of a young man who, after suffering from discharge from the right ear for eighteen months, began to suffer from convulsions limited to the left arm. A *post-mortem* examination showed a portion of the brain lying on the petrous portion of the temporal bone in a state of putrid abscess. He quotes a similar case from Sandifort, and concludes, "that one may, from careful comparison of instances, indicate the seat of lesion in the brain according to the limb which is affected by paralysis or convulsions. Thus, if the right arm be affected, the lesion will be in that portion of the brain which corresponds to the upper left parietal bone, and *vice versâ*" (*Anatomie comparée du Cerveau*, quoted by CHARCOT and FERRIER in *Medical Record*, June 15, 1877). In 1827, Bravais described, under the name of "*Epilepsie hémiplegique*," a variety of epilepsy commencing in one limb, or on one side of the face, or on one side of the body, giving exact descriptions of the phenomena; but he completely neglected the anatomico-pathological aspect of those cases (FERRIER, in *Med. Record*, June 15, 1877). A case related by Dr. Alexander Robertson, of Glasgow, also supports the theory; as do also those cases of percussion of the skull already referred to. Dr. Robertson also writes, with regard to the morbid action in epilepsy being spoken of as one of "excessive discharge," that "some years ago (previous to 1879) in certain cases of epileptic convulsions affecting chiefly one side of the body, I noticed that the side most convulsed was afterwards palsied for an interval varying from a few minutes to some hours. The explanation that commended itself most to my mind was one that had been previously proposed by the late Dr. R. B. Todd—namely, that 'excessive discharge' of the part of the brain most affected was followed by its temporary exhaustion. Dr. Hughlings Jackson, without being aware of these observations, explained the condition in epilepsy by the same hypothesis, but gave it the name of 'discharging lesion.' Finding, however, that he had thus been anticipated, with scrupulous regard for the rights of others, Dr. Jackson at once acknowledged the fact; and further, did me the honour of associating my name with that of Dr. Todd in relation to the hypothesis. It will be clear, however, that all the merit of priority is due to Dr. Todd" (*Glasgow Med. Journ.*, Vol. XII., Dec., 1879, p. 417). Nevertheless "it is to Dr. Hughlings Jackson that we owe the first exact investigation on the relation of partial epilepsy to cortical lesions" (CHARCOT, FERRIER). He has shown that convulsions of an unilateral kind and eclampsia generally may be looked upon as experiments upon the brain made by disease, showing us, in a rough way, the localisation of special classes of movements in the cerebral hemispheres. He has especially urged regarding them,—"(1.) That convulsions, choreal movements, affections of speech, and other motor symptoms are not *only* to be thought of as 'symptoms of disease,' but can be considered also as results of experiments made by disease, revealing in the rough the functions of cerebral convolutions; (2.) that the study of such *motor* symptoms is of direct importance for *mental* physiology; (3.) that if we do our work anatomically and physiologically, and reduce these very *different* symptoms to their lowest terms—to movements, for example—we shall find that there are certain *fundamental* principles common to them all." Hence it is that cases, alike of paralysis and of convulsions, may be looked upon as the results of *experiments made by disease* on particular parts of the nervous system of man. The study of palsies and convulsions, from this point of view, is the study of the effects of "destroying lesions," and of the effects of "discharging lesions" (*i.e.*, the effects of irritation, which excites, but does not destroy the part). "For an exact knowledge of the particular movements most represented in particular centres, we must observe and *compare the effects of each kind of lesion.* It is just what the physiologist does in experimenting on animals: to ascertain the exact distribution of a nerve, he destroys it and also stimulates it. Indeed, *this double kind of study is essential* in the investigation of cases of nervous disease for *physiological purposes.* For, *limited destroying lesions of some parts of the cerebral hemispheres produce no obvious symptoms; while discharging lesions of those parts produce very striking symptoms.* By this *double method* we shall not only discover the particular parts of the nervous system where certain groups of movements are most represented (anatomical localisation); but, what is of equal importance, we shall also learn the order of action (physiological localisation) in which these movements are represented" (*Lancet*, Jan. 18, 1873; *Brit. Med. Journ.*, May 10; and *West Riding Asylum Reports*, Vol. III., p. 176, 1873). In all cases of convulsions the attempt ought to be made to find the seat of the "discharging lesion," and also the pathological process which has led to it; to ask ourselves—"Where is the lesion permitting the occasional excessive discharge? This method is *just the opposite* of the common method. It is immaterial whether the case of convulsions is to be called epileptic or not; but we ought to try to find out in what *part* of the brain is the function disturbed or irritated of which the convulsion is the sudden development. We *do not care* to say that a tumor of the brain (or minute changes near it) had 'caused epilepsy;' but that changes in a particular region of the nervous system, say in the region of the left middle cerebral artery, led to convulsions, in which the spasm began in the right hand, spread to the arm, attacked next the face, then the leg," &c. This method of Dr. Hughlings Jackson has the advantage of showing the relations of different "diseases" or symptoms—a matter of great importance when symptoms are to be looked upon as the results of the experiments of disease. As "examples of the relations of different symptoms, there are several *mobile* counterparts of hemiplegia. There is hemi-chorea, there are certain cases of hemi-spasm, and there is what I may call hemi-contraction, a mixture of palsy and spasm. I call these one-sided mobile symptoms of hemi-kinesis. I believe that each of them depends on *disease* of the same *internal* region as does hemiplegia—the region of the corpus striatum, for the same *external* region is affected in each. To obtain a knowledge of the movements represented in the cerebral district mentioned, we have to study each one of these symptoms carefully." As evidence of the practical usefulness of this method of study, Dr. Radcliffe has written (after stating reasons for localising the changes producing chorea in convolutions near to the corpus striatum),—"For most assuredly the difficulties which beset any attempt to localise the choreic lesion in the nerve centres, are not a little simplified by thus insisting upon the clinical relations between hemi-chorea and hemiplegia as a ground for believing that the region of the corpus striatum is the part affected in both disorders" (REYNOLD'S *System*, 2nd Ed., Vol. II., p. 199). Dr. Alexander Robertson also gives support to this view in these words:—

> "That besides affording a more satisfactory explanation of many forms of epilepsy than previous hypotheses have done, it enables us to account better for the grouping of symptoms, and the general course of some other forms of nervous disease—*e.g.*, general paralysis of the insane. In this disorder psychical disorder usually precedes defect in the articulation of words, but occasionally they begin quite simultaneously. Now it has been known since Calmeil first gave a full description of the pathology of this disease, that at the outset it is peculiarly a disease of the cerebral cortex; so that it was somewhat puzzling to explain the occurrence of a motor set of symptoms, seeing that the nearest motor ganglia were believed to be the corpora striata, but more especially as those concerned with speech were supposed to be confined to the medulla oblongata. But the presence of motor centres in the superficial grey matter of the cortex affords a ready solution of the difficulty, showing how there may be associated two kinds of symptoms apparently so very different from each other as delusion and defective articulation, but really closely allied, since the motor intentions of language are so deeply concerned in the exercise of thought. The morbid changes observed in the brains of general paralytics are in accordance with this explanation of its pathology; for the most distinctive appearance is the adhesion of the *pia mater* to the grey matter of the surface, usually to such a degree that they cannot be separated without part of the latter being torn off. And it is worthy of note, that this adhesion is most marked in the frontal and anterior part of the parietal regions—a fact, it will be apparent, that supports the theory of localisation so far as it applies to motor centres. In the early stages, also, of the maniacal form of insanity motor symptoms are occasionally a prominent feature. The symptoms consist of sudden startings and jumpings of the whole body, and are most marked at night in bed. They are associated with general irritability of mind, and in some cases with various sensory symptoms, such as an occasional feeling of elasticity in walking, which seems to pass up from the legs to the head. . . . This combination of symptoms has also a ready explanation in the theory of the mental motorial relations of the cortex. And, in fact, the theory also accounts well for the various sensori-motor phenomena of fully developed insanity, such as is attended with 'visions' and 'voices.' These we may consider as due to the implication of their higher centres, those, namely, of the convolutions in the pathological process" (*Glasgow Med. Journ.*, Vol. XII., Dec., 1879, p. 419).

Dr. Hughlings Jackson further observes,—

> "The old method of investigation deals with circumscribed entities. It *looks* simpler. Thus many students are interested in being told that a case is one of *true* epilepsy, or of *real* chorea, or of *genuine* aphasia, who show no interest in the description of a paroxysm of convulsion, not even the simplest; who have never tried to form a clear

conception of the sort of movements they can see in a choreal child; and who, whilst they are interested in such discussions as to whether a particular case is one of *genuine* aphasia or not, take no pains to obtain a realistic account of what that patient's condition was—what he really could say or could not say. They have been taught to study cases as the symptoms show *approaches* to certain clinical standards, and not as they show departures from healthy states. The latter plan (to illustrate by a simple case of convulsion) requires a more minute study of the paroxysms, and further, it demands more *extensive* knowledge of *other* nervous diseases or symptoms. Those who only wish to know whether a given convulsion is epileptic or epileptiform, have an easy task; those who wish to know whether the discharging lesion is of convolutions in the region of the middle cerebral artery or not, have a much more difficult task. They have *to note carefully the muscles convulsed*, in order *to see if they are the same as those paralysed*, in cases of the common form of hemiplegia, which symptom is well known to be due to lesion in this region. And in order to be able to note this, they must know thoroughly beforehand the symptoms of the several degrees of hemiplegia; as, for example, that with a lesion of a certain degree of gravity there is deviation of the head and eyes. . . . Not only have I urged that convulsions beginning unilaterally point to *disease* in the region of the middle cerebral artery—a clinical statement; but I have urged the study of them for purposes of localisation of movements—a physiological statement. In hemiplegia the loss is of a certain number of possible *simultaneous movements* of the face, arm, and leg—the sum of a number of possible co-ordinations in space. Similarly, a convulsion on one side is the abrupt development of a certain number of possible *successions of movements* of the face, arm, and leg—the sum of a number of possible co-ordinations in time (*Med. Times and Gazette*, August 15, 1868; also *St. Andrew's Reports*, Vol. III., 1870). To make cases of convulsion of use for the latter purpose, much minute work and great patience are required. And after all the fear is that to many whose opinion are to be esteemed, it will seem a sheer waste of time to note the movements developed in a fit so minutely as is done, for example, in the following remarks on a case of very partial convulsion, a summary of what was observed in several seizures :—The order of involvement was that the mouth all round was first in action, then the mouth was drawn to the left, then both eyes to the left (the head to the left), then the eyelids of both sides (the left the more) closed. The thorax was affected early, and the arm late. In this case the main facts of the autopsy were that the lesion was not sufficiently local to enable one to conclude that fits beginning in the face show damage to any particular convolutional region. For instance, the fits may have been owing either to discharge of the grey matter of the convolutions of the temporo-sphenoidal lobe, or of the island of Reil. In most cases of convulsion beginning unilaterally the cerebral lesion is very extensive. A case of convulsion beginning in the left thumb, in which there was a tubercular tumor, the size of a hazel-hut, in the hinder part of *one* convolution—the third right frontal convolution—is to be found reported in *Med. Times and Gazette*, November 30, 1872, p. 597. By numerous observations of this kind we may confidently expect to arrive at clearer notions on localisation of movements. But it is possible to observe many partial seizures precisely, for some of them last a long time. . . . And *there is no other way* of ascertaining the localisation of movements in the cerebral hemisphere *of man*, than by a study of his convulsive seizures (*West Riding Asylum Report*, Vol. III., p. 181).

Regarding the phenomena of unilateral convulsions, Dr. Alexander Robertson observes:—

"(1.) One-sided convulsions do not always begin in the same part in the same case, even though there be no reason to think that there is any appreciable change in the morbid centre. In one case the convulsions begin in different parts of the body, even though there might be only a few minutes of interval between the seizures ('On Unilateral Convulsions, Localisation,' &c., *Edin. Med. Journ.* for 1869, p. 513). This shows the necessity of not too hastily drawing the conclusion that, because convulsive movement is observed to commence in a special muscle or set of muscles, therefore the source of their motor power is in a part of the brain where there is a lesion. For, it is by no means improbable that a lesion may occasionally prove more irritating to a neighbouring part, or even to one considerably removed, than where it actually exists. This principle is well illustrated by morbid sensations. Thus, a decayed tooth, though it may be but little painful itself, occasionally gives rise to a severe supra-orbital neuralgia; a diseased hip-joint may disclose itself chiefly by pain in the knee; and other similar phenomena might be adduced. At the same time it is to be observed that the order in which the muscular system is involved is usually the same in successive attacks. Dr. Hughlings Jackson has lately pointed out that, when they begin in the upper extremity, the thumb is first affected. (2.) In unilateral convulsions the so-called bilateral muscles are implicated, but the twin muscles of the otherwise sound side in most cases do not contract so firmly as those on the side first convulsed. This fact I have often observed in relation to the abdominal muscles and the *orbiculares palpebrarum* (see paper already quoted), and other facial muscles, as well as those of the limbs. By attending to this, it is very often possible to tell on what side the convulsions began in cases where they were originally one-sided. This is important in reference to the diagnosis of the side of the brain in which the lesion exists. Thus, when a physician is called in to a case of general convulsions which were unilateral at the commencement, and can obtain no account of the course of the convulsive movements, or, what is much the same, cannot rely on the information he obtains, by simply grasping the limbs of the two sides, or even by laying his open hand somewhat firmly on the abdominal wall, he may ascertain for himself, in most cases, which hemisphere is the seat of lesion; for the side on which the muscles are firmer than their fellows is the one opposite to that in which the brain is involved. For instance, should the contracted muscles of the left leg and arm be harder than those of the right ones, the disease is in the right hemisphere. If, however, the convulsions are very severe, there may be scarcely any appreciable difference between the two sides. (3.) There may be *alternate* conjugate deviation of the eyes during the same convulsive seizure. I have recorded a case of this kind in the paper from which I have already quoted. Thus, in one paroxysm the left side was first convulsed, and then the head and eyes deviated to the left. In less than a minute the convulsive action extended to the right side, the head and eyes revolving on their axis, and deviating even more decidedly to the right than they had previously done to the left. In this exceptional case both hemispheres were diseased at the surface, though in very different degrees. (4.) A study of cases reported by other observers, as well as of those in my own practice, leads me to the conclusion that, as a general rule, convulsions beginning on one side, and from a unilateral lesion, are more disposed to become bilateral the higher up in the brain the lesion is situated—*e.g.*, that where it is down in the pons or medulla oblongata the convulsive movements are less apt to involve the opposite members than where it is in the cortex of the hemisphere. A little consideration will show that this is what might be anticipated from the increasing complexity of the relation of parts with each other, both in the same hemisphere and with the opposite one, as they ascend from the spinal cord to the surface of the brain. (5.) Where convulsions begin on one side there is frequently a distinct and sometimes a prolonged interval before consciousness is involved; and it is occasionally retained throughout the whole seizure, should the seizure be a mild one. This happened in the case of a little boy who died in this hospital about four years ago, and was subject to fits of general convulsions beginning in the right hand. At the commencement of one attack he cried out to the patient in the next bed, 'O wife' (Scotch for 'woman') 'see how my hand is going.' While I stood beside another patient on one occasion, observing the contractions of the muscles of the side of the face and neck, he directed my attention to the fact that his right leg had become affected. Soon afterwards the convulsions became general, and consciousness was in abeyance. On another occasion, when the whole right side was affected, I asked him if he knew me; to which he replied, 'Yes, I do.' (6.) There is a decided increase of temperature in the convulsed members, at least in cases where the movements are vigorous. Thus, I have noticed a difference of as much as 2° Fahr. between the convulsed thigh and the unaffected one. This contrasts with the condition in hemiplegics, which varies according to the site of the lesion. Some years since I examined the axillary temperature on the sound and the palsied sides in thirty-nine hemiplegics with the following results: In 22 cases the palsied arm was colder than the healthy one, the amount varying from $\frac{1}{5}$° to $\frac{3}{5}$° Fahr.; eight were warmer, varying from $\frac{2}{5}$° to $1\frac{3}{5}$° Fahr.; nine were alike on both sides. In almost all these cases the hemiplegia was of considerable standing" ("Unilateral Phenomena of Mental and Nervous Disorders," by Dr. A. Robertson, *Glasgow Med. Journ.*, Oct., 1875).

In unilateral convulsions it is the occurrence of *repeated paroxysms* vhich tells us that there is "a discharging lesion." It is then incumbent n us—(1.) to try, from a study of the paroxysm, to find where the discharging lesion is—*i.e.*, to localise the seat of irritation; (2.) to try to trace he steps of the abnormal process of nutrition by which the discharging esion resulted; and, (3.) the causes determining the discharge.

The following are the principles upon which the investigation of cases f convulsions ought to be conducted, as indicated by Dr. Hughlings ackson:—(1.) In simple cases of convulsions the principle ought to be ecognised and acted upon, "that those parts are wont to suffer first and nost which serve in the more voluntary (special) operations, and those st and least which serve in the more automatic (general) operations." 2.) The mode of onset of the fits is the most important matter in the natomical investigation in any case of epilepsy, because it points to the art of the brain which discharges, or where the discharge begins. There re three parts where fits mostly begin—(*a.*) in the hand; (*b.*) in the face or ngue, or both; (*c.*) in the foot. In other words, they usually begin in nose parts of one side of the body which have the most voluntary uses;

and the order of frequency in which parts suffer illustrates the same law. For example, fits beginning in the hand are commonest; next in frequency are those which begin in the face or tongue; and rarest are those which begin in the foot. The law is seen in details, as when the fit begins in the hand, the index finger and thumb are usually the digits first seized; when in the face, the side of the cheek is first in spasm; when in the foot, almost invariably the great toe. (3.) The fits begin in those parts which suffer most in the common form of hemiplegia. (4.) Each of the three varieties of fits is supposed to depend on discharge of more or less of the sensori-motor processes of some particular series; and the mode of onset gives a clue to the series. Thus, those fits which begin in the hand are supposed to depend on discharge of a part of the cerebrum where the hand is largely represented in movement, to which other movements (those of the face and leg) are sequent and subordinate. Again, in fits beginning in the foot, the internal part discharged contains processes for certain movements of the foot and lower limb to which certain other movements (those of the upper limb) are sequent and subordinate: in *this* series the foot is the voluntary (special) part. In fits beginning in the (right) cheek, there is often temporary defect of speech; and the patient tells that the fit affects the tongue; and there is *after* some of these attacks epileptic aphasia analagous to epileptic hemiplegia, and after some fits beginning in the hand (right) there is occasionally temporary defect of speech. (5.) The proximate cause of convulsions is an abnormal increase in the nutritive changes in the nervous centres (Dr. RUSSELL, REYNOLDS)—a nutrition which Dr. Hughlings Jackson thinks is carried on at an inferior level, so that if the nervous matter cannot get enough phosphorus, it takes nitrogen, provided it is under conditions favourable to some kind of nutrition—just as a plant will take soda when it cannot get potash. (6.) It is necessary to note the *order of the spreading of the spasm.* (7.) The bilateral muscles of both sides are involved when the unilateral of but one side are well engaged. (8.) Seek for those symptoms resulting from "destroying lesions" which correspond to those which are the results of "discharging lesions." If, for example, we find *paralysis* of the face, arm, and leg the effects of a destroying lesion of the corpus striatum, there are paroxysms of *convulsions* which correspond, and which depend on discharges of convolutions near to this body. Dr. Hughlings Jackson illustrates this by lesions of *the left side* of the brain in the following tabular form:—

FIRST DEGREE.

Corpus Striatum Palsy.	*Corpus Striatum Epilepsy.*
Mouth turns to left.	Mouth drawn to right.
Right arm paralysed.	Right arm convulsed.
Right leg paralysed.	Right leg convulsed.

This is only a comparison with the first degree of paralysis. A graver lesion of the corpus striatum produces the following symptoms, and there is a further degree of convulsion which corresponds:—

SECOND DEGREE.

Corpus Striatum Palsy.	*Corpus Striatum Epilepsy.*
Head turns to left.	Head drawn to right.
Two eyes turn to left.	Two eyes drawn to right.
Face turns to left.	Face drawn to right.
Trunk muscles weaker on right.	Trunk muscles in spasm on right (?).
Arm and leg paralysed on right.	Arm and leg in spasm on right.

THIRD DEGREE.

There is a still further degree of hemiplegia: a very grave lesion in the region of the corpus striatum of *one* side will produce palsy of *both* sides of the body; then, of course, the term 'hemiplegia' is, strictly speaking, a misnomer. Similarly, the convulsion which begins on one side—in the hand, for example—will spread at length to the other side; it *becomes* universal. A fit which thus *becomes universal* depends on discharge in but *one* hemisphere; for facts seem to show that each half of the brain represents movements of both sides of the body (*Lancet*, February 15, 1873), but that it represents the movements of the two sides in different degrees and orders. I have, however, had few opportunities of watching convulsions which, beginning in one side, have reached the other. One of my patients, whose fits began in his *right* ulnar fingers, said that once, after the right side of the body had been involved, 'it' went across his chest to the *left* arm; 'it' went *down that* arm, but did not reach the fingers. The points to observe about the *second* side are—1. Is the arm or the leg first affected? 2. What part of either of the two limbs does the spasm first reach? Does it first reach their upper parts (shoulder and thigh), or their lower parts (hand and foot)? 3. Does it especially affect any group of muscles?—*e.g.*, the extensors or flexors (Dr. HUGHLINGS JACKSON, l. c., p. 338).

But while irritation or discharging lesions of the cortex, indicated by unilateral convulsions or mono-spasms, are theoretically to be separated from destructive lesions, indicated by paralysis of voluntary motion, practically they cannot be separated from each other by a hard and fast line; for, many cases of limited destructive lesions are associated with unilateral convulsions or mono-spasms. There are numerous cases, however, in which the predominant, and sometimes the only, feature has been a convulsive affection limited to a limb or combined in the way in which monoplegiæ are combined; or which, commencing in a constant and definite way, becomes generalised into a unilateral convulsion, with or without loss of consciousness, or occasionally passes into bilateral convulsions—the pathology of which it has been the merit of Dr. Hughlings Jackson to indicate, and to which the name of "Jacksonian epilepsy" is now commonly applied. The late Mr. Callender also arrived at the conclusion, that convulsions occurred more particularly with superficial lesions of the cortex in the neighbourhood of the middle meningeal artery. But the result of Dr. Ferrier's experience is that from the mere occurrence of an unilateral convulsion, with loss of consciousness, or of an epileptic attack in which the convulsions are exhibited mainly on one side, we can form no opinion as to the nature or seat of the lesion. We may suppose, and with reason, that the opposite cerebral hemisphere is more particularly at fault; but there need be no discoverable lesion; and should a lesion exist, it need not be in any definite position—in accordance with clinical facts and experiments; for Dr. Ferrier has found that long-continued irritation applied to any part of the hemisphere, other than the motor area, may result in an attack of unilateral convulsions. If, however, the convulsion be of the nature of a mono-spasm, or if tending to become generalised, it begins invariably in the same way, and does not cause loss of consciousness; and if it be followed by paresis or paralysis, more or less permanent, we may diagnose an irritative lesion of the motor area of the opposite hemisphere. Also, the diagnosis of the seat of an irritative lesion is naturally more uncertain than that of a destructive lesion, owing to the difficulty of determining the extent of the zone, or the special point in this zone in which vital irritation concentrates itself. Irritative lesions vary in their anatomy. They are all such as tend to induce irritability and hyperæmia of the cortical grey matter, or subjacent medullary tracts—as seen in epileptic convulsions induced by absinth (MAGRIAN); and also after injury to the cortex. The irritative lesion may therefore be an acute inflammation, or a condition of irritability set up by some chronic morbid product —meningo-encephalitis set up by syphilitic disease being by far the most common; so that syphilitic epilepsy is sometimes spoken of as synonymous with "Jacksonian epilepsy." Tuberculous affections, tumors, cysts situated superficially, cicatrices of old wounds, spiculæ of bone, and the like, are all capable of producing irritation.

The valuable researches of Dr. Althaus, extending over the past thirty years (commencing in 1838), show that convulsions or eclampsia constituted by far the most frequent and fatal disease of the nervous system during that period; that more than three-fourths of a million of deaths were owing to them in England and Wales; that nearly one-half of the entire number of deaths from all nervous diseases were due to their influences, the total percentage for the period amounting to 47·84 per cent. At the same time there seems to have been an uninterrupted fall in the mortality from convulsions ever since registration of diseases was commenced—from first to last, of 18·78 per cent. The influence of age and sex on the production of convulsions show that infants are more liable to them than adults or the aged. The earlier the age the more fatal. For example, while 18,680 deaths occur in the first year of life, 2,108 occur in the second, 859 in the third, 472 in the fourth, and only 228 in the fifth. Thus, the first period of five years shows a mortality of 23,347; so that convulsions are, without exception, the most fruitful source of infant mortality. No other disease equals this mortality. In 1874 convulsions caused 12·49 per cent. of the entire infantile mortality under five years of age, but after that age the numbers for the subsequent quinquennial periods markedly and precipitately diminish, till the disease ceases to be important after ten years of age. Convulsions are also found to be much more fatal to boys than to girls, the ratio being as twenty-five for the former to twenty for the latter (*Diseases of the Nervous System*, p. 55).

The following substantive diseases may now be described in a group in which convulsions or other abnormal motor phenomena play a conspicuous part, but each of them having special peculiarities of its own.

INFANTILE CONVULSIONS OR ECLAMPSIA.

LATIN EQ., *Membrorum Distentio Infantalis;* FRENCH EQ., *Convulsions de l'Enfance;* GERMAN EQ., *Kinderkrämpfe;* ITALIAN EQ., *Convulsioni de 'Bambini.*

Definition.—*Convulsive seizures, consisting of contraction of muscles by gradual but rapid shortening of the muscular fibres, causing such hardness and stiffness of the muscle or limb that it cannot be overcome. This period of tonic contraction is followed by clonic spasmodic phenomena, characterised by the occurrence of alternating movements of contraction and relaxation independent of the will, which is as powerless to suspend or moderate them as to excite them* (TROUSSEAU).

Pathology.—*Causation.*—Convulsions may occur from the time of birth until the end of the seventh or eighth year, and arise from the following causes:—

(1.) From manifest anatomical lesions of the nervous system, especially during the progress of acute diseases of the brain or spinal marrow, which are analogous to forms of epileptoid convulsions arising from cerebral tumors and other chronic disorders of the brain. (2.) As the expression of many very different diseases, or the premonitory beginning of them; for example—(*a.*) Of *Epilepsy;* (*b.*) *Hysteria;* (*c.*) *Chorea;* (*d.*) *Tetanus;* (*e.*) Blood-poisoning, as of uræmia and of narcotic poisons; (*f.*) Material of contagion of certain diseases, which by convulsions usher in the attack, as of *scarlet fever, measles,* or *small-pox;* (*g.*) The high fever-heat of such diseases as pneumonia and other inflammatory diseases of childhood (hyperpyrexia); (*h.*) The result of terror and other mental emotions, causing excitement of the cerebral ganglia, transmitted thence to the cortex or medulla oblongata; (*i.*) Irritation of peripheral nerves from teething, intestinal worms, indigestion, or painful injuries of the skin, like burns, or from application of blisters, or sinapisms, or painful eruptions, which induce that morbidly irritable state which is expressed by "reflex convulsions." (3.) Infantile convulsions also occur as the expression of an *hereditary predisposition.* This nervous susceptibility manifests itself in different generations in the same or in different ways. It commonly happens that parents—mothers especially—who in their infancy were subject to fits, give birth to children who, in their turn, are affected in the same way (TROUSSEAU, NIEMEYER). Infantile convulsions are thus often congenital, all the children of a family being sometimes affected in the same way. (4.) Infantile convulsions also occur as a result of *insufficient feeding,* or of bad food; or in those who have lost large quantities of blood from *spontaneous hæmorrhage, venesection,* or *leeches,* or who had profuse *diarrhœa* persisting for a long time. In proportion as the nutritive and vegetative functions are feeble and languishing, so will nervous phenomena be mobile, exalted, and irregular. The dependence of the normal state of the nervous system on the blood and the nutritive functions being mutual, is most strikingly marked in children, confirming the law enunciated by Hippocrates—*sanguis moderator nervorum* (TROUSSEAU). (5.) As a consequence of albuminuria—as an acute affection—as a result of scarlet fever, or of Bright's disease. (6.) Traumatic injury, such as penetration of the brain by fine needles. Examples of this occurrence are cited by Trousseau and Underwood, where, on removal of the child's cap, a bit of coloured thread twisted on the scalp being laid hold of, a long needle to which it was attached was pulled out. (7.) In the *acute exanthemata* and in *pneumonia* convulsions in children often seem to take the place of the chill or rigor, which is the precursory sign in older people of the commencement of such acute diseases; and which in reality is a convulsion of a small degree due to alternate contraction and relaxation of muscles.

Circumstances apparently the most insignificant may bring about convulsions in children *pre*disposed to them; so that there are children who are liable to be convulsed with as much facility as other children will sleep, dream, or become delirious. This predisposition is chiefly hereditary.

(*b.*) *Morbid Anatomy* discloses many morbid conditions of a very different nature, associated with fatal cases of convulsions; but in the majority of cases they are due to the mode in which death has taken place. They are generally those of death from asphyxia; but the cases may be broadly classed into—(1.) Those which arise from manifest anatomical lesions of the nervous system; and, (2.) Those which seem to be caused by no material change, or at least where the most inquisitive examination after death has failed to reveal the existence of any organic lesion to which the convulsions may be ascribed. To the convulsions associated with the first of these two classes the name of *symptomatic* has been given; to those of the second class, the name of *idiopathic convulsions*, as well as *eclampsia*, have been assigned.

But although no anatomical change can be demonstrated, it is not to be inferred that they are independent of any material change in the nervous system. For the most part the lesions that have been found in the nervous centres in these and similar diseases are for the most part secondary—the result or consequences of irritative disturbances in the nervous centres. Morgagni expressed this belief long ago, when he wrote that "the cause of convulsions, which consists in an invisible change that has occurred in the brain and nerves, cannot be detected by our senses after death. Its effects alone are seen, and these vary according to the violence and duration of the convulsions." The more often, therefore, convulsions take place, the more liable will the child be to the occurrence of permanent and visible lesion of the nervous system as a result of them. Their repeated recurrences tend to weaken the intellect and impair the general health.

It is the *idiopathic convulsions—eclampsia*—those which are connected with well-defined physiological conditions, which ought to be considered under the head of "Infantile Convulsions." Those which are symptomatic, such as the convulsions of *cerebro-meningitis*, or of other diseases, form part of the history of the particular disease of which they are one of the expressions. When, however, they are the result of the existence of those diseases, and come in at or toward their termination, instead of at the commencement, then they properly belong to this class—that of *eclampsia,* the particular disease being then regarded as their exciting cause.

Symptoms.—The rigidity of *tonic contraction,* for however short a time, always precedes the *clonic spasms.* These may be very violent, while the preliminary contraction may be very slight and transient. Hence, the tonic contraction is apt to be overlooked, or lost sight of. In other cases the *tonic contraction* alone constitutes the convulsion. A period of rigidity is the essential element of all convulsions. It is never absent, and may alone be present. *Collapse, stupor,* or *coma* may also occur; they are, however, no necessary part of the seizure, but the effect of the extension of the irritative lesion causing the convulsions.

Infantile convulsions may come on suddenly, like an epileptic fit in the adult, and with no premonitory warning to indicate the invasion of the disease; or preceded by symptoms showing undue excitability of the nervous system. The child may be cross, self-willed, passionate, and fidgety, its facial expression at once weary and restless; sleep is disturbed by grinding of the teeth and sudden screams; somnolence prevails in the daytime with muscular twitchings, particularly in the face. Hence the sardonic grin from spasmodic movements of the zygomaticus major and platysma myoides muscles; breathing becomes shorter and more rapidly performed; and then the fit of eclampsia usually breaks out suddenly (ALTHAUS, l. c., p. 71). The child may utter a cry, lose consciousness, become rigid as a deal board, and ultimately commence to struggle with a fixed chest and suspended respiration. The physiognomy becomes vacant; the face, pale at first, becomes red, livid, and distorted; the eyes fill with tears, which run over the cheeks; and the veins of the neck are turgid, and project like knotted cords. The eyes are sometimes pulled back into the orbits and rolled upwards. Carbonic acid accumulates in the blood, respiration being arrested by spasm of the glottis and rigidity of the respiratory muscles. The *clonic spasms* then set in, characterised by disorderly and involuntary contractions of many muscles,—the limbs are alternately flexed and extended; the fingers and toes successively bent and stretched out, separated from or approximated to one another, but most frequently in a state of forcible flexion; the thumb is adducted and hidden by the fingers. The head is drawn backwards or is bent forwards; and sometimes it is pulled laterally and backwards by irregular and jerking rotatory movements of the sterno-cleido mastoid and trapezius muscles. The muscles of the face share in the *general convulsions;* the eyes are the seat of jerking movements, and roll in their sockets; they are generally drawn up under the upper eyelid; more rarely they are pulled downwards, and there is *strabismus convergens.* The labial commissures are dragged upwards and outwards, hence the distorted face is sometimes frightful to beholders; and then, on each convulsive shock, the air passes through the kind of funnel formed by the corners of the half-opened buccal orifice, making a suction noise, accompanied by a flow of frothy, and sometimes of bloody saliva. The tongue is apt to be protruded, and may be bitten, and so lacerated by the teeth. As the muscles of the trunk are affected during the tonic stage, the inspiratory muscles are fixed, and the larynx, spasmodically contracted, no longer permits the free passage of air. Contraction and convulsion of the abdominal muscles cause expulsion of urine and of fæces. The *clonic spasms* are at first rapid and limited, but become slower and more extensive, till at last a deep inspiration, followed by complete relaxation, announces the end of the fit. The child is then apt to fall into a state of somnolency and stupor. Such a fit may last one or two minutes, and may be repeated over and over again during half a day, a whole day, or more days, even to eighteen days, —as in one case, during hooping-cough. Death may take place suddenly during the first attack (TROUSSEAU, ALTHAUS). Such attacks may last from two to five minutes.

A repetition of fits may be anticipated, when the sleep is not sound after the first fit has subsided, and when the child throws itself about, gnashes and grinds its teeth, and twitches its limbs from time to time. If frequently repeated death may follow by coma gradually deepening. In such fatal cases, where attacks incessantly repeat themselves, and collapse and coma continue between and after the attack, pulse and respiration become accelerated, and a rapid *increase of temperature* occurs (108° to 109° Fahr.), attaining a greater temperature a few hours after death (ALTHAUS, l. c., p. 73).

General convulsions from irritative causes affecting the pons and medulla oblongata are always bilateral; but there are also localised convulsions to be recognised, such as *partial convulsions* of one-half of the body, of the trunk of the body, of the muscles of the face, of the muscles of the eye (unilateral convulsions), so that the child, always drawn to the same side, may fall out of bed. In general bilateral convulsions the pulse is generally quick, full, and dicrotous. A series of attacks rarely last beyond a week, with intervening states of exalted excitability, as shown by jerky respiration, squinting, sardonic grin, flexion of toes and thumbs.

Another form of convulsion consists of partial spasms of the pharynx, larynx, and muscles of respiration, especially accompanied by movements of the globes of the eyes, to which the vague names of "*inward fits*," "*inward convulsions*," or "*inward spasms*," have been given. Babies a few days after birth are apt to suffer from slight convulsions of this kind. The child lies as if asleep, but rolls about the eyes—the eyeballs are turned upwards with the corneæ under the upper lids. The eyelids move; and there is gentle moaning, or difficult breathing; there are twitches of the muscles of the face, and the sardonic smile, with flushing or lividity. It is to these phenomena that nurses have given the name of "inward fits."

Prognosis.—If the tonic stiffness or spasms continue affecting the trunk, the sudden arrest and complete suspension of the respiratory functions which it entails may be fatal in course of time; so also "*inward fits*" implicating the larynx. But except convulsions occur during hooping-cough, laryngismus stridulus, or apoplexy, or after long exhausting diseases, a child rarely dies actually in a fit; and it is the ever recurring convulsions which are apt to prove fatal. Death by asphyxia is the usual mode of fatal termination, or sometimes by syncope. Convulsions which take the place of chill, occurring at the outset of diseases, are not generally attended with danger; but those which occur during the course of general diseases, such as eruptive fevers, enteric fever, pulmonary or intestinal inflammation, and the like, and especially towards their close, are significant of great danger, arising from grave complication in the condition of the patient. Convulsions under such circumstances generally usher in death. They are more particularly liable to occur thus in cases of scarlet fever, in the third stage, associated with commencing albuminuria. Generally speaking, convulsions are less dangerous in proportion as they are more easily excited. Danger is great in attacks during the first months of life, for then the brain is extremely delicate, very vascular, and growing very rapidly. The excitability of the cerebro-spinal centre is also excessively great at this time, and slight causes, which would pass without effect in adults, are in infants sufficient to excite grave mischief. Hence in older children prognosis is more favourable. The frequency and fatality of convulsions diminish as the development and organisation of the nervous system become more perfect with advancing years.

Treatment.—Of all the causes mentioned in connection with convulsions, none are so common or universal as *indigestion.* Hence the quantity and quality of the food and condition of the evacuations, by vomiting or by stool, ought to be inquired into at the first. If some unwholesome diet has been given, a good purgative of *calomel,* mixed with powdered sugar, should be put on the back of the child's tongue. If indigestion is traced as the source of the fits in an infant or young child, a few drops of *aromatic spirit of ammonia,* or the same quantity of *ether,* may be given in a teaspoonful of water; or a drop or two of *anise oil,* rubbed up with sugar, may be given from time to time, by laying the powdered sugar so saturated on the tongue of the child. It is in such cases that mere flatus in the bowels may cause a fit in a predisposed child. The belly will be found tumid, when gentle friction over the abdomen (following the direction of the colon), combined with the warm bath and those soothing aromatics, will give relief.

The clothing must be loosened completely, so as to admit air freely, and *anti-spasmodics,* such as *ether* alone, or combined with *musk* or *belladonna,* may be given, till the progress, duration, seat, or probable cause of the convulsions may be ascertained. An *emetic,* a *purgative enema,* a *searching purgative,* such as *calomel,* combined with *jalap* or *rhubarb,* may be indicated; or the removal of a pin in the dress, which had been long pricking the skin, or too tight a dress, may, on removal or rectification, cause the convulsions to cease.

In all cases, however, when a child we have never seen before is found in a fit, it is best to order a warm bath to be prepared, and to have the room freely ventilated. The next points to inquire into are the condition of the pulse and of the child's head as to form and completion of its bones. "Pass one hand quietly and carefully over the child's head while the pulse is felt with the other. Fulness or weakness of the pulse will be a guide to diagnosis; so also if the head be hot or cold, if the anterior fontanelle be tense and protruding, or sunk and retracted, and if the face be flushed or pale" (ELLIS, *Diseases of Children,* p. 127).

When the child is completely stripped of its clothing, it should be observed if the legs are drawn upwards to its belly; and if so, and the head be hot, and the fontanelle prominent, there is congestion of the head, which may either be the cause or the consequence of the fits. In any case the child should be placed first with its feet in the bath, in which a handful of mustard may be mixed (two ounces of mustard to four gallons of water). At the same time vinegar and water, or spirits and water lotion, may be applied to the child's head; while hot flannel cloths wrung out of the hot mustard and water bath (with more mustard sprinkled on the surface of the flannel) should envelop each leg and foot. If the head be cold or the fontanelle depressed, the entire body of the child should be immersed in the mustard and water bath (temperature about 98° Fahr.), and friction employed over the whole skin. A little *sal-volatile* may be held to the nose, and a few drops of brandy in a teaspoonful of water may be used to moisten the lips. It is possible the child may be in a state of faint or syncope, and not in a fit.

During these proceedings it is important to find out the previous medical history of the child, as to,—(1.) Which of the acute specific diseases it may have had. (2.) State of dentition, and also condition of gums to be determined, by passing the finger along them. (3.) What the child may have been eating recently. (4.) The previous existence of diarrhœa. (5.) The previous occurrence of carpopedal twitchings, or drawing in of the hands and feet, with general irritability. If this latter condition is found to have existed, and if the fontanelle be prominent, there is some abnormal condition of the nervous system. A calomel purgative may be required, or leeches, or a rubefacient behind the ear or over the vertex. With headache, vomiting, and some feverishness, a pulse of 130 would rather point to stomachal disturbance; whereas with a slow pulse (say at 40), these symptoms would rather indicate an early manifestation of tubercular meningitis, where *bromide of potassium* or *ammonium* may be required after suitable purgation. If recently convalescent from scarlatina, and there be albuminuria or anasarca, a hydragogue cathartic may be required, in the form of twenty grains of *compound jalap powder* with a grain of *scammony*, to a child five years of age, repeated every two or three hours till the desired result is produced. It may be necessary at the same time to dry-cup the loins. If convulsions come on during hooping-cough, apply ice or cold to the head, with free purgation, and use a sedative cough mixture (Ellis, l. c., p. 129).

A clyster of one part vinegar and three parts water, the application of cold compresses to the head, or of leeches behind the ear, if the convulsions do not subside, are remedies advocated by Niemeyer.

If symptoms denote stupor, cold affusion should be prescribed; if, on the other hand, they denote some collapse, *wine*, *camphor*, *musk*, and other *stimulants* must be used.

If dentition is causing the convulsions, the dense tissue of the gums over the tooth ought to be relieved by lancing it, or cutting it across over the crown of the tooth.

Dr. Tanner advises the administration of *bromide of potassium* as a medicine from which more may be hoped for than from any other. If there be inability to swallow, it ought to be given in *enemata* of *beef-tea.*

Where great restlessness prevails, *dilute hydrocyanic acid*, with *tincture of hyoscyamus*, may be given.

Compression of the carotid arteries and chloroform inhalation, or a mixture of ether and chloroform, are only to be had recourse to in cases of prolonged duration, depending on such irritation as that of dentition. Anæsthesia thus induced is of great value.

Trousseau is strongly opposed to blisters and general "revulsions to the skin," except in some cases of "inward fits" involving the diaphragm or the heart. In such cases the action of the remedy, as *ammonia,* must be immediate.

PUERPERAL CONVULSIONS—Syn., ECLAMPSIA PUERPERALIS.

Latin Eq., *Membrorum Distentio in Puerperis,* Idem Valet *Eclampsia;* French Eq., *Convulsions Puerpérales*—Syn., *Eclampsii;* German Eq., *Puerperal-Convulsionen*—Syn., *Eclampsia;* Italian Eq., *Convulsioni Puerperali*—Syn., *Eclampsia.*

Definition.—*An acute affection of the nervous system, characterised by loss of consciousness and sensibility, by tonic and by clonic spasms. It occurs only as an accessory phenomenon of another disease (generally Bright's, in an acute form) which, under certain circumstances, spreading its toxæmic effects on the nutrition of the brain and the whole nervous system, gives rise to convulsions. The blood-poisoning is generally to be referred to anæmia, or to the products of the changes in urea which are retained in the blood, or to the retention of other excrementitious constituents of the urine* (Braun).

Pathology.—The affection here defined does not include all cases in which convulsions occur during pregnancy or childbed. It is a disorder distinct from epilepsy; although in some conspicuous phenomena it is closely allied to it. It has a certain train of symptoms peculiar to itself; and a certain train of pathological conditions are essential to its development. Exceptional varieties of convulsions also occur when the blood is impure from imperfect elimination of carbonic acid through the lungs, or from the retention of bile, or from septic poisons pervading it, as in *typhus fever.* Puerperal eclampsia is also to be distinguished from those convulsions of a reflex origin due to irritation of peripheral nerves, and associated with hysterical manifestations.

(*a.*) *Causation.*—Puerperal eclampsia is now regarded as essentially connected with uræmic poisoning, associated with an albuminous condition of the urine; and there are some conditions essential to the puerperal state, which modify the significance of *abluminuria* occurring for the first time during the course of pregnancy, as compared with its occurrence independent of gestation:—(1.) There is a certain constitutional sensitiveness eminently characteristic of pregnancy which has an all-pervading influence over the nervous system (Leishman). (2.) There is the peculiarly altered condition of the blood in pregnant women, which predisposes them to ailments peculiar to their state (see Vol. I., page 815). (3.) The large part which is played in every act or process of the generative function by the emotional and convulsive elements. Albuminuria and puerperal eclampsia are in some way mutually dependent upon each other; but it is not quite determined between the albumen and the paroxysm as to which is the cause, and which is the effect. There are also cases in which no uræmic poisoning exists; and the rule is not absolute that Bright's disease is *the* cause of puerperal eclampsia; nor has it been proven that all cases of albuminuria are necessarily examples or results of true Bright's disease. Although fibrinous exudation and albuminous urine indicate, undoubtedly, the first stage of Bright's disease, and so are of inflammatory origin, it by no means follows that the same symptoms cannot, by any possibility, proceed from other than inflammatory causes (FRERICHS, LEISHMAN). The theory has also been advanced, that the albuminuria, and the constant succession of pathological changes, were due to pressure on the renal veins; while there is also the theory, that the nervous system, and not the vascular system, may after all be the starting point of puerperal eclampsia. "In such cases the disease appears to depend upon reflex irritation of the kidneys by the gravid uterus, similar to the irritation of the salivary glands, the mammæ, and the thyroid; and not upon mere pressure alone" (TYLER SMITH).

(*b*.) *Morbid Anatomy* throws no new light on the subject. A fatal result may depend "in a great measure upon the extent to which the structure of the kidney has been involved; and if in fatal cases the hyperæmic or exudation stage has rarely been observed, we may be sure that it is because these cases usually recover. If, on the other hand, the stage of atrophy has been reached, we cannot wonder that such irremediable disorganisation should culminate in a fatal result, with or without convulsions. Besides the morbid appearances which are characteristic of lesion of the kidneys, the only observations of importance which have been made are, that the lungs are constantly œdematous, and sometimes emphysematous—the result, it is assumed, of the straining from the fits. The spleen is almost always enlarged; but this should not be mentioned as characteristic of the disease in question, as it is well known that enlargement of this organ is very usual, if not invariable, during pregnancy and the puerperal state, associated, probably, with some compensatory changes in the circulation" (LEISHMAN, *A System of Midwifery*, p. 769).

Symptoms.—The convulsive manifestation of puerperal eclampsia may be divided into two distinct periods. The first is characterised by tonic convulsions, seldom lasting more than twenty or thirty seconds; the second period, that of clonic convulsions, lasts much longer—from one to five minutes, or even more. The gradual restoration of the respiratory function during this second period prevents any special danger to life; and it is therefore during the first or tonic period only that there is immediate risk. As to premonitory symptoms of some kind, there are few cases in which they may not be detected. One of the most important is *œdema*—of common occurrence in the ankles, feet, and labia majora—generally developed some weeks before the appearance of the first fit. It is also occasionally, though rarely, to be noticed in the upper part of the body and in the face. The urine ought to be at once examined (if it has not been examined before), and the examination repeated from day to day, as the albumen may only be present intermittently, and therefore a negative result is no conclusive evidence of a satisfactory state of the kidney. The albumen will be shown by the ordinary tests of heat and nitric acid; while the microscope will in most cases reveal the presence in the urine of hyaline tube casts, with or without blood corpuscles; or it may indicate the presence of more advanced and serious renal disorder. The quantity of albumen has generally an intimate relation to the extent, intensity, and duration of acute Bright's disease; but not so constantly to the violence of the eclampsia; and the more acute the Bright's disease, the darker is the urine, and the more numerous, generally, are the blood-corpuscles. The other premonitory symptoms are especially three, which demand special attention—namely, *cephalalgia, derangements of vision,* and *epigastric pains* (CHAUSSIER, quoted by LEISHMAN).

Headache is the most frequent of all. It is extremely acute, and is usually complained of in the frontal region. At first it is intermittent, but subsequently, and especially when the fit is near at hand, the pain often becomes continuous.

Disturbance of the Sense of Sight is a symptom of grave import. It may be at first either cloudiness or dimness of vision, or that peculiar indistinctness which gives one the idea of looking through the highly rarefied atmosphere over a furnace—sensations common in those subject to biliary derangement. Or, objects may seem to have peculiar colours, and the vision becomes gradually more impaired, although in some instances the affection is intermittent. Often, loss of sight does not come on till immediately before the fit, and sometimes during labour; or before it the patient may complain of sudden and complete loss of vision, and in a few minutes, or, it may be, seconds, she is overwhelmed with the most violent eclamptic seizure.

Epigastric pain is of the least frequent occurrence. It is extremely severe, and may last for hours; and when it is of unusual severity, it is said to be an almost certain precursor of a convulsive attack.

In a considerable proportion of all the cases of puerperal eclampsia, no albumen is to be detected during the premonitory phenomena—a fact which must be carefully borne in mind.

The convulsive seizure of true puerperal eclampsia sets in after some of those precursory symptoms. The patient seems deeply absorbed and preoccupied; then her gaze becomes fixed for a few seconds, and the fit at once commences by rapid contractions of the muscles of the face, of the eyelids, and of the eyeballs, which seem to roll in their sockets. These twitching movements, which give to the countenance a most painful expression, presently give place to tonic contractions of the same muscles and of the neck. The mouth is first twisted towards the left, and the face is slowly turned towards the shoulder of the same side. The upturned eyeballs show, through the half-closed eyelids, the inferior segment of the sclerotic. After being slowly turned to the left, the face, by a movement in the contrary direction, turns towards the right shoulder. From the head, the convulsive phenomena rapidly extend to other parts of the body. The extension of the trunk, thrown into violent contraction, tends to bend the spinal column backwards (*oposthotonos*). The body becomes perfectly rigid. The hands close with force, the thumbs being bent inwards upon the palm, and grasped by the other fingers. The superior extremities may assume a semiflexed position. Finally, the diaphragm and respiratory muscles become involved. Respiration is suspended. The face becomes livid. The muscles of the larynx, and possibly those of the throat, being strongly convulsed, close these orifices. The tongue may be cut by spasmodic closure of the jaws upon it. Consequently, the air compressed by the convulsive stricture of the thorax can only escape with great difficulty, and produces *a peculiar, interrupted, hissing expiration.* There is complete loss of consciousness and of all sensation. The patient neither sees nor hears; and if we pinch or burn the skin, she makes no attempt to withdraw from an irritation of which she does not seem to have the slightest perception. In less than half a minute from the commencement of the fit, clonic convulsions, affecting the whole muscular system, succeed the tonic variety, when jerking movements of the head, trunk, and limbs take the place of the general rigidity of the preceding period. Frightful contortions of the countenance are the result of the irregular movements of the mouth, eyelids, and eyeballs. Respiration meanwhile becomes re-established, when a frothy and often bloody foam is forced from between the lips. The movements of the trunk and limbs consist of twitchings, so trifling in extent as merely to move the body without displacing it, so that there is not the same necessity for restraint as in some other convulsive diseases. The pulse is strong and full at the commencement of the fit, is rapidly accelerated under the influence of the muscular and respiratory disturbance, and becomes extremely feeble towards the height of the paroxysm. It sometimes happens that the contents of the bladder and rectum are voided during the attack (CHAUSSIER, LEISHMAN).

As the fit passes off, the symptoms progressively decline. The balance of the respiratory and circulatory systems is restored; the colour of the surface becomes natural; the movements of trunk and limbs become feebler and less frequent, and the convulsions finally cease.

Diagnosis.—All cases of convulsions occurring during pregnancy and childbirth, are to be regarded as cases of puerperal eclampsia; but hysterical and epileptic fits are to be excluded, and such fits as may arise from apoplexy or meningitis. The previous history of the patient as to hysteria or epilepsy may aid the diagnosis. Contrasted with epilepsy, there is no initial cry or scream, consciousness is more gradually lost, and completely so only when the attack reaches its acmé; and when a series of attacks takes place, consciousness does not return before the patient dies, or till after delivery has taken place in cases which recover, when the attacks cease completely. Stupor may continue for days; and is as a rule so deep, that the mother on recovering consciousness is unaware of having been delivered, and may refuse to acknowledge the child as her own (Dr. ALTHAUS, l. c., p. 77). In convulsive seizures from anæmia, and in uræmia generally, in patients who are not pregnant, the temperature is considerably lowered from the beginning; and as the symptoms of uræmia become more strongly marked, the temperature continues to fall, reaching a minimum of 82·5° Fahr. previous to death. On the other hand, in puerperal eclampsia the reverse occurs; the temperature rises rapidly as soon as the convulsive attack commences—the maximum (amounting to 110° Fahr.) is attained just previous to or soon after death.

Prognosis.—After the fit has entirely ceased, the patient may lie in a comatose condition for some time, perhaps for many hours. A dull languor or a confused feeling with headache is then generally complained of; and it may be some time before the patient completely recovers. In extreme cases, the tonic spasms may be so severe in intensity and duration, that the patient's life is at once sacrificed in a few seconds. And in those cases in which the fits succeed each other with great rapidity, the patient never regains consciousness, but remains in a condition of complete coma —only disturbed by the recurrence of paroxysms—which persists till the case terminates either in recovery or in death.

About thirty per cent. of mothers have hitherto fallen victims to the direct or indirect effects of this disease; and if such a death-rate is to be reduced, it must be by a careful and earnest investigation of pathological theories, and an observation of clinical facts. The life of the fœtus is always in great danger, and the infant dies in about one-half of all the cases; and almost always when the symptoms are severe, and the attacks come on in rapid succession. The toxic influence from the blood of the mother extends to that of the child. "If, after numerous uræmic convulsive fits, the child is born alive, a large quantity of urea is found in the blood taken from the umbilical cord; but if it is born dead, we can immediately after the birth demonstrate the presence of carbonate of ammonia in the fœtal blood" (BRAUN, quoted by LEISHMAN, l. c.)

Next to rupture of the uterus, eclampsia is the most disastrous affection which it is possible to encounter in the practice of obstetrics; and the points in prognosis which chiefly suggest anxiety are,—(1.) Scanty secretion of urine, with an abundance of albumen (as when the urine becomes solid on boiling); (2.) violent fits with short intervals; (3.) profound coma. The converse of these conditions give good grounds for hope in recovery. The relative frequency of the three periods at which eclampsia most develops itself is in the following order:—most frequent during pregnancy, next during labour, and least frequently after delivery; and the average of English and continental practice yields about one case in 350 labours (LEISHMAN). In England and Wales Dr. Althaus' statistics give one in 558; the Vienna Maternity gave one case in 544 births (BRAUN). The death-rate is generally put at 33 per cent.—one case in three proving fatal (ALTHAUS, l. c., p. 79). The disease appears to be more frequent in the first than in subsequent pregnancies; and in the young, robust, and full-blooded, rather than in weak and elderly persons. Those who have once suffered are again liable in subsequent confinements. The dangers of eclampsia depend in no slight degree upon the condition of the woman, and especially the period as regards pregnancy, labour, and after delivery, at which symptoms first manifest themselves. When eclampsia occurs during pregnancy, it is almost during the last three months that the first attack takes place, the vitality of the child being in most cases undoubted. It rarely happens in those cases—and then only when the symptoms are moderate—that pregnancy is permitted to go on to its natural term; and this alone, irrespective of toxæmic action, is apt to compromise the life of the child (LEISHMAN). In one-fourth of the cases the toxæmia is sufficient, without the occurrence of eclampsia, to induce premature labour; but if the convulsive disorder should be developed, the chances of mature gestation and of the life of the child are still further reduced (BRAUN, l. c.) It is a matter of dispute whether the eclampsia which develops itself for the first time after delivery, is or is not more dangerous than when it develops at the other periods. In the cases in which fits have come on before delivery, the completion of labour, although it usually produces a marked amelioration of the symptoms, by no means places the patient out of danger. There is in such cases a special tendency to post-partum hæmorrhage; and a proclivity to the various inflammatory affections to which a parturient woman is liable, such as uterine phlebitis, peritonitis, pelvic cellulitis, and the like (PAGET, BLOT, LEISHMAN, l. c.) With regard to the existence of albuminuria as a ground of prognosis, it is to be noticed that under ordinary circumstances the habitual presence of albumen in the urine is looked upon as symptomatic of very serious disease; and clinical experience shows that we have only too good reason to look forward, in such cases, with gloomy anticipation as to the future—except in cases which arise in the course of scarlatina, in which prognosis is vastly more favourable. Hence the importance of the question as to "what is the nature and exact import of albuminuria when it is observed for the first time in the course of pregnancy." The question in fact is,—"Are we to consider the albuminuria of pregnancy as indicative of serious disease of the kidneys? or are we, on the other hand, to look upon it as an exceptional symptom in pregnancy, and one to the disappearance of which, after delivery, we may confidently look forward?" (LEISHMAN, l. c., p. 257.) The peculiar condition of the constitution of the pregnant female must be remembered:—(1.) The albumen in the blood is diminished during pregnancy; (2.) albumen exists in the urine in more than 20 per cent. of pregnant women; and in the case of primiparæ the percentage is considerably higher even than this. Albuminuria therefore, under these circumstances, is not of the same pathognomonic significance with that which occurs independently of gestation. If it was so, the mortality of pregnancy and childbed would be very greatly augmented. Hence, it must be admitted that the albuminuria of pregnancy is, comparatively speaking, an innocuous disease. A large proportion of the cases are unattended by any marked symptoms, and run their course, it may be assumed, without the nature of the case being so much as suspected; and experience has fully shown that in the majority of cases in which albumen is actually detected in the urine during gestation, the general health is little, if at all, affected; and the normal constitution of the urine is restored within a short period after delivery (LEISHMAN, l. c., p. 258). Hence the ordinary course of an uncomplicated case of albuminuria in pregnancy must be regarded from a favourable point of view. But it is the intimate association which exists between convulsions and albuminuria which renders such cases extremely anxious ones,—to the demonstration and elucidation of which the late Professor Sir J. Y. Simpson so much contributed. The instances in which no serious kidney lesion exists, constitute happily the great majority of those in which, at some period in the course of a pregnancy, albuminuria makes its appearance for the first time. In such cases the albumen usually disappears shortly after delivery. But in others, just as after scarlatina, it may persist for many months. If renal degeneration exist, a microscopical examination of the urine will aid prognosis—demonstrating the absence or the presence of tube casts and their characters. Headache, sickness, and the various forms of digestive disorder, so frequently associated with pregnancy, are, under the influence of albuminuria, greatly aggravated (Leishman, l. c., p. 260). If the unfavourable symptoms which accompany albuminuria continue unchecked, the general health of the patient will deteriorate—a deterioration which will make itself manifest by the anæmia and waxy pallor which are so characteristic of the more advanced stages of Bright's disease.

In cases that are to end favourably, the body-temperature falls as the fits cease and the coma diminishes.

Treatment.—If *œdema* with albuminuria and tube casts in the urine are observed during the course of pregnancy, the morbid condition may be kept in check, and the extension of the disease may be prevented by hygienic management and medicinal remedies. The watery and otherwise deteriorated condition of the blood is to be ameliorated by a liberal and properly assorted dietary, and by ferruginous tonics; and a good general effect is often produced by the use of tepid and vapour baths (Cazeaux, Leishman). It has also been suggested by Frierichs that *tartaric acid, benzoic acid,* or *lemon juice* should be regularly given, with the idea that these will prevent decomposition of the urea in the blood, or neutralise the carbonate of ammonia already formed. The functions of the bowels must be regulated with care. Purgation as a prophylactic must be resorted to with caution. *Compound jalap powder,* in electuary, with or without *guaiacum,* are the most efficient and least depressing medicines for this purpose. The quantity and the microscopic appearances of the urine furnish the best indications as to the necessity for the use of diuretics. When exudation has taken place into the Malpighian capsules and the tubes, it is desirable to clear them of the obstruction, and prevent its reformation. The copious use of diluents may effect this desirable result. But if the secretion of urine be scanty, and uræmic intoxication threaten to come on, the secretion of urine must be increased. The acids already named, and the mineral waters of Seltzer and Vichy, are the remedies best adapted for this purpose (Braun, Leishman). I find also frequently repeated small doses of *bicarbonate of soda* combined with *spirits of nitrous ether,* in large quantities of cold barley water, an efficient diuretic in such circumstances. Pills of *tannin* and *extract of aloes* may be used for restoring the normal tone. Labour should only be provoked when the symptoms are such that the life of the mother is in danger (Braun, Leishman).

In the treatment of the fits, procedure is different according as they happen during pregnancy, during labour, or after delivery. During the paroxysm, the indications are the same in all cases. As to blood-letting, there are certain cases in which the constitution and temperament of the woman, the violence of the attack, combined with evidence of vascular tension within the cranium (such as throbbing carotids, marked suffusion of the eyes and face), warrant us in believing that by venesection we give the patient the best chance of recovery, or in such cases leeches may be freely applied to the temples and ice to the head. In cases which do not present these indications for blood-letting, the administration of chloroform, ether, and other anæsthetic agents, produces a beneficial effect. Symptoms which suggest the approach of a first seizure, or a repeated paroxysm, are a sufficient warrant to adopt this method of treatment. It is the premonitory symptoms which must be held in control by anæsthetics,—their use is too late when the respiration is impeded. *Chloral hydrate* has also been strongly recommended, and it has this further advantage, that "when it is pushed further it produces an anæsthetic effect, under the influence of which a woman may be delivered without experiencing the slightest suffering" (LEISHMAN). In ordinary sedative doses—not more than thirty grains—its effect is safe, and in most cases efficacious; but if larger and repeated doses are required, its cumulative action is such that very alarming symptoms are occasionally produced, and death has even been the result of what might be considered quite an ordinary dose. Four drachms have been administered by enema within three days. Sponging with warm water, or tepid vinegar and water, has been found useful. *Opium* is not to be administered in cases where albuminuria is present. (For obstetric details, see LEISHMAN'S *System of Midwifery*, 3rd Edition, p. 776.)

SPASM OF MUSCLE, FUNCTIONAL SPASMS.

LATIN EQ., *Spasmus Musculorum;* FRENCH EQ., *Spasme;* GERMAN EQ., *Krampf;* ITALIAN EQ., *Spasmo.*

Definition.—*An affection characterised by pathological contractions of muscles, some painful and some not, but manifested only during the exercise of certain voluntary or instinctive movements* (DUCHENNE).

Pathology.—It is difficult to identify any anatomical lesion in connection with such functional disturbance, which some believe is associated with trifling and transient lesions of the nerves and their sheaths. Some regard them as a kind of rheumatic affection, and attributable to hyperæmia, with œdema of the neurilemma (NIEMEYER). These muscular spasms are apt to occur during the course of, or to succeed convalescence from acute or chronic disorders, such as *typhus, enteric fever, malarious fevers, Bright's disease,* and *epidemic diphtheritis;* and it is argued that because these diseases have a pernicious effect upon the assimilation and nutrition of the tissues, that the spasm is therefore probably idiopathic. Common cramps from fatigue are the result of some such slight derangement of the nerves as have been noticed.

Such *spasms, cramps,* or *hyperceneses,* occur in various situations, more or less limited to certain muscles or groups of muscles.

(*a.*) *Spasm of Facial Muscles,* sometimes called *mimic spasm of the face,* or *convulsive tic,* is generally due to some noxious influence acting on the surface of the face itself, such as *cold, contusions,* or other *lesions;* but whether the influence is direct or reflected is unknown in most cases. Sometimes the spasms are thus far reflex, that they seem to arise from the irritation of such remote organs as the rectum or intestines from parasites, or the uterus in *hysteria,* or diseases of that organ. Sometimes the spasm is confined to the eyelids, or to the muscles of the ear, or to the lips, a lip, or portion of a lip. When fully expressed, in the words of Romberg, "grimaces occur, either intermittent or constant, involving one side of the face, and more rarely both sides. In the former case they consist chiefly of elevation or depression of the *occipito frontalis* muscle, corrugation of the eyebrows, blinking and closure of the eyelids, twitching and sniffling of the *alæ nasi*, and drawing up and down of the corners of the mouth. These attacks set in suddenly, and as suddenly subside, to recur with equal suddenness at short intervals. In permanent tonic contraction of the facial muscles, the furrows and hollows in the affected side of the face are deeper, the tip of the nose, the commissure of the lips and the chin are drawn to the convulsed side. The muscles are hard and tense, and impede motion so much that one eye cannot be so completely closed as the other" (quoted by NIEMEYER).

(*b.*) *Spasmodic Wry-Neck, spasms in the region of the spinal accessory nerve* are sometimes assigned to twists of the neck, cold, or disease of the vertebræ. The spasms are either of a tonic or clonic character, and expressed in the muscles supplied by the nerve—*i.e.*, the *trapezius* and *sterno-cleido mastoideus;* and according as one or other muscle is more affected, so are the movements of the head in one direction or another. The paroxysms generally cease during sleep, and the clonic spasms develop in a slow and gradual manner. The affected muscles present increase of electric irritability and electric sensibility (Dr. RUSSELL REYNOLDS). It is an affection of adult life in either sex. When the muscles on both sides are similarly affected, the movement is that of incessant nodding (salam convulsions), an instance of which occurred in a soldier at the Invalid Depot at Fort Pitt, Chatham, in 1861; and about the same time and place another soldier had lateral spasms, so that the head rolled constantly from side to side, except when asleep, and the occipital region was soon bare of hair by the incessant rolling on the pillow. These movements do not commence till the head and neck have become permanently twisted, and they are entirely beyond control. Duchenne also gives other examples of this affection; such as when the biceps and deltoid contract painfully on any attempt to use the hands, as in the spastic disorders of shoemakers, tailors, and the like; and to the case of a fencing master who, when he placed himself in certain attitudes of his art, was seized with sudden rotation of the arm inwards and violent extension of the forearm. Dr. S. Weir Mitchell has also called attention to another class of cases nearly related to these, but differing from them in several important particulars. They consist of three classes:—(1.) Those in which the functional activity of a muscle or set of muscles gives rise at times to an exaggeration of the motions involved naturally, and sometimes also to more or less spasmodic activity in remoter groups; (2.) those in which the functional action of one group results only in sudden and possibly in prolonged spasmodic acts, tonic or clonic, in remote groups of muscles not implicated in the original movement; (3.) those in which standing or walking occasions general and disorderly motions, affecting the limbs, trunk, and face, giving rise to a general and uncontrollable spasm without loss of consciousness. He gives examples of each. The first class is rare, but some cases of writer's cramp nearly fulfil the conditions. But in them pain or sense of fatigue is generally superadded, and the overaction is not merely in excess, but almost inevitably involves in-coordination. A watchmaker in the constant habit of picking up and adjusting tiny screws, continuing to pick, he found at last that his finger and thumb became fast "locked" upon one another, by a spasm much stronger than his normal power of pressure between fingers and thumb. They were forced open with great difficulty. This "locking" would take place ten or twelve times a day, and last from ten to thirty minutes. These spasms came sooner if he placed himself in the habitual attitude for work; but were delayed if he stood on his feet to work. There was no pain, and only at times a tremor. All treatment failed. Nothing but rest aided him, and at last he retired from his work to reside on a farm in the country. In other cases of this "lock spasm" Dr. Weir Mitchell has seen benefit from the use of the induction current, combined after a time with hypodermic injections of *atropia,* also the combined action of *gelseminum* and the *bromides,* in full doses. Voluntary motions may thus give rise to various forms of spasm, so also may morbid sensation; or an over-excited state of motor centres, or even a normal sensation, may occasion spasm, as in tetanus (*American Journal of the Medical Sciences,* Oct., 1876). Dr. Poore has come to the conclusion that all such forms of spasm are due to abuse of the implicated muscles, which become "tired out" and degenerate into a condition of chronic fatigue or irritable weakness. He shows also that, contrary to the general belief, the affected muscles are absolutely weaker than their healthy fellows; and that their electrical irritability is diminished.

(*c.*) *Idiopathic Cramp of Muscles of the Limbs* is not uncommon as a form of rheumatism among children, and is sometimes the result of cold or of reflex intestinal irritation. The calves of the legs are the most frequent seat of cramps, which often awake the patient suddenly by their pain, and the desire to grasp the limb or press the foot against a firm resistance.

(*d.*) *Spasms of the Muscles of the Bladder* frequently arise from the irritation of foreign bodies in that cavity, such as *calculi;* and the combined conditions lead to some organic lesion. The causes of spasm of the bladder have been arranged by Romberg into *cerebral, spinal,* and *reflex.* Mental emotion, such as will induce the "goose skin," through the sympathetics, will, under like circumstances, also induce violent contraction of the *detrusor urinæ,* with intense inclination to pass water, and even to the extent of passing it, so that individuals have been known to micturate from terror or sudden fright. *Vesical spasm* is generally of reflex origin, and induced by such conditions as irritation of the urethra, as by introduction of a catheter,—by irritation of the *rectum,* as from anal fissure or hæmorrhoids,—or irritation of the womb.

Treatment in all these cases must be directed towards the removal of the probable cause, and improvement of the general health (see next topic).

SCRIVENER'S PALSY.

LATIN EQ., *Paralysis Notariorum;* FRENCH EQ., *Crampe des Écrivans;* GERMAN EQ., *Schreibekrampf;* ITALIAN EQ., *Crampo degli Scrivani.*

Definition.—*Morbid excitement of the motor fibres of the nerves of the muscles of the fingers and thumb holding the pen, resulting in cramps, so as* *completely to prevent writing, and thus the condition becomes equivalent to a local palsy or paralysis.*

Pathology.—(*a.*) *Causation.*—A want of co-ordination of the muscular movements engaged in writing seems to be the first pathological element in this affection. In this respect it approaches in form to locomotor ataxy, where there is partial loss of controlling power. The constant practice of the same movements sets up some irritation producing pain.

The disease is not entirely limited to the operation of writing. Shoemakers, milkmaids, or milkers of cows, goats, and ewes, nailsmiths, musicians, compositors, saddlers, sempstresses, and men who handle small hard articles with considerable muscular grasp, are subject to similar cramps. Hence the disease is known under a variety of names—as *cobbler's spasm, milker's spasm, nailer's spasm, writer's cramp.*

One theory regarding the production of the cramps implies that they are reflex through the excitement of the *muscular nerves* or *muscular sense.* Hence, holding the hand even in the *attitude* of writing, although it does not grasp a pen nor touch the paper, will induce the spasms.

On the other hand, it is believed that the irritation is similar to what takes place in the convulsive movements of chorea and stammering.

The affection is much more frequent among men than women, and between the ages of thirty and fifty years. It chiefly occurs among professional penmen, clerks, teachers, and those who habitually write monotonously according to prescribed and constantly repeated forms—as in mercantile books and the like details of form in business. It rarely is seen among shorthand writers, whose brain is so constantly occupied with the subject in hand that attention to the mechanical process of writing seems in abeyance. Nor is it seen among judges, or lawyers, or professional men who also write much, but with little attention to the mere mechanism of writing—so much so, that the writing of many such men is hardly readable, or at least may be legible only to a few when accustomed to read the writing. An eminent judge is said to have had three different hands of writing—one he could read himself, and no one else could read; another that his secretary or clerk could read, and which he could not himself read again, nor anyone else; and the third that neither himself nor his clerk, nor anyone else could decipher. These results were not due to writer's cramp or spasm of any kind. The hard pen, the formal writing, and the mere mechanism of the art, seem to be the conditions tending to induce the characteristic spasms. In seventy-four cases of impaired writing power and writer's cramp analysed by Dr. G. V. Poore, the cases fell naturally into six groups:—(1.) Paralytic; (2.) spasmodic; (3.) degenerative; (4.) neuralgic or neuritic; (5.) writer's cramp; (6.) anomalous cases.

In the analysis of these cases Dr. Poore calls the fact prominently to notice, that the ulnar nerve supplies thirteen and a half out of the eighteen intrinsic muscles of the hand. Its integrity is very necessary for writing power.

"Since the ulnar nerve supplies thirteen and a half out of the eighteen intrinsic muscles of the hand, its integrity is more necessary than that of any other nerve of the hand for all delicate manipulation, especially writing. The spasms which affect the hand, and which are particularly prone to follow attacks of hemiplegia, owe their character, if not their origin, to a faulty antagonism (due to a secondary paralysis or paresis) among the muscles of the paralysed limb. Although it is commonly received that such spasms are due to disturbance of the grey cerebral matter, it is well to look also to the peripheral aspects of the question. Provided a nervous impulse issuing from the brain be distributed in a limb to equally irritable muscles which mutually antagonise each other, it is difficult to conceive that spasm of definite form should be produced; but, should the equilibrium of antagonisation in the limb be destroyed by a secondary lesion, the production of definite spasm is easily conceivable, especially when voluntary control is lessened by a lesion of the central ganglia. In some cases of localised spasm, there is no evidence of central change, and it is theoretically possible that the action of a disordered centre on a healthy periphera, and the reaction of a disordered periphera to a healthy centre, may be identical in their results. Loss of writing power is often the first and most prominent symptom of degenerative change occurring in the spinal cord or brain.

"The neuritic or neuralgic group is characterised by a painful and tender condition of the nerves of the limb, which may be induced solely by overwork, but more frequently by a strain or similar injury, combined with exposure to cold and a depressed state of health. Any attempt to use the arm either for coarse or fine acts produces fatigue, pain, and neuralgia. It is not always easy to distinguish these cases from true 'writer's cramp,' and, indeed, there cannot be said to be any hard and fast line between the two groups; but it is characteristic of the neuralgic group that—1. The symptoms involve a wider area; 2. the symptoms are sometimes induced without excessive exercise of any function; 3. nerve-tenderness or neuralgia is a prominent symptom.

"In the group of true 'writer's cramp,' considerable care is necessary to detect peripheral evidence of mischief; but in every case of impaired writing power which Dr. Poore had seen, there had been evidence more or less marked of derangement of one or more of the muscles used for writing. This evidence consisted of—1. Obvious failure to use certain muscles efficiently, either for writing or for some other less complicated act; 2. the occurrence of consentaneous movements or tremor when certain muscles were put in action; 3. depressed or exalted electric irritability; and, 4. the occurrence of sensory derangement or nerve-tenderness. The muscles which are most frequently involved are those of pen-prehension rather than those of pen-movement. Reviewing the cases as a whole, certain points are important in diagnosis—1. The inferences which may be drawn from an inspection of the handwriting; 2. joints found to be implicated, the joint affection being rheumatic, neuropathic, gouty, or due to strained position; 3. a difficulty in writing is not very infrequently hereditary, or developed very early in life; and that, 4. any evidence of involvement of the nerve centres is decidedly rare. 'Writer's cramp' has been spoken of as a disease of 'faulty co-ordination,' and there can be no doubt that such is the case, for it is evident that the muscles used for writing fail to work orderly together. We are not, however, justified in assuming the existence of a special co-ordinating centre for the controlling of the act of writing. The existence of such a centre appears improbable, for the following reasons:—1. A case of writer's cramp has never been seen without peripheral evidence of change; and in the majority of cases there is no evidence of any change other than peripheral. 2. If there be a co-ordinating centre for writing, it must be created, as it were, by education. The co-ordination of writing, which we are many years in acquiring, must be distinguished from those co-ordinated movements (such as the symmetrical movement of the two eyes) which are wholly independent of education. The fact that no two people hold their pens exactly alike, and that it is scarcely more difficult to write with the toes than with the fingers, is much against the probability of the existence of a writing centre. 3. Writer's cramp is never suddenly established, as aphasia sometimes is. 4. It is almost certain that a purely peripheral lesion may cause all the symptoms of writer's cramp. 5. The fact that the left hand (if used for writing) sometimes suffers as well as the right, is no evidence that the change is central. In previous writings, Dr. Poore had spoken of 'writer's cramp' as a 'fatigue disease,' and he is still inclined to adhere to the word 'fatigue' as a convenient expression for an easily recognisable and familiar condition, regarding the pathology of which we are uncertain. Occasionally fatigue is the expression of hyperæmia or mild inflammation of a motor nerve, and the same condition may be produced either by overwork or by accidental causes, such as cold, strain, 'rheumatism,' or injury. Fatigue especially attacks those muscles which are subjected to prolonged strain; and it is probable that the relative frequency of 'writer's cramp,' as compared with other professional ailments, is due to the fact that prolonged strain of certain muscles (those which hold and steady the pen) is inseparable from the act of writing. Finally, as to the position of 'writer's cramp' in the catalogue of diseases, Dr. Poore would feel inclined to class it with neuralgia; that is, with a disease the phenomena of which are purely local, but which we recognise as being due not only to conditions affecting the sensory area involved, but also to molecular change affecting any part of the sensory fibre, whether before or after its junction with the nerve-centre" (*Med. Ch. Soc.*, Feb. 12, 1878).

Symptoms.—The indications of the disease commencing are quite painless, beyond a feeling of tension or stiffness in the muscles of the arm and forearm, or fingers only of the hand. There may be a feeling of fatigue of the hand, and a tendency to grasp the pen more firmly, especially towards the end of the day's work, which are generally relieved by a night's rest. It is not till the disease is fully expressed that convulsions or spasms occur. Movements become unsteady by the thumb being drawn towards the palm, while the index and middle finger become contracted with more or less rigidity. Every attempt to write calls forth uncontrollable movements in the thumb, the index finger, and middle finger, so that the pen starts up and down on the paper. The handwriting ceases to be legible—a mere scrawl results, or grotesque interrupted scribbling. The more the patient persists in the attempt to write, the more does the difficulty of steadying the hand and using the pen increase. The visible and sensible contractions of the muscles of the thumb and fingers are soon followed by similar contractions of the forearms, even extending, in some cases, to the upper arm. Apprehensive attention to the subject, and dread of the occurrence of spasm, are generally sufficient to ensure a paroxysm after the disease is well established. The hand and arm seem capable of every other combined movement except that of writing; and when all attempts at writing cease, then the spasms also subside and entirely disappear. On the other hand, the more the patient attempts to continue writing, so much the more violent does the spasm become.

Treatment.—Complete rest from the usual mechanical use of the hand must be insisted on. Nourishing food, with a milk diet in abundance, or cod-liver oil, are essential. In one case division of the muscle in which the cramp seemed first to commence had a good effect (STROMEYER). In fourteen days after subcutaneous section of the long flexor of the thumb, the patient was again able to write. Dieffenbach often repeated a similar operation, but without success. These may be regarded as very radical surgical methods of securing absolute rest to the wearied muscles. Mr. Solly, in his lately published surgical experiences, described several cases of this peculiar affection, in which tonic treatment, with rest from writing, was attended with good results. Niemeyer has been successful with the galvanic current. He applied the current to the muscles of the thumb and index finger. Exercise of the affected muscles materially tends to increase the disease.

As to medicines, when the disease has not gone beyond the sensation of heat and cramps in the ball of the thumb, I have seen good results from the *syrup of the phosphates of iron, quinine,* and *strychnia;* and generally those remedies noticed under the subject of anæmia may be prescribed. Mechanical appliances, such as contrivances which fill up, as by a ball, the palm of the hand on which the fingers rest in writing, have been of service; and there are also appliances by which writing may be accomplished without the aid of the fingers, and which therefore may be of service in securing rest to them.

LARYNGISMUS STRIDULUS—Syn., SPASM OF THE GLOTTIS; SPASMODIC CROUP; CHILD-CROWING.

LATIN EQ., *Laryngismus Stridulus*—Idem valent, *Spasmus Glottidis, Angina Spastica, Clangor Infantium;* FRENCH EQ., *Spasme de la Glotte;* GERMAN EQ., *Millarsches Asthma*—Syn., *Krampf der Glottis, Spasmus Glottidis;* ITALIAN EQ., *Spasmo della Glottide.*

Definition.—*A paroxysmal spasmodic disease in which the muscles of the glottis are contracted, the vocal cords tightly stretched and so approximated that the glottis is for a time partially or completely closed. In the latter case there is arrest of the respiratory movements and apnœa. In the former case there is stridulous inspiration and dyspnœa. The affection depends upon some morbid excitement of the* par vagum *and its nerve centres, direct or reflex, and is usually seen in infants, especially during the period of first dentition, and in children before the completion of the second year; and in the great majority of cases between the fourth and tenth month. It is also seen in adults.*

Pathology.—(*a.*) *Causation.*—There are two points which require special notice:—(1.) "That the immediate cause of the phenomena of the stridulous inspiration and apnœa, is spasm of the adductors of the vocal cords; and, (2.) that whatever may be the exciting cause of the attack, whether it be the irritation of teething, or the presence of indigestible food or worms in the intestinal canal, &c., in all cases it acts through an altered state of the nervous centres. The facts which point to an alteration in the brain substance (whether recognisable or not) are first, that both sides of the body (both vocal cords) are affected; secondly, that the various alleged causes (such as dentition, indigestion) are not only often in operation without the production of laryngeal spasm, but that when they do give rise to that symptom, they necessarily act through the brain; thirdly, that frequently other admitted symptoms of cerebral irritation, such as the carpo-pedal contractions, are present. That the phenomena of the disease depends on spasm of the adductors of the vocal cords is probable from the sudden onset and paroxysmal character of the disease, and by it being frequently accompanied by other symptoms of a spasmodic nature, such as carpo-pedal contractions. Paralysis of the abductors of the vocal cords, the result of pressure on the pneumogastric or recurrent nerves, never causes such complete closure of the glottis, unless the impaired innervation be associated with induced or accidental changes in the mucous membrane; and in the paralytic affection, the dyspnœa is a constant, not a varying symptom, as in laryngismus" (MORELL-MACKENZIE).

"A rachitic condition of the bones of the skull has frequently been noticed. Out of ninety-six cases of laryngismus examined by Lederer, there was craniotabes in ninety-two. The experience of Sir William Jenner and Dr. Wiltshire is of a similar character. It has been suggested that the thinness of the cranial bones in rickets allows pressure to be exercised on the brain in the occipital region, when the child lies on its back (ELLÄSSER); but it is more probable that the rachitic dyscrasia is accompanied by morbid changes of a nutritive character in the structure of the brain itself.

"Scrofula has also been regarded as an active predisposing cause of the disease (MARSH). Sometimes an attack is brought on by tossing the child in the air, and it still more often comes on in sleep. These facts have been adduced by some as an evidence of the cerebral nature of the disease; but it must be remembered that both in sleep and in sudden movements of the body the function of respiration, not less than the cerebral circulation, is modified, and that the spasm of the glottis may originate in either process. Disease of the cervical portion of the spinal cord sometimes gives rise to it (MARSHALL HALL). In cases of disease of the brain or medulla, external pressure applied over these parts has been known to cause laryngismus. Hydrocephalus exists in some cases; and mental emotion—especially terror and rage—occasionally gives rise to the spasm."

There is ample evidence that *cerebro-spinal* irritation is the more immediate cause of the disorder. This has been shown especially by Dr. Marshall Hall. The irritation which establishes the reflex excitement consists mainly of three kinds :—(*a.*) The irritation of teething, through the *trifacial nerve.* (*b.*) The irritation of the gastric intestinal tract by overfeeding or improper food, through the *pneumogastric nerve.* (*c.*) The irritation of constipation, diarrhœa, or intestinal obstruction, acting through the *spinal nerves;* the action of cold on the skin; and the irritation of a chronic skin affection.

These act through the medium of the *spinal marrow*, and influence—(1.) *The constrictor of the larynx*, through the *inferior or recurrent laryngeal nerve;* and, (2.) the *motions of respiration*, through the *intercostal* and *diaphragmatic* nerves.

Indirectly, the condition is excited by the passage of liquids into the larynx in sucking or swallowing, or from slight catarrh of the larynx.

Congenital predisposition exists, as in many families almost all the children in succession are affected (ROMBERG). Children brought up by the bottle, and in towns, are also said to be more liable to the disease than children brought up in the country, and suckled at the breast.

(*b.*) *Morbid Anatomy.*—The *bronchial glands* and the *thymus* gland were observed long ago, by Peter Frank, to be enlarged in this disease. Since then the disease has been sometimes referred to the *thymus* and sometimes to the *bronchial gland enlargement*, by Hood, Marsh, Ley, J. H. Kopp, Caspari, Paganstecker, and Hirsch. These morbid conditions act through pressure on the *par vagum* and *recurrent branch* of the *inferior laryngeal* nerve, the *superior laryngeal* remaining unaffected. These enlargements, however, are now known not to be constant, and never cause complete closure of the glottis.

Symptoms.—The age of the patient destroys the value of subjective symptoms, but those of an objective character are sufficiently pronounced. The following is a common mode of onset. A child put to bed, apparently in its ordinary state of health, wakes up suddenly at midnight with difficulty of breathing, inspiration being accompanied by a crowing noise similar to that heard in croup. After a few of these stridulous inspirations, the frightened child bursts out crying, and in a few minutes is fast asleep again, as if nothing had occurred. But all cases do not commence in this abrupt way. The parents may have noticed that for a few days previously, the child has been peevish and fretful, that its appetite has fallen off, or that there has been a slight "catch" in the breath. The first attack most frequently occurs in the evening or night, but not necessarily so. "The next day the child may be quite well, and there may be no further return of the symptoms; but it often happens that another attack comes on about the same hour the following night. The second attack is generally more severe than the first, both in its character and duration. In severe cases, indeed, the paroxysms are of a most urgent kind and of the most frequent occurrence. A severe fit of laryngismus may be thus described: the breathing suddenly becomes greatly embarrassed, each act of inspiration being much prolonged and accompanied by a harsh stridor: suddenly the sound ceases, the glottis is completely closed, and the respiratory movements of the chest are suspended. The flush which first lit up the countenance gives way to pallor, and afterwards to lividity. The eyeballs roll, the veins of the neck are turgid, the fingers close on the thumb, which is bent in the palm, and the hands are flexed on the wrist. Spasm likewise affects the feet, the great toe is drawn away from the other toes, and the foot is flexed and rotated slightly outwards. These so-called 'carpo-pedal' contractions are probably sometimes accompanied with great pain." Sometimes the affection commences by these spasms of the fingers and toes preceding the crowing inspiration. Dr. Craigie mentions a case in which these symptoms preceded the laryngeal phenomena for weeks before a paroxysm of spasmodic breathing took place. The duration of the disease varies from three or four weeks to several months. The duration of the fit lasts from a few minutes to ten or a quarter of an hour; and several fits may take place in the course of a day, after which a week or more may pass without their repetition. It is in very bad cases that the fits repeat themselves frequently, and in which the general convulsions are apt to occur, and the case to prove fatal. Even when a child has remained for months without a paroxysm, there is always a tendency to relapse. During these fits of suspended respiration, there is great fear of death, and the child may really seem dead, till after a period of about two minutes the characteristic gasp is followed by the thin stridulous breathing.

On stripping the child it is seen that during each inspiration the epigastrium and intercostal spaces sink in instead of expanding as they should do; and on listening to the chest it is found that very little air enters during inspiration. Notwithstanding the severity of the paroxysm just described, the patient may survive it, the apnœa being succeeded by stridulous breathing, and by relaxation of the spasmodic contractions of the feet and hands; but when the symptoms are of the dangerous character just described, the paroxysm is sure to be quickly followed by others—in one of which the child dies. The severity of the attacks varies between the mild paroxysm which has been described as occurring at the commencement of the disease, and one sufficiently intense to cause death. The spasm is characterised by its sudden onset and by its complete remission, and as a rule by the entire absence of fever, and by the progressive severity of the spasm, as regards recurrence, duration, and intensity. In some cases there is a certain amount of febrile disturbance, when the spasm has been more prolonged but less intense. The temperature in such cases is often as high as 99·5° or 100° Fahr., but rarely higher. The urine both during and shortly after the attack, contains a small quantity of albumen, especially if the embarrassment to the breathing has been of long duration. Some of the associated symptoms of laryngismus may be present, such as hydrocephalus, a rachitic condition, or enlargement of the thymus body.

Diagnosis.—It is the sudden, violent interruption of respiration by spasm which is pathognomonic of the seizure. There is no cough, and no hoarseness—the *par vagum* alone is affected; so that *croup*, or *laryngeal catarrh*, is not to be confounded with *laryngismus stridulus*. The distinctly intermittent nature of the affection, and, as a rule, the absence of febrile disturbance, differentiates it from true croup, and its own distinctive characters from all other diseases. Sometimes, however, there are difficulties of diagnosis; the practitioner may be called suddenly to see a child gasping for breath, and the bystanders may be too excited or too stupid to give an intelligent history. One should therefore never neglect to take the temperature of the body, or to examine the fauces for false membrane.

Prognosis.—The prognosis depends on the character of the paroxysm and its supposed cause. The cases mainly dependent on reflex causes (dentition or irritation of the alimentary canal) generally do well, whilst those due to direct pressure and those mainly caused by cerebral irritation are more frequently fatal. Thymic asthma is especially dangerous, and if there is evidence (such as considerable enlargement of the gland) to show that the spasm is of that character, the most unfavourable opinion must be given. The length of the intervals between the paroxysms is a good prognostic guide—the longer the intervals the better the chance of recovery (MORELL-MACKENZIE). More than 300 children in England and Wales die yearly from this affection, of whom about 200 are infants under a year old.

Treatment.—Two important indications have to be fulfilled in carrying out the treatment—viz., the relief of the paroxysm, and the removal of the morbid condition, whether local or general, which gave rise to it.

> "The immediate treatment generally falls to the nurse or mother. The little patient should be raised and placed in a sitting posture, and then he may be slapped on the back, cold water may be dashed in the face, and *ammonia or strong acetic acid* held to the nose. These measures are often successful by giving rise to violent expiratory actions; but remedies calculated to relieve spasm are equally successful. The warm bath may be used and emetics given directly there is a sign of the stridor —when the paroxysm is on, the child will not drink. A favourite remedy in Germany, and one that is highly successful, is tickling the fauces with the finger or a feather until vomiting is produced. Depressing enemata, such as tobacco, have likewise been recommended, but their use is attended with considerable danger. The ordinary rules for the treatment of disease apply here; that is to say, gentle remedies should be used in mild cases, and those of a more powerful character in dangerous ones. Putting the lower part of the child's body in a hot bath and dashing cold water in its face, is a simple and sometimes successful plan. The inhalation of *chloroform* is a very valuable remedy, but of course must be used with great care, and cannot safely be employed by non-professional persons. If the child appears to be sinking from the apnœa, the trachea must, of course, be opened, and artificial respiration resorted to. Indeed this should even be adopted by the practitioner should he arrive shortly after the apparent extinction of life. Some practitioners recommend the use of antispasmodic remedies (whether animal, vegetable, or mineral) between the fits" (MORELL-MACKENZIE).

Where the exciting cause of the attack can be traced to dentition, the gums should be lanced; if the affection should appear to be due to intestinal derangement, means must be taken to remove any source of irritation from the alimentary canal, and for this purpose perhaps the best remedy is a small dose of castor-oil. Thymic enlargement should be treated by the application of leeches (according to the patient's age and strength), and afterwards by counter-irritation. As regards the constitutional state predisposing to the disease, suitable treatment will be found detailed in the various articles in these volumes which treat of scrofula, rickets, hydrocephalus, parasites, and the like. Spasm of the glottis has hitherto been treated as an infantile disorder, but it sometimes occurs in adults. It is usually met with in women, and is commonly regarded as hysterical. In these cases, sedative inhalations (which cannot well be used in the treatment of children) generally give prompt relief.

During the intervals of paroxysm means must be taken to prevent its recurrence. These mainly consist of attention to the state of the alimentary canal. A searching purgative is generally called for to clear out the contents of the bowels—*calomel*, *jalap*, and *castor-oil* are the most useful agents. *Belladonna*, to the extent of one-sixth of a grain thrice daily, combined with *bromide of potassium* or of *ammonium*, may be of use. Sinapisms and linaments to the spine are also of use. The diet ought to be carefully regulated. Change of air is generally called for, and the tepid and cold bath must be in daily use.

SHAKING PALSY.

LATIN EQ., *Paralysis Agitans;* FRENCH EQ., *Paralysie Tremblante;* GERMAN EQ., *Paralysis Agitans;* ITALIAN EQ., *Paralisi Tremula.*

Definition.—*Involuntary tremors, continuous and permanent, with lessened muscular power, agitating various members increasingly, allowing no peace; and while purposed movements may exaggerate the shaking, repose does not cause its disappearance, and sleep alone puts a temporary stop to the spasmodic agitation of the affected members. The affection may commence in the hands, arms, or head, and gradually extend over the whole body.*

Pathology.—In making a distinction between different kinds of *tremor*, Charcot mentions that they may be divided into two groups:—one, in which tremor is in some sort *permanent;* another, in which tremor only supervenes on purposed movements. *Paralysis agitans* illustrates the former; the *tremors* which attend *disseminated sclerosis* illustrate the latter, when the tremors only appear under certain conditions. This distinction is of the highest importance in the analysis of the history of chronic diseases of the nervous system accompanied by tremors. *Paralysis agitans* was first described by Dr. Parkinson in 1817, under the name of "Shaking Palsy;" and Charcot has most recently collected all the facts relating to it.

(*a.*) *Causation.*—(1.) External causes are noted, such as the influence of violent shocks to the nervous system—fright, terror, the sudden communication of bad news, &c.; (2.) influence of prolonged exposure to moist cold; and, (3.) the irritation of certain peripheral nerves supervening in consequence of wounds or contusions. Of *predisposing causes*, it has now been noted that it is not always a senile disease. Although it usually sets in after forty, cases are cited in which it began as early as twenty years of age. It is found as commonly in males as in females. The actual causes remain unknown; but Charcot assigns the following:—(1.) *Damp cold*, such as arises from a prolonged sojourn in a badly ventilated apartment, or in a low dark dwelling on a ground floor. (2.) *Acute moral emotion.*—The disease is generally one of advanced life (those especially who have passed their fortieth or fiftieth year), and progresses slowly—so slowly that cases within my own knowledge have continued during the past thirty years with little or no change. It is not, however, to be regarded as quite a senile disease. Duchenne relates the case of a patient who was a lad of sixteen. The intellect remains unimpaired, and it is only when the movements become so constant as to prevent sleep and lead to exhaustion that any danger is to be feared. Extension of the disease is marked by deglutition and mastication becoming difficult, by the walk becoming unsafe from tripping up of the feet. The constitution begins then to suffer, and general paralysis with coma finally prove fatal. It is a state of decay.

(*b.*) *Morbid Anatomy.*—No important visceral lesion is found after death, such as are often found in disseminated sclerosis or progressive locomotor ataxia. M. Charcot has described two kinds of lesions which he has found. In the first kind the following morbid appearances were constant:—(1.) Obliteration of the central canal of the spinal cord from germination of the epithelial elements which line the ependyma; (2.) germination of the nuclei which surround the ependyma; (3.) pigmentation of nerve cells, most marked in Clarke's column chiefly. Of the second kind of lesion, two of the three cases had a multiplication of the amyloid corpuscles; and in the third case there was a patch of sclerosis on the posterior surface of the *bulbus rachidicus.*

Symptoms.—Besides the phenomena stated in the definition, it is to be noted that the lessened muscular or motor impotence appears to be due in part to the rigidity which prevails in the muscles. Frequently also, at an early, but usually at a late period, the patient loses the faculty of preserving equilibrium whilst walking; or there is a tendency to propulsion or to retropulsion. Without feeling any giddiness, the patient is (in the first case) propelled forward, and, as it were, compelled to adopt a quick pace. He is unable, without extreme difficulty, to stop—being apparently forced to follow a moving centre of gravity. With these phenomena there are also associated a peculiar attitude of the body and its members, a fixed look and immobile features. Although the march of the disease appears to be slow, it is nevertheless progressive; and the fatal term arrives either by the advance of age, or because of intercurrent diseases, such as pneumonia, which may be accidental or the result of marasmus, or mere confinement to bed, death taking place from a sort of nervous exhaustion, in which nutrition degenerates, the patient cannot sleep, and bed-sores form.

The disease commences in one of two ways—either slowly and progressively, or in an abrupt manner. In the majority of cases the invasion is insidious, the tremor being limited to the thumb, the hand, or the foot. This trifling symptom may long remain solitary, and presents certain characteristics. If the hand is attacked, its several parts oscillate over each other. The patient "closes the fingers on the thumb, as though in the act of spinning wool; at the same moment the wrist is bent by rapid jerks upon the forearm, and the forearm on the arm—the tremors being merely passing and transitory, breaking out when least expected, the patient at the same time enjoying complete repose of mind and body, while the tremors frequently occur without his being conscious of their occurrences. As the disease augments in intensity, the tremor invades little by little the parts which have remained sound—affections of the feet following those of the hand. The tremors are commonly confined for a long time to the members of one side (*hemiplegic type*), or to the two lower extremities (*paraplegic type*). Sometimes tremor is not absolutely the first symptom. It may be preceded by a feeling of fatigue; or by rheumatoid or neuralgic pains, which are occasionally most severe, occupying the member or region which shall soon be seized by the convulsive agitation. An *abrupt invasion* of the disease is usually the consequence of some moral cause, such as terror. It may then occupy one member only, or in all the limbs at once; and after a series of alternate improvements and exacerbations—from one to three years—it becomes permanent. When perfectly developed the movements are almost incessant in severe cases; but the intensity is not the same at all times. Moral emotions and voluntary movements augment it; and paroxysms of movement occur spontaneously, without appreciable cause. Natural sleep, and sleep induced by chloroform, always annihilate the convulsive jerking for the time being. The tremors are characterised by rhythmical and involuntary oscillations, as of different parts of the hand, recalling the appearance of certain co-ordinated movements; as when the thumb moves over the fingers, as when a pencil or paper ball is rolled between them; or movements more complicated, as when a piece of bread is being crumbled to pieces. M. Charcot considers these peculiarities as specially belonging to the tremor of *paralysis agitans*, not to be found in any other species. The handwriting also exhibits characters which are somewhat special—great irregularities, for instance, in the thickness and heaviness of the strokes, with irregularity and sinuosity of the lines, especially in the up-strokes. The head and neck usually remain unaffected. The muscles of the face are motionless, so that there is a fixity of look, and the features wear a permanent expression of mournfulness. The tongue is not uncommonly tremulous. There is no real difficulty of speech; but utterance is jerky and slow. M. Charcot further points out another characteristic to be seen in the *rigidity* to be found in the muscles of the extremities, and also in those of the neck at a certain stage of the disease. Cramps are first complained of, after which, when stiffness is established, the attitude becomes characteristic. There is a slowness in carrying out intended movements. The head is greatly bent forward and fixed. The body also is slightly inclined forward when the patient is standing. The habitual attitudes of the hands are also peculiar, simulating those of primitive chronic articular rheumatism, but wanting in the articular tumefaction and stiffness, and in the osseous deposits and crackling which are observed in nodose rheumatism. Uneasy sensations all over the body also prevail, especially an habitual sensation of excessive heat. Cramps are complained of, and a feeling of utter prostration exists, especially after an aggravated fit of trembling, even after insignificant efforts; and there is a perpetual desire for change of posture. The terminal period commences when nutrition of the muscles has begun to be impaired by fatty degeneration; and, at the same time, the mind becomes cloudy and memory is lost. General prostration sets in, urine and fæces are passed unconsciously, and eschars appear upon the sacrum. At this terminal period it is also frequently noticed that the tremors diminish, and may even disappear.

Treatment.—*Hyoscyamine* is a palliative merely, while *strychnine, ergot of rye, hyoscyamus* and *opium, nitrate of silver,* and *Calabar bean* all aggravate the tremors. The use of the continued current of a galvanic pile has been attended with success in the hands of Remak and Russell Reynolds.

CHOREA—*Syn.*, ST. VITUS'S DANCE.

LATIN EQ., *Chorea;* FRENCH EQ., *Chorée;* GERMAN EQ., *Veitztanz*—Syn., *Chorea;* ITALIAN EQ., *Corea.*

Definition.—*An irregular spasmodic convulsive action of the voluntary muscles, ceasing during sleep, of a clonic kind, especially a profusion of movements of the face in smiles and frowns, and of the trunk and extremities in gesticulations. The movements are either entirely withdrawn from the control of volition, or but little under the direction of the will. The disease is brought on by fear or emotion, or without obvious exciting cause. It has special relation to certain other nerve-disorders, also to rheumatism and to heart affections.*

Pathology.—(*a.*) *Causation.*—The history of this disease is a picture of superstition. As late as the close of the fifteenth century it does not appear to have been studied by physicians, but was supposed to depend on supernatural causes, or what was termed "demoniacal possession." In Germany it was said to have been epidemic for two centuries; and the patients (probably many of them maniacs) were wont to join in frantic dances. As late as 1673 it was the custom for them to go in procession to the church of some favourite saint, of whom St. John, St. Guy, and St. Vitus were the most in repute; hence the name of St. Vitus's Dance, by which the disease is sometimes identified. Medicinal remedies were supposed to be unavailing in such a disorder, hence the priests said masses, sung hymns, and sought by such means to exorcise the fiend believed to be in possession of the patient.

The disease frequently attacks children otherwise in good health, and without any obvious cause. When any cause is assigned, it is usually terror. In Dr. J. T. Arlidge's analysis fright stands as causing one-fifth out of 156 cases. The symptoms have been known to follow the fright in a day or two; at other times about three or even six weeks have elapsed before the disease became manifest. The causes producing this affection, however, have also been referred to mental impressions. For example, Dr. Mayo relates the case of a woman in the fourth month of her pregnancy who had a disgusting object thrown at her bosom. She continued for two months in a state of extreme nervous illness from this circumstance, but recovered, and went her full time, remarking, however, that the child was extraordinarily lively in the womb, and that she was often overcome with the sensations it produced. At birth, the child (a girl) displayed the writhing motions of chorea, and continued to suffer throughout life. When she was about thirty years of age she had the appearance of an elderly child, with a head remarkably small, and a mind hardly removed from complete idiocy.

Chorea is limited, or nearly so, to early life, and is rarely seen after twenty. It seems to be most frequent between the ages of ten and fourteen; and it is more common in the female than in the male, three-fourths of the patients being females (HEBERDEN). Dr. Rufz regards the ages from five years to fifteen as the most liable to chorea; and that girls are three times more frequently sufferers than boys. Dr. Todd's experience shows that chorea generally occurs between nine and fifteen years of age. It is a disease of childhood, greatly more prevalent at the period of *second dentition* and at the period of *puberty* than at other periods; and although symptoms somewhat resembling those of chorea are sometimes seen in adults, and at more advanced ages, still such cases are exceptional. It may even be questioned whether they are due to exactly the same morbid condition as that which gives rise to the ordinary clonic convulsions of early life. Before the sixth year the disease is rare, and is equally uncommon after the age of fifteen. In a careful analysis of 156 cases of chorea by Dr. John T. Arlidge, Physician to the North Staffordshire Infirmary, the results as to age and sex were confirmatory of the general statement that chorea is especially a disease of girls under puberty. His analysis showed a rapid rise in the numbers affected after the eighth year is attained, and this reaches its maximum in the course of the tenth year; it then falls as rapidly till the sixteenth year, when an increase again, of smaller amount, is met with, and is maintained almost to the same extent in the following year, after which cases are few. It is also worthy of note that the cases among girls of fourteen and upwards comprise a very considerable proportion of relapses; and that not a few of them are attended with evident nerve lesions, accompanied with heart disease; or, they are of reflex origin, as from uterine disorders or pregnancy. In certain cases an hereditary tendency to the disease has been observed (NIEMEYER); but the results of Dr. Arlidge's analysis show that the "predisposition is very rare." The mimic influence of example must not be lost sight of as a cause, especially in the epidemics of chorea, which are apt to prevail in boarding schools, just as they occurred in the Middle Ages; and, lastly, reflex causes, such as *irritation from worms in the intestines, onanism, pregnancy*;—each must have due consideration in all inquiries as to the causes of chorea. Much evidence has been brought forward in favour of the humoral or rheumatic character of the disorder. Dr. Copland has the merit of having been the first to indicate the complication of chorea with that class of diseases, (*London Medical Repository*, Vol. XV.) His views have been subsequently confirmed by Drs. Pritchard and Roeser, and more recently by the more elaborate researches of the late Dr. Begbie, of Edinburgh, and Dr. See. Numerous instances have also been adduced by Andral, Bouillaud, Bright, Mackintosh, Watson, R. B. Todd, Kirkes, and others, in which diseased conditions of the heart and pericardium have been attended with, or have given rise to, spasmodic diseases of the nature of *chorea, paralysis, mania*, or *dementia*; and the evidence of these writers is amply sufficient to prove that a considerable number of individuals affected with *chorea* have suffered from *cardiac* or *synovial rheumatism* previously. Dr. Sidney Ringer has observed one or two cases of chorea with considerable elevation of the temperature, but without any of the ordinary evidences of *rheumatism*. He believes such cases may tend to show that even in those in whom there has been no evidence of previous rheumatism, it nevertheless probably existed, but was latent in respect of all the symptoms, except elevation of the body temperature. But it is unquestionable that all have not so suffered. The presumed blood-condition, similar to that which exists in *rheumatism*, can only thus be regarded as one of many occasional causes, the real essence of the disease being a perverted nervous function, the exact sources or morbid anatomical changes to account for which are still not exactly determined.

Choreic patients are nearly always in low health. They are often anæmic, and generally of emotional or excitable nervous temperament, with feeble nerve-power. The disease is more prevalent among patients engaged in the pottery manufactures of North Staffordshire than among the other inhabitants of the district. In about one-seventh of the number examined by Dr. Arlidge *rheumatism* or *rheumatic pains*, or *heart-disease*, was found to have affected one or both parents; *consumption* also appears to have been as frequently a family inheritance; and the same may be said of nervous disorders among immediate relatives. Of the number of patients in whose cases the fact was ascertained, *rheumatism* had occurred previously to the chorea in about one-fifth; but it would seem, as far as inquiry was made, that *scarlatina* was about as frequent an antecedent as rheumatism. Distinct evidence of cardiac lesion was found in rather more than one-fourth of ninety-two cases in which it was sought for and noted. In thirty-seven of these, which were the most recently collected, it was discovered only in one-fifth. Impaired nerve-power, either congenital or acquired, must be admitted to exist in all the subjects of chorea. The condition will oftener be congenital, the children being born delicate—*i.e.*, born with impaired powers of resistance to any injurious impression. When such children become *anæmic*, we get the double condition out of which chorea springs; then add *rheumatism*, *indigestion*, some say *worms;* or add *fear*, or *emotion*, and when lesions of *irritation* are set up, the balance of nerve power is destroyed, and we get chorea (Dr. ANDREW CLARK, *Brit. Med. Journ.*, Oct. 21, 1876).

As to assigned causes, sudden fright and previous *rheumatic* and *choreic attacks* severally are productive of the disease in a like proportion—namely, *one-fifth* each. Of the remaining two-fifths, *reflex irritation, antecedent epileptic fits*, and *injuries to the head*, were made accountable for a considerable proportion; the cause being unknown or unassigned in the rest.

As to a classification of the cases, Dr. Arlidge's analysis shows—(1.) A group such as are met with in children up to the age of puberty, and in most of whom the disorder exhibits itself in its simplest and highly curable form. In this simple form, rheumatism, cardiac and cerebral lesion, do not constitute an element; but there are exhaustion, anæmia, a disturbed nervous condition, and defective nutrition always present. This form best deserves the special name—chorea. (2.) A group connected with the occurrence of rheumatism, and with cardiac lesions traceable thereto. This does not mean that the chorea is due directly to embola in the great ganglionic centres of the brain, although it may probably be due indirectly to them, and to their paralysing and inhibitory action upon the controlling power of the cerebral lobes. (3.) There appear grounds for believing (although further investigation is necessary to establish the relation and its frequency), that there may be a group of *scarlatinal cases* of chorea, parallel with the rheumatic, and probably nearly or often having concomitant cardiac lesions. Henc

Dr. Arlidge's analysis does not admit of the doctrine that chorea is always of cerebral origin. (4.) Clinical experience certainly indicates a not uncommon spinal origin of cases of chorea—namely, such as are attributable (a not inconsiderable proportion) to reflex causes, such for example as the chorea of pregnancy (Arlidge, *B. M. Journ.*, p. 799, Dec. 8, 1877.)

Cardiac affections are apt to supervene in cases of chorea, differing in kind as well as in degree, namely,—(1.) Rheumatic endocarditis, or pericarditis, resulting in organic change; (2.) Functional derangement and cardiac murmurs, due to an impoverished condition of the blood (Romberg); (3.) A chronic affection of the heart itself, "evidenced by the existence of a systolic murmur at the left apex, which cannot be referred to inflammation or organic change of the mitral valve, which has not the usual accompaniments of a hæmic murmur, but which does seem plausibly ascribable to disordered action of the muscular apparatus connected with the valve" (Walshe). The late Dr. Kirkes showed, from *post-mortem* evidence, that particles of fibrine from the heart's valves might be carried to the brain vessels, causing plugging or embolism of one or more of them; and he also argued that chronic symptoms might, in some cases, be produced thereby. These were the next important discoveries and steps in the elucidation of the pathology of chorea.

(*b.*) *Morbid Anatomy.*—The morbid appearances of the body which have been observed more recently in cases of chorea, and recorded especially by Drs. W. H. Dickinson, Broadbent, Bastian, Hughlings Jackson, Charcot, Jaccoud, Trousseau, Ogle, Barnes, Wilks, Moxon, Bristowe, Stephen Mackenzie, and John T. Arlidge (of the North Staffordshire Infirmary), have recently cleared up many points regarding the pathology of this disease, which were left in darkness or obscurity since the time of Sydenham, Cullen, Rostan, Bright, Stoll, Pinel, and others, who had frequent opportunities of examining cases of chorea. These early investigators failed to detect any morbid appearances other than those which were commonly seen in affections of the brain and spinal cord. Accordingly, various views have been entertained regarding the pathology of chorea, which may be classed as follows:—(1.) By some pathologists chorea has been regarded as a disorder entirely functional or dynamic, and independent of organic change—a belief rapidly being set aside, as facts relative o the existence of specific lesions are accumulating from more exact and better methods of observation. (2.) It is believed by some, and not without good reason, that the blood, at all events in some cases of chorea, s primarily diseased, or secondarily becomes diseased constitutionally, the precise nature of the change being as yet unknown. (3.) Associated with ome other diseases, whose pathology is better known, chorea has been egarded either as a concomitant feature, or as a consequence of their revious existence: such, for instance, as *rheumatism* and *disease of the heart.* 4.) Pauses in the muscular movements of chorea occurring during sleep, nd from the action of chloroform, render it probable that the perversion f motor influence is derived from the brain rather than the spinal cord.

The structures which most obviously manifest disordered action during fe are the nerves and muscles; and for the following reasons we are led o believe that they are maintained in their disturbed and excited state by some morbid condition of the *central parts of the brain, and not of the spinal cord,* either directly or by reflex action:—(1.) *Clonic spasm,* of the incessantly repeated character peculiar to chorea, is not a phenomenon of persistent spinal irritation; while *tonic spasm* is a mark of such a condition (REYNOLDS). (2.) The movements can generally be, in some measure, controlled by the Will, unless they are very severe; and even then they are so controlled to some extent (REYNOLDS). (3.) The spasmodic contractions cease during sleep, whereas phenomena of an excito-motor character are increased by the removal of Volition. Fixing the attention also on some other object likewise diminishes the intensity of choreic movement (REYNOLDS). (4.) The special occasions of increase or of induction of choreic movements are the attempts at volitional action and emotional changes (REYNOLDS). (5.) The phenomena of chorea during life, in accordance with the views expressed by Drs. R. B. Todd and W. B. Carpenter, tend to refer the exciting cause of the disease to changes going on in the central ganglia of the brain, such changes being expressed in a healthy state through "Volition, Perception, or Emotion, or the balancing and co-ordinating of movements." (6.) Experiments on living animals, and observations in morbid anatomy, tend to prove that injury to the *optic thalami* is productive of considerable disturbance to the movements of the body. (7.) An opportunity was afforded me, when Pathologist to the Glasgow Royal Infirmary, of examining carefully a case of acute chorea, which terminated fatally after a most violent attack, the acute symptoms lasting ten days. The result of the examination showed decided changes in the *corpora striata* and *optic thalami,* as indicated by the following observations:—

> "The specific gravity of the *corpora striata* and *thalami optici* was different on the two sides of the brain. The *central ganglia* on the right side were of the specific gravity of 1·025, those on the left side of 1·031; and this difference appeared from the hydrostatic experiments, as well as from those made with the gravimeter, confirming in some measure the accuracy of the general result. The vascularity of these central parts of the brain, when compared with the grey matter of the spinal cord (which was healthy), was so well marked as to leave no doubt of its abnormal increase. Microscopic examination confirmed the existence of increased vascularity, for numerous capillary vessels, in unusual abundance, existed in every section examined. Some of these were irregularly dilated, as in a varicose condition, and were filled to a greater or lesser extent with the red corpuscles of the blood. [Phenomena of embolism or of thrombosis?] The amount of granular substance in these parts of the brain, on both sides, appeared to be greater in proportion to the fibrous substance than in the same parts of healthy brain with which I compared them" ("Contributions to Pathology," *Glasgow Med. Journal,* No. I., 1853).

The late Dr. R. B. Todd remarked, with reference to these facts, that "further observations on this subject are greatly needed, and will no doubt throw much light on the pathology of chorea and other allie affections." Accordingly, since that time a most complete account of th morbid changes found in chorea has been given by Dr. W. H. Dickinso and others, and the opinions elicited in the discussion from Drs. Broa bent, Hughlings Jackson, and others, have greatly contributed to set the pathology of chorea in a clearer light (*Med. Chir. Society*, Oct. 12, 1875). Dr. Dickinson describes the morbid appearances found in seven fatal cases of chorea, in the order of their acuteness, those being placed first in which death ensued earliest. A summary of the state of the nervous centres in each was enough to show that the changes were remarkably constant in *kind* and in *place*. *In kind*, the changes were all directly connected with vascular disturbance. The injection was general to vessels of every kind, but most marked in the arteries throughout the substance of the brain and cord. When the sources of exudation or of hæmorrhage could be determined they were always arterial; the degenerations were usually periarterial, and the spots of sclerosis similarly placed. The first visible change would seem to be injection or distension of the arteries, this being succeeded by exudation of their contents to the irritation or injury of the surrounding tissue. *In place*, the changes affected both brain and cord—the cord perhaps the more severely. Whether in brain or cord, the changes on the two sides were generally symmetrical, both being often affected at the same spot, in the same manner, and nearly to the same extent; and in instances where no symmetry was apparent, a tendency to it could be traced, as in the existence of vascular distension on one side corresponding to hæmorrhage on the other. In the cord the changes attained their maximum in the posterior and lateral parts of the grey matter, and in the upper portions. To elucidate some of the points regarding arterial repletion as the origin of the pathological changes, and the phenomena of embolism or thrombosis, the clinical history of the disease was represented by a tabulation of twenty-two fatal cases (all examined after death), and seventy cases under observation during life only. The prominent fact brought out by these cases was the frequency of mitral endocarditis in connection with the disease. This existed in seventeen out of the twenty-two fatal cases, while there was stethoscopic evidence of it in forty out of the seventy non-fatal cases. It was thus shown that endocarditis was associated with chorea independently of the frequent origin of the chorea in rheumatism, and when the nervous disturbance could be definitely traced to mental causes. Thus, while in rheumatic cases endocarditis often preceded the chorea, it was equally evident that chorea in other circumstances preceded the endocarditis. This duplex relation between the nervous and the cardiac disorders showed that chorea was not in any way—neither embolically nor otherwise—a result of endocarditis, though often associated with it, as having a common origin in rheumatism; while endocarditis was continually a result of chorea, set up possibly by the muscular irregularity of the heart, and the mitral regurgitations thereby occasioned. In conclusion, chorea is regarded by Dr. Dickinson as depending upon *a widely spread hyperæmia of the nervous centres*, not due to any mechanical mischance, but produced by causes mainly of two kinds, one being the rheumatic condition, the other comprising various forms of irritation (mental and reflex) belonging especially to the nervous system. Given the irritant—mental, reflex, or rheumatic—the course of the disease had been sufficiently traced in hyperæmia and its results.

Hence Dr. Dickinson concluded that the embolic theory is inconsistent with the character and distribution of the morbid appearances. The spots of perivascular change were widely scattered through that large region which lies inferiorly to the *cerebral convolutions*, between the *corpora striata* and the lower part of the *cord*—the district of the *motor* and *sensory*, as distinguished from the *mental* functions. The result, chiefly in muscular excitement rather than in paralysis or loss of sensation, was associated with the character of the lesions, which were points of irritation calculated not so much to cut off as unnaturally to excite nervous function. In looking at the nature of the lesions, proceeding as they do from vascular distension to perivascular change, it was not possible but to connect them with those of a large group of nervous disorders, prominently with those of diabetes. In diabetes the changes are of similar origin and character, though usually seen at a later stage, and attended with more excavation. They are, however, somewhat differently disposed in the two disorders: in both they are widely distributed—in diabetes the stress falling upon the brain, in chorea upon the cord. Every period of life has its own regions of nervous susceptibility: in childhood, the motor; in adolescence, the emotional; in advancing years, the mental, and coevally, or nearly so, that part of the nervous mechanism which instigates glycosuria. Much the same mental impression may make a child choreic, a girl hysterical, or a man diabetic; and thus, both in external origin and in the nature, though probably not in the site of the organic changes, we see resemblances and alliances between nervous disorders which in their symptoms betray little similarity.

On the other hand, Dr. Broadbent puts a different interpretation upon Dr. Dickinson's facts as to the seat of the change in the nerve-centres which give rise to the choreic movements and the nature of the lesion. While Dr. Dickinson considered the pathology of chorea to imply a generally distributed hyperæmia of the nervous centres, special prominence being given to its existence in the cord; Dr. Broadbent holds that chorea is a *symptom* of change (possibly of various kinds) in a particular part of the nervous system—the *corpora striata*,—such change stopping short of destructive lesion such as would give rise to hemiplegia. That, in fact, just as the common form of hemiplegia is due to structural lesion of the *corpus striatum*, which might be from hæmorrhage, embolism, tumor, &c., so chorea is due to changes in this ganglion of slighter character—capillary embolism, or Dr. Dickinson's hyperæmia, and the like. Accompanying chorea there are other symptoms of disorder of the nervous system, partly intellectual, partly emotional, partly referable to the functions of voluntary muscles—delirium, imbecility, aphasia (or at least speechlessness), and impaired sensibility. These are not by any means always proportionate to the violence of the movements. When these symptoms occur in association with chorea, Dr. Broadbent attributes them to similar change in other parts of the nervous centres to that which in the *corpora striata* give rise to choreic movement, referring thus the chorea to the *corpora striata*, the delirium and imbecility to the *hemispheres*, the *aphasia* to the *third frontal convolution*, the impaired sensation to the *thalami*. The impairment of sensation is best shown in *hemichorea* when the sound side could be used for comparison; but in severe general chorea sometimes, when the skin is worn into holes by the continued friction, there is little evidence of suffering. The phenomena of *hemichorea* Dr. Broadbent considered to establish conclusively that the choreic movements were not due to changes in the cord. It was inconceivable that these changes could involve one-half of the cord along its entire length, and be strictly confined to this half. Again, when sensation is impaired, as is not uncommon, the impairment is on the same side as the movements; whereas, if the spinal cord were the seat of the lesion, sensation and motion ought to be affected on opposite sides. Further evidence against the spinal cord as the source of the irregular movements exists in the fact that tickling does not provoke unusually violent reflex action, as might be expected were the cord in a condition of undue excitability. Positive indications of the *corpora striata* as the seat of the change to which the choreic movements are due are the following:—The striking parallelism between *hemichorea* and *hemiplegia,* as regards the affection of the face, arm, and leg, but especially in the fact that the parts of the body which escape paralysis in hemiplegia are bilaterally affected in hemichorea. The bilateral association of nerve-nuclei which brings bilaterally associated muscles into relation with both *corpora striata,* permits these muscles on the paralysed side in hemiplegia to be reached by stimuli from the uninjured *corpus striatum,* and also permits them on the sound side in *hemichorea* to be affected by the irregular discharges from the affected *corpus striatum.* Dr. Broadbent also refers to a case in which there was, first, chorea affecting one side of the face and the corresponding arm and leg; then that side of the face was paralysed, the choreic movements continuing in the arm and leg; and finally, as recovery took place, the chorea returned in the face. Here an aggravation of a condition which gave rise to chorea produced paralysis, and the only interpretation the facts would permit of was, that the hemiplegia of the face and the hemichorea of the limbs were due to change of the same kind and in the same situation, which could only be the *corpus striatum.* Another case was one in which injury on one side of the head gave rise to pain in the head, peculiarities of conduct and temper, hemichorea on the opposite side, and double optic neuritis. Dr. Dickinson's cases might also be shown to support the conclusion that the choreic movements had their origin in the *corpora striata,* and not in the cord. In the first case the chorea had been comparatively slight, while the hyperæmia of the cord was marked. In the last case, again, the patches of sclerosis were mainly in the corpora striata, and in all the cases the ganglia at the base of the brain were much affected. As to the nature of the change, Dr. Broadbent had only had one *post-mortem* examination of chorea following acute rheumatism, and then capillary embolisms were found in great numbers, especially in the vessels of the *corpora striata.* Many cases have, however, been recorded.*

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* In the case of acute chorea dissected by the author at Glasgow, in 1853, the fact is noted that "the capillary vessels of the central ganglia were irregularly dilated, varicose, and filled with the red corpuscles of the blood." The phenomena of embolism and thrombosis were not well known at that time (twenty-seven years ago), but now such a description would be certainly suggestive of thrombosis.

There are also the positive observations of Tuckwell, of Oxford, and Bastian, as to the phenomena of embolism. Dr. Bastian found plugging in three cases, most likely by the process of thrombosis. He also believes, however, that in some cases there is no organic change: and there are other facts supporting the doctrine. Hence the question is still an open one; and clinical investigation one of the best means of investigating the problem. Dr. Hughlings Jackson is of opinion that there are three distinct lines of evidence in support of the hypothesis of embolism:—(1.) Embolism is just the pathological process which would theoretically account for the production of that condition of the nervous tissue which must exist in such a disease as chorea—a condition in which the nerve-tissue is "over-acting," or "excitable," or "unstable": there are frequent nervous discharges. This over-expenditure implied increased nutrition; and significantly, the process of embolism is one which leads to increased afflux of blood in those organs (brain, retina, &c.) which have "terminal" arteries. In some cases of plugging of a branch of the retinal artery, hæmorrhages are to be seen by aid of the ophthalmoscope in the early stage of the embolic process. (2.) There is the evidence of clinical facts. The frequency of rheumatic fever and of heart disease shows that there is often a condition for embolism, or for thrombosis, in persons who have chorea. If a child of from five to twelve, and the subject of heart disease, or one who had only had rheumatic fever, were to become hemiplegic without convulsion or loss of consciousness, the diagnosis of embolism would be made by most physicians. Even if there were no history of rheumatism and no heart disease, embolism or thrombosis would be the most likely explanation of most cases of hemiplegia in the child. The exceptional occurrence of chorea in adults contributes further evidence, seeing that most of the patients are puerperal women—a significant fact, because hemiplegia in such persons is mostly owing to arterial plugging. (3.) There is the evidence from facts bearing on localisation. The difference of opinion as to the localisation of changes in chorea is mostly as to whether they are seated in the cord, or in the higher parts of the nervous system. About twenty years ago, Dr. Russell Reynolds brought forward evidence to show that the *corpus striatum* was the seat of disease.* Dr. Hughlings Jackson has since held that clinical facts point to the conclusion that either this centre, or (as he thought more probable) the convolutions adjoining it, was in fault in chorea; that hemichorea was one of the motor analogues of that form of hemiplegia which was dependent on destruction of the *corpus striatum.* The same parts suffered most in the two affections; moreover, they suffered and also recovered in the same order. Hemichorea is also often associated with weakness of the parts affected, and very strikingly it occasionally degenerated into complete hemiplegia, "resembling hemiplegia from a decided lesion of the brain"

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* Before that time Dr. Aitken's case of acute chorea, published in the *Glasgow Med. Journ.*, clearly demonstrated lesions in these central ganglia. That they were of the nature of thrombosis there can be no doubt, and are in harmony with the subsequent hypothesis of Dr. Hughlings Jackson, when he wrote in 1864,—"*I think from many circumstances, that embolism is a frequent cause of chorea. I do not say plugging of the trunk of the middle cerebral, but probably of some of its ramuscles which supply convolutions near to the* corpus striatum" (*London Hosp. Reports,* Vol. I., p. 459).

(TODD). There are also cases of post-paralytic choreal movements *following* hemiplegia (WEIR MITCHELL and CHARCOT). With regard to the counter-hypothesis, Dr. Todd (who believed the pons Varolii was diseased in chorea) said that the hemiplegic nature of the affection was inexplicable on the supposition that its seat was in the cord. The affection of the face in chorea was left unexplained. That the spinal cord is a centre of co-ordination of movement is not denied; but the question is, "Does it serve in such co-ordinations as are seen to be elaborate even in the disorderly movements of chorea?" The nature of the movements in chorea (a profusion of real movements—smiles, frowns, and gesticulations) was strong evidence that the part diseased served in highly special and complex co-ordinations, and thus that it was a part very high up in the nervous system. In no other affection are such special and complex movements seen. (See Report of Discussion on Dr. Dickinson's Paper, October 23, 1875.) In connection with embolism, it is further of interest to note that its occurrence in the central artery of the retina is an extremely rare result of the endocarditis, which is generally found (*post-mortem*) to be associated with the disease in severe cases. Dr. Gowers notes only two instances on record. In one, the embolism occurred at the time of the commencement of the chorea, and was in the left eye; the choreic convulsions being most severe on the left side. The state of the heart is not mentioned. The other case was not seen till some time after its occurrence. The patient, a child, had suffered from chorea for some years; and, during the chorea, had lost the sight of one eye. The disc was atrophied, and the arteries very small. Dr. Gowers considers it probable that double optic neuritis, slight in degree, may in rare instances occur during the height of the chorea, just as it does in other diseases attended with intense functional disturbance of the brain. Dr. Hughlings Jackson records such a condition in a case of right-sided hemichorea. The discs were hyperæmic, badly margined; the veins large and irregular. These appearances increased with the chorea, and disappeared with recovery. Dr. Gowers has found optic neuritis only in one case of chorea; the change was then due to albuminuria. Bouchet has figured white exudations on and about the disc in a case of severe chorea—third attack (*Medical Ophthalmoscopy*, by Dr. GOWERS, p. 156).

Nevertheless, there are several objections to each of the hypotheses mentioned in explanation of the pathology of chorea:—(1.) In a series of cases published by Dr. Ogle there was no evidence of rheumatism; (2.) cases have died and have left no evidence of embolism or of thrombosis. (3.) In cases where thrombosis or embola have been found, may they not (as suggested by Dr. Andrew Clark) have been but a factor in the fatal issue? (4.) Can the phenomena of chorea coming on instantaneously be accounted for on the theory of embolism? (5.) Drs. Wilks and Moxon, in their extensive experience, have examined many cases of chorea after death, and have been unable to record any constant morbid appearances in the nervous system.

No constant morbid appearance can thus as yet be given as an explanation of the pathology of all cases of chorea. In some there may be evidence of *thrombosis* (AITKEN, BASTIAN); in others evidence of embola

(TUCKWELL, HUGHLINGS JACKSON, STEPHEN MACKENZIE), vascular phenomena (resembling embola, regard being had to the size of the arteries plugged) (DICKINSON). But while Drs. Barnes, Dickinson, Bristowe, and others reject the hypothesis of embolism or of thrombosis as a cause of chorea, one fact stands prominently forward regarding the localisation of lesions in this disease—namely, the constancy with which they have been found in the *central ganglia*, or convolutions in immediate connection with them.

Symptoms.—Chorea principally consists in singular and involuntary movements of the whole muscular system, which prevent the patient from being able to lay hold of any given thing with certainty; or to carry that object, be it a spoon or a glass, with any certainty to his mouth, or to any other place. (1.) As to voluntary movements:—If the patient is asked to touch (say) her nose with a finger, or to hold out her arm, she can do so, but with a jerky and uncertain manner. There is still power of the will over movements, and yet movement asserts itself sometimes in spite of the will. Thus, the face-muscles twitch, and express indifferently smiles, grins, or grimaces, the neck is suddenly and capriciously pulled to either side, fingers are straightened or clenched, the trunk and extremities execute more or less violent gesticulations. The muscles of the face and neck are sometimes seized with this species of convulsion, when the head is not only tossed about and the mouth contorted into the most singular grimaces, but it may require two or three persons to feed the patient—one or more to hold, and another to watch the proper moment to pop the food into the mouth. Sometimes the motor nerves of the fifth pair are affected, and then the jaw closes with a loud snap, or the articulation of the voice is affected, or the effort of swallowing may be difficult. (2.) As to associated movements, as in walking, the gait is unsteady, the limbs are thrown about, and the power of co-ordination is deficient, (Dr. ANDREW CLARK, l. c.) The lower limbs are generally as much affected as the upper, and the patient can with difficulty walk in a straight line, or if he does, it is always by a series of movements which tend towards the object, counteracted by another series which altogether diverge from it,—his feet turning in and out, upwards and downwards, in every possible direction.

Thus the essential phenomena of chorea are motorial, consisting of spasmodic involuntary contractions of the muscles. These have been classified as,—(1.) *Clonic spasms,* of great frequency, unattended by pain, resembling the restless movements of a child who has been irritated or put out of temper. Such spasms occur independently of any attempt at voluntary movements, and are in slight cases almost unobserved. (2.) The patient is agitated by all sorts of odd motions, and has often a vacancy of countenance which gives him a fatuous appearance (Dr. RUSSELL REYNOLDS). These symptoms are constant during the day, but during sleep they generally cease altogether. They affect both sides as a rule, and in a very few cases one side only. The patient is then said to labour under *hemichorea.* The child's health may appear to be good, his pulse natural; and his bowels, though occasionally constipated, are by no means uniformly so, but for the most part act regularly. The spasms are generally increased by emotion. The heart acts irregularly, probably owing to the anæmic state generally associated with chorea. Dr. Addison describes a bellows murmur, often mitral, but sometimes aortic, and probably due to the same cause.

These phenomena are developed so gradually, that in most cases the disease is not recognised till it has made some progress. The symptoms reach a certain point of intensity, and remain at such a point for a variable period. Premonitory symptoms are neither frequent nor characteristic; but a certain susceptibility to nervous disturbance, and irascibility of temper, are not uncommon. General ill-health is usual, arising from various causes, as delay of menstruation, and constitutional morbid states, such as *rheumatism,* or the existence of some other general disease. The commencement of the symptoms is also sometimes sudden. They may consist at first simply of restlessness, or of hurried and somewhat clumsy movements. The left side and the upper limbs are frequently affected first (hemichorea); but subsequently the whole body is involved. The malady, in half the number under observation, affected both sides of the body; of the remainder, one side was affected more seriously than the other in two-thirds; and in the rest it was sufficiently one-sided to be called hemichorea. In more than one-half of the cases, the muscles of the face were involved. In one-sixth, the speech was affected.

Dr. Walshe (Emeritus Professor of Medicine of University College) was the first to call attention to a not unfrequent occurrence in the course of chorea—namely, a high specific gravity of the urine, which is most marked where the choreic movements are most active; and no doubt it indicates increased waste of tissue, consequent on the disturbed state of the muscles and nerves (*Lancet,* Jan. 27, 1849, p. 85). In the acute case of chorea so carefully recorded by Dr. Walshe, four phases were observed in the characters of the urinary discharge:—*First,* During the first five days it was "*febrile;*" that is, of high specific gravity, deep brownish-gold colour, strong urinous odour, and depositing lithates in abundance. *Second,* There came a period during which a great excess of urea gave a special character to the fluid, while alternating improvement and recrudescence marked the course of the chorea. This superabundance of urea is, in the present state of our knowledge, referred to the muscular waste entailed by the constant convulsive movements. *Next,* there appeared oxalates in the urine passed on the twenty-sixth day, on which day the improvement in the case was so marked that the child might have been considered convalescent. *Subsequently,* an abundant precipitation of phosphates took place, the indubitable result of previous nervous waste. These observations, originally made by Dr. Walshe, were subsequently confirmed by the late Dr. Todd, and by Dr. Bence Jones. Generally speaking, the density of the urine has been found highest in those cases in which the movements were most general and most active, and falling steadily with their diminution, and with the restoration of a greater controlling power on the part of the patient. But no positively general rule exists regarding the urine. Its variations are great in different patients, and at different times in the same patient.

There are still some points in the clinical history of chorea which require notice; especially some to which Dr. W. R. Gowers has called attention. These are—(1.) As regards *the irritability of nerve and muscle*—a point which can only be determined in cases of hemichorea in which the irritability of the unaffected side can be taken as a standard for comparison. An examination of such cases shows that a few weeks after the onset of the affection, there is to be observed in most, though not in all the cases, *a distinct increase in the irritability of both nerve and muscle on the affected side, the increase being made evident both by faradisation and the voltaic current.* The difference varied in the cases examined from one to three *centimètres* of the secondary coil of Stöherer's larger induction apparatus, and from two to five cells of the Stöherer or Leclanché voltaic battery. The change was always in the same direction to both faradisation and the voltaic current. The increase in irritability was found to subside with the subsidence of the chorea. The existence of these changes in hemichorea justifies the inference that they exist in cases of general chorea; and are to be regarded as the expression of a change in the nutrition of the nerve-fibre, in connection with the grey matter of the cord, probably on the nerve-cells of the anterior cornua, with which they are connected, and of which they are in effect the processes. "Indeed, it is probably not too much to assert that, in testing the irritability of the motor-nerves, the nutrition of which is unaffected by any local cause, we are really testing the state of nutrition of the motor nerve-cells of the spinal cord. *The increased irritability is, therefore, the indication of a change in the nutrition of those nerve-cells,* similar to that which is expressed by the granular and other changes lately found by Mr. Sankey and myself in these cells in the chorea of the dog. The change in their nutrition is no doubt the result of an irritation propagated down the cord upon them from the changed encephalic centre, and is similar to that which occurs in some cases of hemiplegia, from an irritative lesion in the corpus striatum; and it is an instance, if slight yet distinct, of the way in which 'functional' disturbance is attended with nutritive change" (Dr. GOWERS, *Brit. Med. Journ.*, March 30, 1878, p. 448).

(2.) *As regards the relation of the muscular disturbance in chorea to voluntary movements.*—Two elements may be, and commonly are, distinguished in the choreic disorder of movement: first, the spontaneous movement; and, secondly, what may be termed the inco-ordination of voluntary movement. A distinction of the two elements is sufficient for the statement that this inco-ordination of voluntary movement and the spontaneous spasm, although frequently proportioned, bear no *necessary* relation to one another, either when compared in different cases or even in different periods of the same case, but vary independently. This variation must depend upon one of two causes:—either the power of the will to overcome the spontaneous spasm is subject to variations altogether irrespective of the degree of that spasm; or else there is an inco-ordination to be recognised which varies independently of the spontaneous movements. It will be found that, when such a disproportion exists, the inco-ordination is in excess of the spontaneous spasm early in an attack, and the spontaneous spasm is in excess of the inco-ordination late in an attack and during relapses. The independent variation in these two elements in chorea suggests that they may depend on an affection of distinct and separate regions of the nerve-centres. It is not easy to conceive that so considerable a variation as was noted in some of the cases can be due to a variation in the affection of one region (Dr. GOWERS, l.c.)

(3.) *As to the distribution of hemichorea*—it is sometimes strictly unilateral, affecting conspicuously the arm and the leg. Not unfrequently, however, the condition of the two legs is the same, when one arm only is affected. (*a.*) The choreic movements may be confined to one arm, both legs being free from movement, although that in the arm is violent. (*b.*) When the chorea affects considerably one arm and leg, although the other arm is quite free from movement, it will generally be found, on close examination, that there is some degree of affection of the other leg. (*c.*) Occasionally the affection of the other leg is as great as that of the leg of the side on which the arm is affected. The distribution of the chorea may thus be unilateral in the arms, bilateral in the legs. No doubt the explanation is to be found in the anatomical relations which underlie the common joint action of the legs and separate use of the arms. The same thing may be occasionally noted in unilateral convulsions, in which, as in one case recorded by Dr. Gowers (*Lancet*, Nov. 6, 1875), the convulsions were the result of an unilateral meningeal hæmorrhage: the fits affected one arm and both legs equally, leaving the other arm free. This distribution of the spasm in hemichorea is thus quite in harmony with an unilateral pathological cause. In fourteen *post-mortem* examinations, out of thirty-five cases of hemichorea, Dr. F. Raymonds records that in every case the lesion was found to occupy the same region—namely, the base of the corona radiata at its posterior part, and those fibres which lie immediately in front of, and somewhat externally to, Meynert's bundle of sensory fibres, from the posterior pyramids passing directly upwards through the corona radiata to the cortex of the posterior lobe (*London Med. Record*, July 15, 1877, p. 299).

(4.) *As to the relation of chorea to convulsive seizures.*—If chorea depend on an unstable condition of the nerve-centres, it is not surprising to find that the affection is occasionally associated with other diseases which we ascribe to a similar instability of nerve-cells, such as hysterical and epileptoid seizures of various kinds, and even genuine epilepsy. The association is probably, in some cases, that of a common origin, in imperfect development or nutrition, of the nerve-centres; but, in others, it would appear as though the morbid process, constituting the one affection, entailed an instability of tissue, which resulted in the other disorder.

Dr. Gowers' cases also illustrate the frequent association of chorea and epilepsy, and several of the ways in which that association is produced. The possibility of a close connection between the two forms of spasmodic disorder is shown in those cases in which paroxysmal convulsive seizures were part of a severe attack of chorea. In them, although the spasm was in part that of chorea, so intense as to assume a convulsive character, there was also loss of consciousness, and spasm of a tonic character preceding and accompanying the choreoid convulsion. The significance of the case is that the convulsion must be ascribed to the same morbid process as that which was the cause of the chorea. In three other cases the chorea succeeded epileptoid seizures. In one, a single general convulsion occurred a month before hemichorea; in the other, unilateral fits for six months were succeeded by an attack of general chorea. In these two cases there was no relation to be traced between the distribution of the chorea and that of the convulsion. In another case the epileptic attacks occurred during almost the whole of life, and were interrupted by an attack of chorea. Both chorea and convulsions agreed in their distribution, and were most severe on the left side. No significance can be attached to the cessation of the fits during the chorea, since it is common for attacks to cease during the course of any acute disease. In still other cases, convulsive attacks succeeded chorea. In one case, chorea, worse on the right side, was succeeded immediately by right-sided convulsions; and it is, therefore, highly probable that the two were the expression of the same morbid change. In another case, permanent epilepsy of marked character succeeded, at intervals of six months, an attack of chorea; and in yet another instance, convulsive seizures commenced two years after the termination of an attack of general chorea. A similar, although rather more distant relationship, was observed in another; while in the last two cases, Dr. Gowers relates the interval between the chorea and the epilepsy was so long that probably the only significance is that of a common predisposition.

Several of these cases presented evidence of organic heart disease; and it may be urged that in this there is a common condition, which may be a cause of the two sets of symptoms. Heart disease is common in chorea, and Dr. Gowers has found that it is sufficiently frequent in epilepsy to make it probable that there is a causal connection between the two conditions. But such an explanation leaves unexplained the several cases in which no heart disease could be discovered, and the remarkable association in several of the cases between the distribution of the chorea and the convulsive seizures, which point unmistakably to the origin of the two diseases in a common pathological state of the nerve-centres.

Diagnosis.—On this point Dr. Andrew Clark observes generally as to all important cases of diseases as well as chorea, that the best mode of diagnosing any disease *is to doubt its existence*, and to proceed to question the patient in detail. With reference to chorea, question the whole muscular system in detail:—(1.) As to voluntary motion: Let the patient touch, say, the nose with a finger; let the arm be held out, for no part manifests choreic movements more characteristically than the upper extremity. Compare the movements with the monotonous tremor of palsy; they are constantly varied. Compare them with the spasmodic actions of hysteria, which are *purposive*, and are sometimes quite intermitted; those of chorea are *purposeless* and constant (during waking hours). All the segments of the arm are involved in the *purposeless* movements. The shoulder is hitched. The upper arm is moved to and from the side. The forearm goes through all the possible movements of flexion, extension, supination, and pronation; while the hands and fingers execute movements of the most grotesque and inco-ordinate description (BRISTOWE). (2.) As to associated movements: Observe the patient walking. The gait is unsteady; the limbs are thrown about; the power of co-ordination is deficient. Ask him to look so as to turn the eyes inwards. *By consensual movement, the pupils should contract.* Observe the power of swallowing, and listen over the œsophagus to notice any gurgling or sign of difficulty in the action. (3.) As to involuntary movements and electro-motility: Ascertain whether the sphincters act or no. (4.) As to reflex movements: test them. (5.) Test sensation: also the muscular sense, whether the position of the limb is known without looking for it; test the thermic sense, and change the hands about from hot to cold media, to note if readily recognised; test the tactile sensibility by needle-points (but observe that, if two be felt at half an inch on one hand and only at one inch on the other, it is not to be hastily concluded that in the latter sensation is impaired; in the former, it may be exaggerated); then test the electro-sensibility and the sense of pain. Pass on to test the special senses; then the intelligence, the power of articulation, of association of ideas; and do not omit to investigate the sympathetic. Take next the thoracic organs. Concerning cardiac *bruits*, Dr. Andrew Clark finds that, if there have been no previous heart disease, *the bruit will certainly disappear.* He speaks not only of the anæmic *bruit*, but of the one which has the characters of mitral reflux. If there is no *bruit* in the case, it may be that *you are examining when the* child is quiet. Make the patient run round the room. *Never pronounce a heart healthy until you have heard it in strong action as well as in quiet.* By this precaution you may sometimes detect commencing aneurisms; and, with all care, cases of this malady will sometimes remain undetected. One patient came to Dr. Clark complaining of ill-defined pain over the heart; many of us had seen him and detected nothing. I made him go up and down stairs, still nothing; and then I encouraged him as having nothing the matter. A fortnight later, after the same exertion, I found a faint to-and-fro sound; and the end, which was not long afterwards, revealed a small aneurism of a sinus of Valsalva. Never say, then, there is no heart disease. If there be any doubt whatever, say only, *I find no evidence* of heart disease. Next, examine the digestive organs: see if there be any source of irritation. Note the condition of the urine; its high density, first noticed by Walshe; the presence of oxalates, and of a peculiar acid; also its tendency to rapid putrefaction. Lastly, inquire into the previous history. "What else has been the matter?" Giddiness, rheumatism, measles, hooping-cough, or scarlet fever? (Dr. Andrew Clark, l. c.).

Prognosis.—The recovery of the patient from chorea, with very few exceptions, may be always prognosticated. The disease will in general gradually decline, with complete removal of the spasms. The mean residence in hospital for patients has been found to be thirty-one days (Rufz), although recovery is not complete at that time. Those cases only are apt to terminate fatally which occur during an attack of rheumatism or pericarditis, or when the disease assumes an intensely acute form, the patient losing rest at night and becoming exhausted; then emaciation progresses rapidly, and death occurs in from nine to twenty days. (See account of such a case by the late Dr. Wm. Weir, in *Glasgow Medical Journal*, No. I., 1853.) Dr. Walshe's experience leads him to believe that when the disease is slowly ushered in, it is more obstinate and enduring than when it is suddenly developed. An irregularly remittent course he regards as an inherent quality of the disease, (*Lancet*, l. c.) When of long duration the accuracy of judgment becomes impaired, and disorders of the intellect are apt to follow. The course of the disease is essentially chronic, rarely terminating before six or eight weeks, and as often prolonged for three or four months. In rare instances it becomes persistent, and lasts more or less through life—marked by remissions and exacerbations. It ought to be remembered that this tendency to relapse is a special feature of the disease, and obtains in about one-fourth of the number of cases according to the analysis of Dr. Arlidge.

Treatment.—Having ascertained a time limit to the disease we are able to judge as to the effect of medicinal remedies, observing by how much they shorten its duration. The remedies given may be classed into three divisions: (1.) *Anti-rheumatic remedies*, as *potash, iodides, bark;* (2.) *nervine tonics, arsenic, zinc, iron;* (3.) *empirical*, as *ice* or *ether over the spine, galvanism, sedatives.*

From the use of *ether* to the spine—a plan so strongly commended by a few—Dr. Andrew Clark has seen no benefit. In his experience rest and the free use of *arsenic* are the remedies which shorten most the duration of the disease. *Arsenic*, in the form of *Fowler's solution*, is also well spoken of by Romberg, in doses of three to five drops. Dr. Bristowe also is in favour of *arsenic*, given in small doses, and continued for some length of time. Experience also shows that, in most simple cases, any special treatment by drugs may be dispensed with; and that the chief matter to attend to is the general health and nutrition of the patients, which may be sufficiently dealt with by good diet, the withdrawal from causes of excitement and annoyance, and by discipline and sensible management. There are, however, various drugs which may prove valuable auxiliaries; as, for instance, *the salts of iron* when there is anæmia, and in all cases cold or tepid bathing; or the cold douche to the spine, or the shower-bath. In the rheumatic group of cases, much the same course of tonic treatment is generally applicable; any special cardiac symptoms calling for appropriate remedies. In the reflex group, the rule of action is to remove the cause of reflex irritation. Cases with high temperature and a febrile condition indicate the presence of collateral disease, only indirectly associated with choreic movements, and call for treatment directed to the removal of the organic mischief. In several cases of very severe chorea, *tartar-emetic* has been found of great service; and in many of moderate severity, *bromide of potassium* was apparently useful (ARLIDGE).

Generally, therefore, the indications for the treatment of chorea are—(1.) To remove, if possible, all morbid states of the body which may tend to aggravate the disease, such as *constipation, anæmia, amenorrhœa, worms;* (2.) By well-regulated purgative medicines to subdue any cerebral congestion; (3.) To sustain the strength and improve the vigour of the nervous system by tonic and stimulant medicines, by food, and by the cold bath. The particular tonic is not of much moment. Dr. Wood recommends the *powder of the black snake root* (*cimicifuga*), in doses of from half a drachm to a drachm, or from one to two fluid ounces of a decoction; or from one to two drachms of a saturated tincture should be given three or four times a day, and continued for several weeks, the dose being gradually increased till it produces headache, vertigo, or disordered vision. The *sulphate of zinc* has also had the credit ascribed to it of curing a large number of cases, beginning with a grain in the form of a pill, three times a day, and increasing the dose till it reaches seven or eight grains daily. The *preparations of iron* are also frequently resorted to with benefit. But Dr. Walshe did not find any of these remedies at all useful in the acute case he has described. Of all the remedies he tried, the "*extract of cannabis Indica*" was followed by the most satisfactory results. It exercised a sedative influence on the muscular action in a marked degree, and that immediately. The *Indian hemp* was given in doses of one-fourth of a grain of *the extract* thrice daily. The dose was subsequently increased to half a grain, and at the same time one-grain doses of *nitrate of silver* were administered, and a draught containing eight drops of *dilute nitric acid*, (*Lancet*, l.c.) *Nitrate of silver* he found to have no mean influence in aiding the cure. Dr. Corrigan had also previously used *Indian hemp* with much success in chorea (*Med. Times and Gazette*, 1845, p. 29; also *Dublin Hosp. Gazette*).

The tincture of Calabar beans (ʒi. to ℥i. of alcohol), beginning with twenty minims three times a day, is also recommended by Dr. J. W. Ogle; the dose to be increased by ten minims a dose, up to ʒi. The student is recommended to consult a valuable paper "On the Uses of Indian Hemp in Nervous Diseases," by Professor RUSSELL REYNOLDS, in BEALE'S *Archives.*

Trousseau and Niemeyer consider *narcotics* to be of great value in the form of large doses of *morphine;* others are of opinion that *narcotics* are not well borne in chorea.

Gentle applications of the *galvanic current* along the spine, the patient standing erect, have been successful in the hands of Benedikt. The current should be just strong enough to be felt distinctly, without pain. Chapman's ice-bags to the spine are also of use.

Chloroform has been used with benefit to control the violent movements.

The best treatment is the improvement of diet, change of surroundings, and moral discipline. Hence it is that children with chorea improve so much in well administered hospitals, where the food is abundant and discipline well maintained. But choreic patients must not be associated together, because the disease tends to be kept up apparently by imitation, especially amongst children.

Of other classes of stimuli, *camphor,* in five-grain doses, has acquired much reputation, especially after the alvine discharges have become healthy by the action of purgatives. Many young women who attribute the attack to fright, frequently get well from the simple administration of the *spirit of nitrous ether* in one fluid drachm doses three times a day, combined with the officinal *camphor mixture.* The catalogue of remedies which have been proposed is endless. In many instances, however, the above medicines may be continued for weeks without any manifest improvement. In such cases the *cold bath,* or the *cold shower-bath,* is an excellent adjuvant; and, unless the patient is suffering from some structural disease, the case uniformly yields to this conjoined treatment; great care and attention being bestowed on the regulation of the diet, which should be light, nutritious, and easily digested.

HYSTERIA.

LATIN EQ., *Hysteria;* FRENCH EQ., *Hystérie;* GERMAN EQ., *Hysterie;* ITALIAN EQ., *Isterismo.*

Definition.—*A complex disorder of all the cerebral functions, of a chronic kind, occurring mainly in females from the age of puberty upwards. It is probably associated with some morbid state of the emotional or sensori-motor centres, and presents every variety of alteration, so that the phenomena of hysteria are protean, simulating or mimicking the phenomena of almost every other disease, while the most common and characteristic features of the affection are certain motorial changes of a convulsive nature, usually of paroxysmal occurrence.*

Pathology.—(*a.*) *Causation.*—The remote causes of this affection are rather moral than physical; and in a young person predisposed to the disease almost any mental emotion will excite hysteria, as anger, disappointment, jealousy, protracted expectation, the loss of a husband, a friend, or a child; indeed, all that brings the Passions, Emotions, or Affections of the mind into play is a cause of this disease; and many women cannot go to church, or witness a tragic representation, without suffering from hysterical paroxysms.

Hysteria almost exclusively attacks females; and usually makes its appearance for the first time between the ages of fifteen (on commencing puberty) and thirty; or during that period of a woman's life when the generative functions are fully developed and in their greatest vigour. Those most liable are the unmarried or continent, and those that labour under amenorrhœa or menorrhagia. The married woman often suffers just after conception, or before parturition, or subsequently as a consequence of protracted suckling. The barren woman, however, is more liable, probably from her mind being acted upon by a greater number of exciting causes, such as disappointment in the prospect of being a mother. Taking classes of women, the higher classes, from their artificial modes of life, are greater sufferers than the lower orders. But although hysteria is a disease almost peculiar to woman, it is not entirely so, but occasionally affects the male sex under conditions of mingled debility and excitement. In them the disease does not tend to come on in early life, as it does in woman. Shakespeare makes Lear exclaim, when Glo'ster relates the cause of his being put in the stocks—

> "Oh, how this mother swells up toward my heart!
> *Hysterica passio!*—down, thou climbing sorrow,
> Thy element's below!"

The predisposition to the disease is most manifest in that peculiar condition of the nervous system for which we have no more precise or definite expression than *nervous irritability* or *mobility, nervousness* or *hyperæsthesia*—a condition which is more common in women and children than in men, and more common in all persons when in a state of weakness than when in the full enjoyment of muscular strength. In women the affection is more common about the menstrual periods, and immediately after parturition, than at other times: more common likewise among those in whom the monthly discharge is habitually *excessive*, or *altered* (as in *leucorrhœa*), or suddenly *suppressed*, or more gradually obstructed (as in the different forms of *amenorrhœa*). In this condition of mobility both Sensations and Emotions are intensely felt, and their agency on the body is stronger and more lasting than usual; continued voluntary efforts of Mind, and steady or sustained exertions of the voluntary muscles, are difficult or impossible; the muscular motions are usually also rapid and irregular. A tendency, either congenital or acquired by injudicious training, has an obvious influence upon the induction of *Hysteria.* It rarely manifests itself before the twelfth or fifteenth year of life; and, although it seldom appears in old age, it frequently outlasts the period of child-bearing.

The immediate causation of hysteria is thus very obscure; but the most important influences are emotional disturbances, sexual conditions, and occupation. Like chorea and epilepsy it is contagious in the sense that it is apt to spread by imitation.

(*b.*) *Morbid Anatomy.*—Three theories have been entertained relative to the nature of this disease, and to the primary seat of the affection:—(1.) Some, with the ancients, refer it to a morbid condition of the nerves of the uterus and organs of generation, because the disease is almost exclusively peculiar to females between the age of puberty and that of the extinction of the sexual functions, and is accompanied by morbid conditions of the sexual organs, especially *infarctions* of the womb, ulcerations of the *os uteri* and *flexions of the uterus, dermoid cysts of the ovaries, irritation of the genitals* from sexual excitement, imperfectly effected or excessive *coitus*, or *onanism.* (2.) Others consider it exclusively due to a morbid state of the cerebral structures. (3.) A third class refers the phenomena to a morbid excitability, due to "*nutritive derangement* of the whole nervous system, *both central and peripheral*" (HASSE), which renders it liable to be thrown into disorder by causes insufficient materially to disturb its action in health—thereby implying paralysis of some nervous centres.

Post-mortem examinations of the bodies of those who have died from other diseases, while suffering from hysteria, have yielded negative results.

Symptoms.—The forms and degrees of hysteria are so numerous that the difficulty of describing this disorder is very great. The modifications of age, temperament, states of nervous sensibility, physical and moral education, and grades of society, so influence its aspect that it is only possible to give a mere general outline. It is usually divided into three forms: *first*, that which is characterised by what is termed the "*globus hystericus*," in which the sensation of a ball rising in the throat, or a feeling of suffocation, is experienced by the patient, but without convulsions; *second*, its paroxysmal form, or that in which the *globus hystericus* occurs *with convulsions;* and *third*, those irregular and anomalous phenomena which often manifest themselves during the intervals of severe attacks.

The milder forms are those which terminate without the formation of the paroxysm. They commonly begin with pains in the epigastrium, in the left side, or in some other part of the abdomen; or the patient is unusually nervous, her feelings excited, or depressed. These symptoms having existed for a longer or shorter period, the patient experiences the sensation of a ball, the "*globus hystericus*," rising apparently from the lower portion of the abdomen, and proceeding upwards with various convolutions to the stomach, thence to the throat, and causing sometimes an intense sense of suffocation. At this point the slighter forms frequently cease, but are followed by headache, stiffness of the neck, general weariness, a profuse discharge of a light-coloured limpid urine, and by great flatulence, the abdomen becoming almost instantaneously distended.

When hysteria assumes a paroxysmal form or "fit," it may be preceded by the pains and mental feelings which have been described; but not unfrequently the attack is sudden, and is often caused by some transitory occurrence. In such a case the patient bursts out into a fit of immoderate laughter or crying, the *globus hystericus* begins to form and to rise, and no sooner reaches the throat than she falls to the ground, apparently unconscious and violently convulsed. The fit is now said to be formed, and while in general the convulsions are easily controlled, yet—not only in the strong and plethoric, but sometimes also in delicate-looking slight-made girls—many persons are sometimes necessary to restrain the patient, who writhes her body to and fro, agitates her limbs in various directions, and beats her breast repeatedly with her arm and hand. During the fit the patient also may knock her head against the bed or floor, tear her hair, scream, shriek, laugh, cry, or sob alternately. The respiration is slow, and is rendered still more laborious by spasms about the pharynx and glottis, so that the patient often grasps her neck and throat, or rubs or strikes the epigastrium and side with her hand. During this struggle she may bite her own arms or those of the bystanders, and will sometimes move round the room while lying on her back, by means of the muscles of the dorsal region. The abdomen is often singularly distended with flatus; but in other cases the muscles of that region are tense and irregularly contracted. The pulse is in some cases increased by the violence of the exertion, but in others its beat is natural. The veins of the neck are distended, the carotids beating with more than usual violence. The face is flushed, and "the head is generally thrown back, so that the throat projects; the eyelids are closed, but tremulous; the nostrils distended; the jaws often firmly clenched; there is no *distortion* of the countenance, and the cheeks are at rest," unless when giving expression to some of the above-mentioned phenomena. The temperature of the extremities is often lower at the commencement than natural, so as to cause a momentary shivering, but as the paroxysm forms, the heat is usually restored and sometimes increased. The phenomena attending the subsidence of the paroxysm are various; sometimes a flood of tears, a fit of laughter, or an exclamation, is followed by a great flow of limpid urine, after which the recovery is generally rapid and complete. In other cases the action of the stomach becomes reversed; and the sympathising attendant, perhaps watching the patient with the tenderest care, receives its contents over her person, after which the patient may lapse, most unconcernedly, into a profound sleep. In others, again, the fit only partially passes off, and the patient lies, to a certain extent, sensible of what is passing about her, perhaps jaw-locked, the secretion of urine suspended, unable to talk, and often obliged to be fed. The fit having entirely subsided, the patient lies exhausted and unwilling to be disturbed, and although more or less conscious of what has passed, she wishes to be thought ignorant of all that has taken place. A want of consciousness may exist when the fit assumes a severe or epileptic form; but this is not a common symptom of the purely hysterical convulsion. In some few cases the patient appears to be delirious, and makes the most extraordinary noises, such as barking or howling like a dog. The duration of the fit varies from a few minutes to two, three, or more hours. These fits readily recur, and no sooner is one fit ended than the patient suffers from another; and in this manner the whole attack may last twelve, twenty-four, or even forty-eight hours. In general the intervals are much longer, and not subject to any general law of recurrence, except that they are more common about the period of menstruation.

In the interval between fits, the symptoms are extremely anomalous and irregular, and more strange and difficult to describe than even those of the paroxysm. Some have their senses so acutely alive, that although the window and bed-curtains may be drawn, still they are pained with light, and the slightest noise distresses them. In some, again, the sense of touch is so exquisite, that they can scarcely bear the weight of the bed-clothes; and to others odours are similarly intolerable. Besides this extreme acuteness of the senses, others suffer pains under or in the mammæ, known to surgeons as the "hysterical breast," lumbar pains, pains in the knee-joint or hip-joint, headache fixed to one spot, *clavus hystericus*, and palpitation. Pain in the region of the spine (*spinal irritation*) is also frequent, and often so intense and so exquisitely increased by pressure that it has often been mistaken for ulceration of the intervertebral cartilage. The late Sir B. Brodie recorded that he had seen numerous instances of young ladies condemned to the horizontal posture, and to the torture of issues and setons for successive years, whom air, exercise, and cheerful occupation would have cured in a few weeks. *Hyperæsthesia* is exceedingly common. It may be hemiplegic or paraplegic, may affect a limb or a joint, a mamma, an ovary, the vulva, or the vagina, the spine, or indeed any part, surface, or organ. The pain is peculiar in this, that while the patient may shrink from the slightest touch, or even have the pain aggravated by the attempt, or expressed intention, to touch the part; yet if her attention is distracted and drawn to something else, she will allow the painful part to be touched, compressed, and even violently handled. As to painful affections of the joints, it has been stated by the same high authority, that at least four-fifths of the females among the higher classes who are supposed to labour under diseases of the joints, are suffering from hysteria, and from nothing else. The morbid sensibility is chiefly in the integuments, as in the case of the hip-joint, and if they are slightly pinched or drawn from the subjacent parts, the patient complains more than when the head of the femur is pressed against the acetabulum. There is likewise no wasting of the *glutei* muscles, nor flattening of the *nates*, nor painful starting of the limbs. In some instances the patient becomes paraplegic, and is unable to walk, while others suffer temporarily from *hemiplegia*, steadfastly resisting all plans of treatment likely to be of use.

It is the extreme acuteness and exquisite sensibility of the senses in hysteria which has led those less skilled in female arts to believe in the instances of animal magnetism and mesmerism they seemed to exhibit, which formerly attracted so much public attention. A most interesting account of the vagaries of hysteria may be read in Sir Thomas Watson's thirty-eighth lecture *On the Practice of Physic.*

Diagnosis.—The best diagnostic guide may be obtained by classifying the symptoms according to Dr. Reynold's plan. There is to be noticed—

1. *The Mental State.*—Volition is deficient and misdirected. The emotions and ideas exhibit excessive activity; and to the combination of these two conditions is to be attributed many of the peculiar and characteristic features of the disease. *Assertions* by the patient are being constantly made—such as that she cannot control her thoughts, emotions, expressions, or general voluntary movements: or that she cannot move this or that limb, that she cannot open her eyes, that she cannot stand or walk; and if she makes the attempt under such impressions, she certainly fails; and she may simulate the real inability so completely, and so well, that it seems almost incredible that nothing but defective will is the real source of the failure. If, however, some strong motive, emotion, or sensation come into operation, she may for a moment forget her condition, clap the hitherto moveless hands together, open the closed eyelids, and, with the rapidity and energy of robust health, run across a room or up a staircase with her *quasi*-palsied limbs. It does not appear that with all this there is any intention on the part of the patient to deceive any one, more than herself. It is truly a morbid mental condition on her part, and she doubtless believes in the real nature of her symptoms. Often, also, a species of delirium prevails, in which nonsensical sentences are pronounced in an excited manner. Uncontrolled sobbing, sighing, and laughing are alternately produced, or accompany each other. Somnambulism, ecstacy, or hysteric coma (which is rare), may prevail. Often the expression of the face is insane. A listless, abstracted, vacant look pervades the countenance, as if the individual cared nothing for the things of this world. Combined with this condition there is restlessness and impatience of temper, with monosyllabic talking.

2. *Sensorial.*—The pain of hysteria, which may be anywhere, but most commonly in the head and mammary region, is always described as "intense," "horrible," or "agonising;" and it is increased when the attention is directed towards it, but lessens when the attention is withdrawn. The patient generally shrieks when the skin is touched. *General hyperæsthesia,* or "nervousness," as it is commonly called, is scarcely ever absent, and may exist for years as the only expression of the disease. It is evinced in several ways; such as—unusual acuteness of the senses, neuralgias, tenderness, as pressure of the spine, painful affections of the joints (*Anthropathia hysterica*), noises in the ears, spots before the eyes. On the other hand, *anæsthesia* may prevail, although it is very difficult to determine whether pain is felt or not by operations which usually cause pain, such as pricking, pinching, or burning parts of the skin.

Palpitation of the heart, or pulsations of vessels, is complained of by nearly all; so is pressure and fulness over the stomach, also *cardialgia,* thirst, desire to micturate, and the like.

3. *Motorial* phenomena, when voluntary, are performed sluggishly and imperfectly. The other phenomena in connection with the motor power are exhibited in convulsions or in paralysis. It is the convulsive paroxysms of hysteria which may be mistaken for epilepsy. When they occur, it is almost always at the period of puberty in young females. They frequently attend the menstrual period, and are preceded by the premonitory phenomena of hysteria already described, and which re-appear towards the close of the convulsions. There is probably never complete loss of sensibility and perception. The spasmodic movements are general, extensive, and of reflex origin, causing morbid excitement of the motor nerves, proceeding from the *spinal marrow* and *medulla oblongata.* She throws her arms and legs about in all directions, works her body into the most grotesque attitudes, and may suddenly raise herself to a sitting attitude and dash herself down again, but always taking care not to hurt herself. The face undergoes little alteration. It is free from the contortions of epilepsy. There is commonly a contractile movement of the eyelids. The patient appears to see, and there is no marked change of the pupil. Foaming at the mouth or a bitten tongue is rare. The attacks are sometimes of considerable duration, and the respiratory movements become very disorderly, noisy, and irregular; but the countenance never becomes livid (as in true epilepsy). After the paroxysm has passed, there is no marked stupor, but merely general exhaustion; and loss of consciousness appears to be very seldom complete, and never occurs at the outset of the attack. "The patient," writes Sir Thomas Watson, "is often able to repeat (although she may not always choose to confess it) what has been said by the bystanders during the period when she seems insensible. This is a point of distinction well worth remembering, for more reasons than one. It not only helps the diagnosis, when the fact comes out, but it suggests certain cautions to ourselves. We must take care not to say anything by the bedside of a hysterical patient which we do not wish her to hear; and we may take advantage of her apparent unconsciousness, and pretend to believe in it, and speak of certain modes of treatment which she will not much approve of, but the very mention of which may serve to bring her out of the fit."

The less expressed forms of hysteria cannot be confounded with the less expressed forms of epilepsy. The non-convulsive form of epilepsy is exclusively expressed through disordered sensorial states, such as by vertigo and a suspension (however brief and transitory) of the mental powers. The non-convulsive forms of hysteria, on the other hand, are chiefly expressed in derangement of the organic functions of the thorax and abdomen (FOVILLE, WATSON). In the *status epilepticus* the temperature of the body rises to 103° or 104° Fahr., but in the hysteric paroxysm it rarely exceeds the normal.

It is often difficult to distinguish between the many painful affections of the joints which arise from hysteria and the formidable diseases of these parts which they simulate, mimic, or copy; and many mistakes have been made fatal to health and even to life. The character, however, of the patient, her time of life, her general good health, the intermitting nature of the pain, and its following the course of the nerve, enable us generally to determine with much accuracy between these different classes of disease. The most common mistake, however, is that of considering the pains under the mammæ as pleurisy or disease of the liver, or the abdominal pain as peritonitis or enteritis, thereby leading to an abuse of remedies usually given for these affections. The state of the pulse, however, the general good health of the patient ("for," writes Dr. Wood, "one of the most striking circumstances connected with the disease is the general integrity of the nutritive process—the patient continues plump and rosy"), and most commonly the existence of some uterine irritation furnishes sufficient grounds for diagnosis between these different diseases.

Nausea, eructations, borborygmi, or *tympanites, palpitation of the heart,* with *syncopal feelings, frequent micturition* of clear pale urine, are characteristics of the hysterical state. A certain constitution is also characteristic of the sufferer from hysteria. The external conformation of the features of the face is often of itself sufficient to indicate the existing tendency. The "*facies hysterica*" may be recognised by the remarkable depth and prominent fulness of the upper lip, which is more or less thick. There is also a fulness of the eye, with a tendency to drooping of the upper eyelids.

Prognosis.—The ultimate result of these cases, though often long and tedious, is always favourable. "In nine hundred and ninety-nine cases out of a thousand, hysteria is attended with no ultimate peril either to mind or body" (WATSON). The attacks of hysteria never lead to impairment of the intelligence or to dementia, as do those of epilepsy. The disease may continue for years with varying intensity; but there is no fixed period of its duration; and the malady may run into *epilepsy* or to some disorder of intellect.

Treatment.—The treatment may be divided into what should be done during the fit, and into what should be done afterwards.

When the patient falls into a fit of hysteria, the first thing to be done is to loosen everything tight about her person. The window should be opened, and the cold air allowed to blow over her. She should then be laid in the horizontal posture on a bed, or on the floor, as a means of rendering the circulation through the brain more equal, and to enable us the more readily to control the convulsive movements of her body. This being done, many modes of further proceeding may be followed. Some recommend, in plethoric cases, that the patient should be bled—a remedy certainly in many instances manifestly improper, and in all of doubtful efficacy. When the jaw is locked, an enema, consisting of the yolk of an egg beat up with two drachms of *asafœtida,* with half a pint of water added, may be administered; or, still better, an enema of *turpentine,* in which half an ounce of turpentine is similarly mixed with the yolk of an egg, and half a pint of water added. These remedies, in some instances, instantly remove the affection, but in other cases not for some hours. Another remedy is to fill the mouth with salt. The remedy, however, which supersedes all others, and is unquestionably the best, is a good drenching with cold water. "I believe there is more virtue in cold water than in any other single remedy" (WATSON). If the patient lie on the bed, the head should be drawn over its side, and a large quantity of water poured on it from a considerable height out of a pail, jug, or other large vessel, and directly over the mouth and nose of the patient, so as to stop her breathing, and compel her to open her mouth. This practice is generally introduced into hospitals; and until it was adopted it was not unusual to see three or four patients in hysteria in the same ward, and at the same time. Under this practice, however, an hysterical case is rare, the fit seldom occurs twice in the same person, and the affection never becomes epidemic.

After the paroxysm is over, if the patient complains of continued headache, a few leeches to the temples may be necessary, especially if the urine be small in quantity and high coloured; but in all other cases leeches, blistering, or cupping should be avoided, as tending rather to aggravate than to control the disease. The next object is to regulate the bowels by such remedies as may be necessary, and at the same time to support and tranquillise the patient by stimulants, such as *ether* or *asafœtida,* combined with *hyoscyamus* in the form of tincture, the *syrup of poppies,* or small doses of *morphia.*

Niemeyer recommends the *chloride of sodium* and *gold* as a restorative nerve tonic. He prescribes it in the form of a pill, as follows:—

℞. *Auri. chlorat. natronat,* gr. v.; *Gum. tragac. Co.,* ʒi.; *Sacc. Alb.,* q. s.; *misce.* Divide into forty pills, of which one is to be taken an hour after dinner, and another an hour after supper. After a time, two are to be taken as a dose at one time, and increased gradually till eight pills a day are taken; and a cold shower bath twice daily.

The state of the uterine functions must always be inquired into. If leucorrhœa be present, or the menstruation be profuse, the *mineral acids,* or the *bitartrate of potass.,* will be found most efficacious, by restoring a more healthy state of the deranged organs.

The urine is often suppressed for a time after an attack of hysteria; but unless the bladder be sensibly, and perhaps painfully distended, no attempt should be made to draw the urine off. Something more should be hazarded to avoid this necessity, for the catheter once passed, that operation will require to be performed morning and night, perhaps for several months. It is important that pity, attention, and sympathy be withheld as much as possible from hysterical patients. Their moral and bodily condition deteriorates under such influences. Firmness is essential for successful treatment.

Certain special forms of hysteria require separate notice:—

I. Hysterical Ischuria is an affection in which the quantity of urine secreted in the twenty-four hours, and withdrawn by the catheter, is remarkably under the normal quantity. During several days there may be absolute suppression (Charcot). It is to be regarded as one of the numerous phases or forms of hysteria, and is an extremely rare affection. Simple retention of urine in the bladder is, on the other hand, a most common condition in hysteria. The ischuria of hysterical patients is also almost always complicated with urethral retention. *Oliguria,* or even *total suppression of urine,* may be only a transient phenomenon in hysterical cases, and one which may occur unnoticed; as at the catamenial periods, when a complete suppression may last from twenty-four to thirty-six hours;

and when a few spoonfuls of urine are expelled the normal state is restored (LAYCOCK, *Nervous Diseases of Women*, 1840, p. 229). But the cases of hysterical ischuria which Charcot describes, when at their maximum development, and when the ischuria has assumed the character of a permanent symptom, are of such a kind that during the lapse of several successive days, even of weeks and months, the quantity of urine rendered in the twenty-four hours may be quite insignificant in amount, almost *nil*, and occasionally there may be *complete suppression* of urine during a series of several days. As a complement to this condition, there occur repeated *vomitings*, which take place daily, and even several times a day, so long as the ischuria continues, the ejected fluid occasionally presenting the appearance of and exhaling the odour of urine. Chemical analysis has detected in it the presence of urea. The phenomena of the affection are more or less a reproduction of some of the symptoms observed in animals in cases of nephrotomy or of obliteration of the ureters by ligatures.

The reality of the disorder has been disputed; for *simulation* is to be met with and to be guarded against at every step in the history of hysteria, and Professor Charcot having passed through a stage of scepticism, has now become an exponent of the disease.

Symptoms.—The following are the general sequence of phenomena. *Anuria* or *ischuria*, with vomiting, exists alone for a certain time; and soon (if the symptoms happen to excite interest and curiosity, as they naturally would) pure urine will come to be thrown up in considerable quantity, and stercoraceous vomiting may even occur, not to mention other too fabulous occurrences, as recorded in some American cases. Apart from hysteria, it is to be remembered that *suppression* of urine, if it persist beyond a few days, say three, four, or five, is an exceedingly serious symptom, which almost necessarily terminates in death, as occurs sometimes in Bright's disease. There are also *typical cases* of anuria caused by calculous obliteration in the ureters, supervening in persons previously healthy. In such cases sometimes one kidney has been reduced to a fibrous husk filled with cysts—incapable of fulfilling its function. Sometimes both ureters are thus obliterated at once. If anuria thus persist beyond four or five days comatose symptoms supervene, with or without convulsions, soon followed by death; but life may be prolonged for a little longer, if even only a small quantity of urine can be rendered. Exceptional cases are quoted by Charcot of—(1.) Anuria lasting ten days, when recovery took place; (2.) Somnolence not appearing till eight days, four days before death; (3.) The comatose symptoms not presenting themselves till the fourteenth day of absolute anuria. On the fifteenth day the patient passed a certain quantity of urine, but the symptoms became aggravated, and death took place on the twenty-third day.

There is thus a great contrast between *calculous ischuria*, which almost certainly kills, and *hysterical ischuria*, which allows the patient to survive during many months without seriously disturbing the general health.

In the case recorded by Charcot there were all the signs of an inveterate hysteria, marked by the characteristic permanent symptoms of—(1.) Vast *contraction*, affecting the upper and lower limbs of the left side; (2.) complete *hemianæsthesia,* occupying both the contractured limbs and the left half of the face and body, with *hemiopia* and *achromatopsia*; (3.) *ovarian hyperæsthesia*; (4.) *retention of urine* and *tympanites.* Special precautions were taken by Professor Charcot to prevent deception on the part of the patient. He is of opinion that he has in women over women the best possible police; and that if women enter into any plot among themselves, they very rarely succeed. Professors Brown-Séquard and Rouget were satisfied with all the precautions taken. The first thing noticed regarding the ischuria was by the woman who had been in the habit of catheterising the patient several times a day. She noticed that the quantity of urine discharged was occasionally very minute, whilst at other times none was yielded for two, three, or even more days, nor were the sheets ever moistened. After three months vomiting appeared and proceeded without effort or straining. The daily average of the vomited matter for two weeks was 1¾ pints nearly. The daily average quantity of urine was about 38½ grains; and during this period there was absolute ischuria every second day. The matters vomited contained urea. The ischuria lasted nearly six months, and eventually disappeared after the administration of chloroform, incontinence of urine following, which lasted for several days, vomiting also ceased, and the urine returned to its normal condition.

II. HYSTERICAL ANÆSTHESIA AND HEMIANÆSTHESIA.—*Anæsthesia,* in hysterical cases, presents itself under two aspects:—(1.) Hemianæsthesia, where one-half of the body is affected; and this is the more frequent form. (2.) Total anæsthesia, where the whole of the body becomes insensible. But it should be remarked that not only is general sensibility diminished or destroyed over the surface of the region affected, but the organs of special sense are likewise subjected to change. Thus, in the case of a hemianæsthetic patient, one-half of the tongue loses the sense of taste, one nostril that of scent, and one eye becomes stricken with amblyopia. In fact, hysteria is capable of producing groups of symptoms similar to those produced by lesions other than dynamic of the central nervous system. It is to be noted that the region in which the anæsthesia occurs is the same in which the other accidents make their appearance. If the anæsthesia be unilateral, then ovarialgia shows itself on the same side; the aura likewise is noticed there, including its cephalic symptoms—namely, whistling in the ear and tapping or beating in the temple. If contracture supervene, its manifestation follows the same rule. On the other hand, if the anæsthesia, instead of being unilateral, be double, then the phenomena referred to are doubled, and show themselves on both sides, though they are often somewhat more strongly marked on one side than on the other. It has been said that, in cases of hemianæsthesia, the left side is most usually affected. That, however, is a point on which no definite information is yet available—the patients who have come under Professor Charcot's notice being as frequently affected on the right side as on the left.

There are also cases of hysteric anæsthesia of peculiar distribution. One such has been carefully recorded by Dr. J. Dreschfeld, Physician to the Manchester Royal Infirmary, as follows:—

The patient (aged 28) presented herself at the out-patient room of the Manchester Infirmary, complaining of numbness in her right leg and left side of trunk, from which she had been suffering for more than a month.

Previous History.—She had always enjoyed good health, and had never suffered from convulsions or other nervous symptoms; had been married for twelve months, and was confined of her first child on May 14, 1878. During the whole time of pregnancy, she suffered but little; the labour, however, was a very tedious one; the patient suffered from labour-pains for more than four days, and had eventually to be delivered by instruments. Two days after confinement, she got out of bed, when she fainted, and for four days afterwards she remained in an unconscious state. She had no fits during the time, but rambled very much. As soon as she regained consciousness, she was able to get about; but suffered from giddiness and headache. She noticed also, at the same time, *numbness of her left breast.* She could not tell whether the "*baby was suckling the left breast unless she looked.*" She noticed the numbness in her right leg also at the same time. The symptoms not abating, she eventually came to seek relief at the Infirmary.

Present Condition.—The patient was of middle stature, healthy-looking, and of a ruddy complexion. The examination of the organs of the chest and abdomen revealed nothing but normal relations. There was no disturbance of either motion or nutrition, and the abnormal phenomena which were observed involved the sensory sphere exclusively. A careful examination elicited the following interesting symptoms:—(1.) *Special Sense Organs:* Vision normal on both sides; the fundus of both eyes normal; no achromatopsia. *Hearing:* Complete deafness on the left side; hearing on the right side normal. *Smell:* Complete absence of smell on the left side; smell on the right side normal. *Taste:* Normal on both sides. No anæsthesia of either side of face and neck. *Trunk:* Anæsthesia of the left side. The anæsthetic portion was bounded by the middle line, both in front and behind. Its upper margin was formed by a line passing horizontally round the left side, just above the third rib, and passing under the axilla to the middle line behind, while its lower border was bounded by a line commencing just above the pubes, and passing at first along Poupart's ligament, and then horizontally round the left side (about one inch below the crest of the ilium) to the lowest part of the sacrum. (2.) *Upper Extremities*: Both were perfectly normal as regards sensibility and mobility. (3.) *Lower Extremities*: The left leg and thigh were perfectly normal, both as regards motion and sensation; the right leg and thigh could be moved with perfect freedom, but were completely anæsthetic. The upper limit of the anæsthetic right lower extremity coincided exactly with the lower limit of the anæsthetic left half of the trunk—*i.e.*, just where the patient began to feel tactile impressions on the right side there the anæsthesia on the left side commenced. There was no ovarian hyperæsthesia.

Examining the character of the anæsthesia more minutely, the following points were made out:—1. Total loss of perception of tactile impressions in all the anæsthetic parts. 2. Analgesia of these parts. The right calf was transfixed by a large dissecting needle without the patient evincing the least sensation; the same needle was passed deeply into the left mamma and other parts of the left half of the trunk with the same result. The points where the needle was inserted bled freely. 3. Loss of muscular sense. The patient, with her eyes shut, had no knowledge of the relative position of the anæsthetic leg. She was told to sit down and shut her eyes, and, on now moving the right leg, she was still under the impression that the right foot touched the floor. 4. Loss of sense for temperatures; neither cold nor heat was perceived by any of the anæsthetic parts. The sense for weights was not examined into.

On July 8, the patient presented herself again, and her condition had undergone no change, and we now determined the behaviour of the anæsthetic parts to the electric current. On applying an induction current of moderate strength to the anæsthetic leg (one pole being placed in contact with the lumbar part of the spine, and the other with the calf of the right leg), the patient felt the current distinctly, and, on now increasing the strength of the current, the patient complained of a painful sensation, and distinct contraction was visible. We then tested the sensibility of a point in the left calf symmetrically situated with that on the right calf, to which the electrophone had been applied; but the sensibility had remained the same, and, therefore, no transfer had taken place. To the galvanic current the anæsthetic parts responded, though less distinctly than on the sound side.

Progress.—July 10. Ever since the application of the Faradic current the patient felt a slight return of sensibility, not only in the right leg, but also in the left half of the trunk. There was, however, still a considerable amount of dysæsthesia. The Faradic current was now regularly applied by Mr. J. Wood, but only to the right leg, and with very satisfactory results; for, on August 15, it was noted that the anæsthesia and analgesia had entirely disappeared from the right leg and left half of the trunk. Hearing and smell were still somewhat impaired on the left side, though a considerable improvement had taken place here too.

Since then, the patient presented herself once more, and reported that she was quite cured. A close examination showed still a slight affection of the organs of hearing and smell on the left side. The patient experienced, however, so little discomfort from this, that she was unwilling to submit to further treatment.

Lactation was in no way interfered with by these conditions, and the left mamma yielded quite as much milk as the right.

The case presents several peculiarities:—1. The distribution of the anæsthesia is, as far as I can make out, unique. Briquet describes several cases of hysteria with alternating anæsthesia; but, in all these cases, the whole of *one* side of the trunk and the opposite side of the face were so affected, and the same distribution has been observed in cases of anæsthesia dependent on organic lesions (*Gazette Hebdom.*, 1878, Nos. 1 and 3). Again, I believe, in all cases of hystero-epilepsy described, the anæsthesia observed was distinctly unilateral, affecting the sense organs, the trunk, and the extremities of the same side. The integrity of the upper extremity of the face and neck, of vision, and of the sense of taste in this case is equally remarkable.

2. In its character the case very much resembles cases of hystero-epilepsy; but here the absence of other hysterical symptoms, especially the absence of any hystero-epileptic attack, will again be remarked, unless we consider the attack of unconsciousness which took place soon after the confinement as a kind of trance or comatose condition, not unlike that observed in a case recently published by Vigoureux (*Prog. Medic.*, 1878, p. 679). The sudden seizure, and the fact that this patient was able to get about as soon as she regained consciousness, make this view highly probable (*Brit. Med. Journ.*, Oct. 12, 1878, p. 553).

Hemianœsthesia occurs in hysterical patients when one entire half of the body, face, neck, and corresponding extremities, lose the sense of feeling, not merely as regards the superficial parts, but also as affecting deeper regions—namely, the muscles, bones, and articulations.

The affection shows itself under two principal aspects—complete and incomplete. In the complete form it has been so frequent in the experience of M. Briquet as to occur in 90 cases out of 400; of which 70 suffered in the right and 20 in the left side. One of the commonest varieties of the condition is that of *analgesia*, with or without insensibility to heat or cold, or thermo-anæsthesia. Hysterical hemianæsthesia is still further characterised by the distinct manner in which the anæsthetic parts are separated from the healthy parts. On the head, face, neck, and body the demarcation is often perfect, and very closely corresponds with the median line. Comparative pallor and coldness of the anæsthetic side are other indications, conjoined with more or less permament ischæmia, in which there is difficulty in drawing blood when the anæsthetic parts are pricked with a pin; and in which leech-bites yield little blood on the anæsthetic side, whilst on the healthy side blood flows from them as usual. Further, the *mucous membranes* are affected on one side of the body in the same manner as the external integument. The organs of the senses also are affected to some extent in the anæsthetic side. *Taste* may have gone from the corresponding half of the tongue, from tip to base. The sense of *smell* is less acute. Vision is weakened in a remarkable manner, so that *achromatopsia* occurs. The notion of colour is perverted. Some hysterical patients see all colours as grey, or only preserve the notion of one or two of them. This has been named hysterical achromatopsia, and may be either partial or complete (GALEZOWSKI). As regards the normal eye, the visual field is not the same for all persons and for all colours. It varies in different individuals. Mr. Landolt, who has made a scientific investigation of this subject, in examining the range and colour-perception of the eye, found the following series: Blue was observed to have the widest extension; then came orange, yellow, red, green, and violet. There are, however, varieties; for some patients the red has the widest extension: hence the "blues" and the "reds" may be arranged into two classes. In the latter class, the colours are arranged thus: first comes red, then follow yellow, blue, orange, green, and finally violet. Violet is always in the centre; its visual field is the least extended. Next to it is green. When the disease first shows itself, or is in its first stage, it is often found that the patient has no perception of violet. This circle narrows and disappears, and the others next to it may also narrow, without quite vanishing; in such a state, the person is achromatopsic for violet and dyschromatopsic for the others. In the second stage, or degree, the green also disappears, and the patient has become achromatopsic as regards these two colours. This process may go on until the achromatopsia is almost complete. When the colours have disappeared to this extent, and return of perception takes place, they are found to reappear in due order from the periphery to the centre, violet coming last of all. There are many hysterical patients who have lost all power of distinguishing violet; they cannot recognise its existence. Several, in fact, lose both the violet and the green.

It is to be observed that the arrangement of colours in these series is not at all the same as the arrangement of colours in the solar spectrum. This supplies evidence that the patients do not deceive us; for, if it were possible to conceive that they had studied the arrangement of colours in the solar spectrum, and could shape their report accordingly, it is impossible to suppose that they could devise an uniform series, differing from any they had ever seen, to which they would constantly adhere.

The condition does not affect the viscera; but usually in such cases the ovary may be found to be in a state of hyperæsthesia. It may be very painful on pressure, while the abdominal wall is perfectly insensible. The hemianæsthesia and the hyperæsthesia of the ovary are usually on the same side of the body; and when the ovarian hyperæsthesia is double, the anæsthesia also presents itself in a generalised form, and occupies nearly the whole or quite the whole of the body. So also with regard to other permanent phenomena—the paresis and contracture of the limbs are also on the same side as the anæsthesia.

Hemianæsthesia thus becomes a symptom of great clinical importance in hysteria; and when once seen it is generally found to be permanent. It is a symptom which requires to be sought for; and is so far characteristic of hysteria, that it is very rare for the condition to be reproduced, with the general grouping of all its characteristics, by any other disease. But there is also to be distinguished a hemianæsthesia of cerebral origin, with derangements of the special senses such as are presented in hysteria, which may in certain cases be produced by a circumscribed lesion of the cerebral hemispheres. In these hemispheres there exists a complex region, lesion of which determines hemianæsthesia. The limits of this region are approximately known; but at present localisation cannot be carried further. Anæsthesia of *general sensibility* alone appears to have been observed as consecutive on an alteration of the cerebral hemispheres, so that *obnubilation of the special* senses would remain as a distinctive characteristic of hysterical hemianæsthesia.

III. OVARIAN HYPERÆSTHESIA is a symptom most frequently associated with hysterical hemianæsthesia. It is a variety of "*local hysteria;*" and is one of the permanent phenomena which remain as more or less constant characteristics of hysteria, the others being *paralysis, contracture,* and *fixed* painful points occupying different parts of the body (*rachialgia, pleuralgia, clavus hystericus*).

The pain, which is felt on one side of the abdomen, especially in the left (but it may occupy both sides—*at the extreme limits of the hypogastric region*), is the *ovarian* pain which is peculiar to certain forms of hysteria. This ovarian pain occurs in two classes of cases:—(1.) Those in which it is a very acute pain. The patients cannot tolerate the slightest touch, nor suffer the weight of the bed-clothes. They also shrink suddenly and instinctively from the finger of the investigator. There may also be a certain degree of tumefaction of the abdomen—*false* or *spurious peritonitis.* The muscles and skin share in the suffering. The pain occupies a considerable extent of the body surface, and is not easy to localise; but there is, in certain cases (as observed by TODD and CHARCOT), a circumscribed cutaneous hyperæsthesia, which occupies a rounded dermal space of from two to three inches in diameter. This hyperæsthesia has its seat partly in the hypogastrium, partly in the iliac fossa, and corresponds to the region of the ovary. (2.) In the other class of cases, the pain does not spontaneously show itself. It must be sought for, and it requires pressure to discover it. The following phenomena are to be noted:—(*a.*) There is general anæsthesia of the *skin;* (*b.*) the *muscles,* if relaxed, may be pinched and raised without causing pain; (*c.*) the preliminary explorations prove that the pain is neither in the skin nor in the muscles; (*d.*) by firm pressure into the abdominal cavity, the real focus of pain is reached, at a constant fixed part, always nearly the same as almost unanimously indicated by patients. From a line uniting the anterior superior spines of the ilia, intersected by the perpendicular lines which form the lateral limits of the epigastrium, at the points of intersection, will be found the focus of pain on pressure being applied by the finger. In deep exploration of this region, the hand will meet with an ovoid body, which when pressed slips under the fingers; and when it is swollen it may be as large as an olive or a small egg. The pain is chiefly determined at the period of exploration, and it has *specific* characters. It is a complex sensation, accompanied by all or some of the phenomena of the *aura hysterica,* as they spontaneously show themselves before an attack. When the sensation is thus determined by exploration, the patients at once recognise it as familiar—as having felt it "scores of times." Irradiations of pain are thus provoked towards the epigastrium, sometimes complicated with nausea and vomiting. If pressure be continued, we get palpitation of the heart, with extreme frequency of the pulse; and, finally, "the *globus hytericus*" is developed in the throat. Charcot further distinguishes certain cephalic disorders which are the continuation of the same series of phenomena, such as sibilant sounds in the left ear, if the left ovary is hyperæsthesic; a sensation as of blows from a hammer falling on the left temporal region; and, lastly, *obnubilation* of sight in the left eye. The analysis cannot be carried further, because consciousness at this point becomes so profoundly affected, that the patients no longer retain the power of describing what they feel; and the convulsive fit soon supervenes, if the experiment be persisted in.

Such is the experiment of Schutzenberger as described by Professor Charcot, by which compression of the ovarian region simply reproduces artificially the series of symptoms that spontaneously present themselves in the natural course of hysteria, in which the *iliac pain* always precedes in point of time, however small the interval may be, the *epigastric pain,* in the development of the aura. Hence it is argued that, to the ovary, and to the *ovary alone,* we must attribute the *fixed iliac pain of hysterical patients,* in a certain group of cases. Experience has also led to one practical result in the hands of Professor Charcot—namely, that energetic compression of the painful ovary has no direct influence over most of the *permanent* phenomena of hysteria, such as contracture, paralysis, or hemianæsthesia; but it has a frequently decisive effect upon the convulsive attack, the intensity of which it can diminish, and even the cessation of which it may sometimes determine.

For the purpose of diagnosis it is necessary to mention other forms of hemianæsthesia than those referable to hysteria. Besides hysterical anæsthesia, there is a loss of sensation due to—(1.) *Chronic* *alcoholism;* (2.) *Chronic brain disease;* (3.) *Lead-poisoning.* The hemianæsthesia from chronic alcoholism and from lead-poisoning are the two most nearly resembling, and allied to the analagous condition in hysteria. But hysteria is not the only condition in which special and general hemianæsthesia is met with. It may be due to a distinct and localised organic lesion which can be accurately defined by examination after death. Attention was first called to such cases by Ludwig Türck in 1859, when he described four cases of hemiplegia with hemianæsthesia, in which necropsies were made. In two of the cases the lesion was outside the optic thalamus of one side, affecting a considerable extent of the *corona radiata.* Two cases are also recorded from the practice of Professor Charcot, and reported by M. Pitres (*Progrès Medical,* 1876, p. 522). One had hemiplegia for four years, and in whom both general and special sensation were lost. The lesion was in the posterior half of the optic thalamus, extending outwards so as to destroy the caudate nucleus, involving the back part of the internal capsule. The second patient had been hemiplegic for twenty years, and appreciation of pain was almost completely abolished. Sight was fairly good in both eyes, but the field of vision on the affected side was concentrically contracted; while the senses of hearing, smell, and taste, were very much dulled on the same side. Professor Charcot has found that there is a frequent connection between *posthemiplegic chorea* and this particular form of *hemianæsthesia,* and his observations have been confirmed by other observers, so far as the sense of sight is concerned. In two cases reported by Bernhardt, loss of sight in the eye of the hemiplegic side was associated with choreic movements in the limbs of that side. In one of Professor Charcot's cases, at the Salpêtrière, an elderly woman who had suffered from hemiplegia for several years following an apoplectic stroke, had *general and special anæsthesia,* associated with *choreic movements.*

We find, then, that an affection of sensation, general and special, of every order, may be met with in a considerable variety of conditions besides hysteria, and that which is due to hysteria differs in no way from that which results from other causes, gross organic lesions not excepted As a rule the whole realm of sensation is involved, but in hysteria, as well as in the other conditions, we may find one special sense or certain districts of skin unaffected; thus, in the case of lead-poisoning, the sensation of the foot was nearly normal, whilst everywhere else the patient was anæsthetic. The affection of sight, again, is in all quite different from that seen in most cases of organic hemiplegia where the optic centres are involved, for in these we find hemiopia—*i.e.,* blindness of that half of the field of vision in each eye which corresponds with the paralysed side. In the other cases, however, we find affection of the eye on the paralysed side only, the other eye being intact; and the affection consists of concentric diminution of the field of vision in all directions, which may, in some cases, amount to complete blindness. Another feature which the hysterical cases have in common with those due to other causes, is the transitory nature of the symptoms (Dr. W. Allen Sturge, *Brit. Med. Journal,* June 1, 1878, p. 783).

RHYTHMICAL HYSTERIC CHOREA.

Definition.—*A choreiform affection under the influence of hysteria, and preceded by it* (CHARCOT).

Pathology.—*Causation.*—This convulsive form of hysteria is most common in the female, and may manifest itself even before menstruation, at a very early age, the permanent local symptom of greatest importance being, even then, ovarian. The convulsive paroxysms of the local phenomena persist after the appearance of the menses, the occurrence of which does not in any way modify the clinical aspects of these cases. Professor Charcot gives numerous instances in which the ovarian hysteria has become developed several years before menstruation. He also has given cases in the opposite direction—namely, those of *hystero-epileptics* in whom the local ovarian symptoms of hysteria persist for years after the occurrence of the monopause. The facts connected with these two classes of cases tend to demonstrate that the activity of the ovary is far anterior to the menstrual function, and survives it.

Symptoms.—They are those of local hysteria. There exists an ovaralgia or *ovarie* on one side, very distinctly marked; the pain by which it is manifested is fixed, always present in a certain degree, and is spontaneously increased at the approach of the attacks. At all times it is increased by pressure. It is at this painful point that the sensation of premonitory aura of the attacks seems to take origin, ascending then to the epigastrium, præcordial region, neck, head, &c. All the one side of the body, head, neck, and trunk and limbs, is in a state of complete, absolute anæsthesia. A large and not well-pointed needle may be passed through thick folds of the skin of the forearm, of the nape of the neck, of the leg, and of the interdigital folds of the hand, without the patient having the least idea of what is being done. On the same side there is also thermo-anæsthesia; and, further, the special senses are also all affected, although in different degrees. There are anosmia in the right nostril; absolute loss of taste in the one half of the tongue, from the point to the base; diminution of the auditory power of the ear; and amblyopia with dyschromatopsy of the eye on the same side. In this eye the patient completely loses the perception of colours called central—violet and green; while she still distinguishes quite clearly the peripheral colours—red, orange, yellow, and blue. To this hemianæsthesia corresponds, as is usual in such cases, a certain degree of hemiamyasthenia. Thus, in a case described, whilst for the left hand the dynamometric pressure is 25 kilogrammes (nearly 54 lbs.), it is represented for the right hand by only 15 kilogrammes (a little more than 32 lbs.).

There is also a remarkable concordance between the seat of the iliac pain and the mode of localisation of the concomitant symptoms. The ovaralgia is seated on one side; and it is the same side that suffers from anæsthesia and cachexia. It is also on the same side that is manifested the contraction of the limbs when it follows the attacks; and it is the limbs of the same side that are agitated by the rhythmical choreiform movements; and it is at least very probable that these phenomena would almost occupy the same side of the body that the ovaralgia is seated on.

But there are some rare exceptions to this rule. Professor Charcot has once seen hemianæsthesia occupy the left side whilst the ovaralgia occupied the right. Dr. Barlow of London has published a fact of the same nature. The rhythmical choreiform movements are attached to the hysteric diathesis as a subordinate affection, in the same way as paralysis, contractions, and convulsions. The rhythmical choreiform agitation is to be considered as the continuation or the prolongation, under a new form, of an abortive hysterical paroxysm. Methodic compression of the pained ovary has also the constant effect of arresting the rhythmical movements. The rhythmic movements, first of the body, and then of the limbs, stop suddenly, although the limbs may not recover their normal functions. They may become the seat of considerable muscular rigidity. The rhythmic chorea is, therefore, to be regarded as an hysterical attack, in some sort prolonged and transformed. But the arrest under ovarian compression is only temporary, so long as the compression is continued. Immediately it is suspended the rhythmical movements return. It is therefore but a varied manifestation of the hysteric diathesis or temperament.

There are also other and different forms of hysterical chorea; for example, those in which the voluntary movements remain regular in the midst of choreic convulsions. Curious movements are executed, so that the patient may be thrown forward, as if by a spring, by involuntary muscular contraction. Thus, he may suddenly jump to seven or eight feet in front of the place where he might be standing, or he may get up abruptly (as if mechanically) from the chair on which he might be sitting. There is a kind of harmony amid the disorder of the locomotor functions. The name of *chorea saltatoria* has been given to this form of chorea (a variety of the *methodical* or *rhythmical* forms of chorea), which includes *chorea festinans* or *procursiva, chorea rotatoria,* and *chorea vibratoria.* In *chorea festinans* the patient is irresistibly compelled to run forward, without being always able to avoid obstacles; or he is compelled to go backwards continuously, without being able to help himself. *Chorea rotatoria* is characterised by rotation or oscillation of the head, or trunk, or of one limb, to the extent of twenty to thirty, forty, or even eighty times a minute. It sometimes terminates in death, and spares neither age nor sex, although it occurs less frequently in children. *Chorea oscillatoria* consists in irregular or measured oscillations, partial or general, of the head, trunk, or limbs (TROUSSEAU'S *Clinical Lect.*, Vol. I., p. 425).

Prognosis.—These conditions may persist for several years, as hysterical affections generally do, and all the forms of hysterical chorea just noticed are distinguished by their extreme chronicity and obstinacy to the influence of all curative remedies.

Treatment.—Professor Charcot recommends that prolonged and repeated ovarian compression might be maintained with benefit to such cases as occur in the female by means of some such instruments as are used for compression of large vessels like the aorta. So also the application of ice bags to the ovarian region for half an hour at a time, repeated several times a day for several days, has had the effect of arresting the attacks, or of lessening their intensity. He prefers, however, the inhalation of *ether* or of *nitrite of amyl.* Professor Trousseau has found cases of those forms of chorea to improve under the influence of *oil of turpentine* in capsules containing ten minims each, of which ten were taken daily for twelve to fifteen days a month; and to use warm baths of several hours' duration.

EPILEPSY AND HYSTERO-EPILEPSY.

LATIN EQ, *Epilepsia;* FRENCH EQ., *Epilepsie;* GERMAN EQ., *Epilepsie*—Syn., *Fallende Krankheit;* ITALIAN EQ., *Epilessia.*

Definition.—*A complex nervous state, characterised by chronic convulsive attacks not due to organic brain disease, tonic at first, but which subsequently become clonic, and ultimately impede the respiratory process. The attack, lasting from an imperceptible period to two, twenty, or more minutes, is followed by some exhaustion and sleep. The expression of the epileptic state varies from simple vertigo to the most severe paroxysm, a momentary suspension of consciousness, a fixity of gaze, a totter of step, and a confusion which appears and disappears almost instantaneously, which the patient only can recognise. The cases comprehend*—(1.) *Simple epilepsy, or epilepsy proper, in which there is a paroxysmal loss of consciousness as the first, or nearly the first phenomenon in the paroxysm, and with this loss of consciousness there is more or less spasm of muscles (including those of the muscular coats of the arteries).* (2.) *Epileptiform seizures, in which there is no evidence of organic brain disease, but developments of the movements normally represented in some particular parts of the cerebral cortex.* (3.) *Hystero-epilepsy* (*Hysteria major* of Charcot, the *Hysteroid* of Dr. W. Roberts, or *the Co-ordinated convulsions* of Gowers) *which presents, with loss of consciousness, varied convulsive movements of more or less co-ordinated spasm, spasmodic movements of a quasi-purposive character, opisthotonos, emprosthotonos, and the like, forming a series from simple hysteria to true epilepsy. The co-ordinated spasm may exist alone, or may be combined with epileptoid phenomena, or with true epileptic symptoms.*

Pathology.—Epilepsy has been known from the earliest antiquity, and is remarkable as being that malady which, even beyond insanity, was made the foundation of the doctrine of possession by evil spirits, alike in the Jewish, the Grecian, and the Roman philosophy. The interest and importance which attach to *epilepsy* cannot be better expressed than has been done by Sir Thomas Watson: "It is scarcely less terrible to witness when it occurs in its severer forms than *tetanus* or *hydrophobia;* but it is not attended with the same urgent and immediate peril to life. Yet it is, upon the whole, productive of even more distress and misery, and is liable to terminate in even worse than death; a disease not painful probably in itself; seldom immediately fatal; often recovered from altogether; yet apt in many cases to end in fatuity or insanity; and carrying perpetual anxiety and dismay into those families which it has once visited." Fully impressed with the responsibility entailed on the physician in the diagnosis between hysteria and epilepsy, he further observes that "it is a dreadful announcement to have to make to a father or mother, that their child is epileptic" (*Lectures* xxxv., xxxviii.). The combination of these two diseases, as expressed in the definition (3.), is now recognised under the name of hystero-epilepsy, as one which presents symptoms of the most characteristic kind. The proportion between the cases presenting these two classes of symptoms are thus stated by Dr. Gowers:—Out of 1000 cases they were purely epileptic in 815, whilst hystero-epileptic symptoms were present in 185, or 18½ per cent. These three kinds of cases must now be considered together. They cannot be entirely separated from each other; and they have no doubt been mixed up in all the statistics of epilepsy that have hitherto been published.

(*a.*) *Causation.*—(A.) *Predisposing.*—(1.) As regards sex and sexual conditions—in infancy, and under seven years of age, epilepsy occurs in nearly equal proportions in both sexes. After puberty, when the distinction of sex is marked, some authors contend that it is more common in males than in females—in the proportion of 27 to 11 (Dr. ELLIOTSON). Esquirol, however, states that, on comparing the number of epileptics at Bicêtre and at Saltpêtrière, the number of women attacked was one-third greater than that of the men. In an analysis of the returns of the Registrar-General given by Dr. Sieveking, with reference to the mortality from the disease in either sex during a period of seven years, it appears that 6,729 were males, and 6,149 females, giving a relative proportion of 52·26 males to 47·73 females. Sir Thomas Watson also states that he has seen "more epileptic boys and men than girls and women." Thus, in most statistics of epilepsy hitherto collected in this country males have predominated. Dr. W. R. Gowers, in his exhaustive analysis of 1450 cases, thinks this is probably due to the inclusion of cases of syphilitic brain disease, which we have only lately learned to separate. He found females to preponderate over males in the proportion of six to five, or, in more exact percentage, of 53·4 females to 46·6 males—*i.e.*, to every 100 males epileptic there were 114 females. In cases presenting hystero-epileptic symptoms (to be afterwards described) females preponderate to a much larger extent, and are to the males as two to one: 66 per cent. being females, and 33 per cent. males.

(2.) *Inheritance.*—Hereditary predisposition plays an unmistakable part in the history of epilepsy; and there are few diseases in which *heredity* is a more important etiological condition. But, on this point, Dr. Gowers justly observes, that—"The difficulties of the investigation of heredity among the poor are well known. Of all diseases, it is probably true that the results obtained fall short of the actual facts; but the discrepancy is probably less in these affections than in almost any others. The occurrence of fits or insanity in any member of a family is generally well known, and there is not the same tendency to conceal the existence of these diseases among the poor as among the rich. The facts ascertained in this inquiry relate to the occurrence, in ancestors or collaterals, of *epilepsy*, *insanity*, *hysteria*, *chorea*, and certain forms of *paralysis*—viz., *paraplegia* and *infantile paralysis.* Ordinary hemiplegia was not included, because primarily due to disease of vessels, &c., and not of the nerve-tissues. Habitual drunkenness, however distinctly allied in some cases to neuroses, is too frequent among the poor to make it possible to attribute any significance to its occurrence as an antecedent of epilepsy. The proportion of cases in which evidence of inheritance is to be traced was found by Echeverra in 300 cases to be 28 per cent.; by Dr. Reynolds, from a smaller number, to be 31 per cent." Dr. Sieveking could trace *heredity* in only 13·4 per cent. of his cases.

Dr. Webster believes, from a combination of his own investigations with those of Esquirol and others, that *one-third* of the cases may be traced to hereditary descent. In Dr. Gowers' cases the proportion is higher than in the records of any other observer :—

"Of 1,250 cases in which the point was carefully investigated, 452, or 36 per cent., presented evidence of neurotic inheritance. There is no difference in the frequency of inheritance in the two varieties of epilepsy and of hystero-epilepsy : in each it is nearly 36 per cent.—that is, roughly speaking, of every eight cases of epilepsy, or of hystero-epilepsy, on an average three will present evidence of neurotic heredity, and five will not. Inheritance does not affect the two sexes equally. In the cases in which there was no inheritance, the males amounted to 48, and the females to 52 per cent. ; but, among those in which the disease was inherited, the females constituted 57, and the males only 43 per cent. It seems, therefore, that *when there is an inherited taint, the females of a family are more likely to suffer than the males.* The same fact has been noted by other observers. In examining the mode of inheritance, we obtain some light on the origin of this excess. Of the 450 cases with inheritance, the disease came from the mother's side rather more frequently than from the father's, the difference amounting to 4 per cent.—from the father's side in 35 per cent. (146 cases), and from the mother's side in 39 per cent. (175 cases), while it was from both sides in about 5 per cent. (25 cases). The side from which the disease is inherited is not unimportant, for it has a distinct influence on the occurrence of the affection in the two sexes. In the cases in which the disease was inherited from the father's side, males were in excess of the females by about 8 per cent. (76 males to 70 females), which is an excess of 14 per cent. above the average for the whole number (46 per cent. males). Whereas, in the cases in which the disease is inherited from the mother's side, females were in excess by about 14 per cent. (107 to 82). *Thus the percentage of the males affected is* 22 *per cent. greater when the disease is inherited from the father's than when from the mother's side.* The affection of brothers or sisters must be regarded as an indication of an inherited taint, although we may be unable to trace antecedent instances. In these cases (89 in number), the females are in very large excess—53 females to 36 males ; but the total number is probably too small to permit safe generalisation. We may now see, in part at least, why females preponderate so much more in the cases of inherited disease than when there is no inheritance. First, the inheritance is more frequently from the mother's side than from the father's ; and, secondly, when the inheritance is from the mother's side, girls suffer in a much larger proportion than boys. Of the relatives who suffer, the mother is herself diseased in a much smaller proportion of cases than the father. The father himself was the subject of neurotic disease in 45 per cent. of the cases in which the inheritance was paternal ; the mother, in only 35 per cent. of the cases in which the inheritance was maternal.

"Of the neurotic diseases which can be traced in the antecedents of epileptics, *epilepsy* is incomparably the most frequent. A history of epilepsy was obtained in nearly three-quarters of the inherited cases. In rather more than half (240) it existed alone, in 54 it was combined with insanity, in five cases with chorea, and in three with both insanity and chorea. *Insanity* occurred in the relatives of about one-third (157) of the inherited cases, and of these it existed alone in two-thirds

Chorea occurred in other relatives in 35 cases, in 24 of which it existed alone, in five it was associated with epilepsy, and in three with insanity. The forms of *paralysis, paraplegia,* and *infantile palsy,* included in the investigation, were not more frequent in the families with other evidence of neurotic tendency than in those who presented no such history. The cases in which an antecedent family history of *hysteria* was obtained were very few. Although the mother was herself less frequently affected than the father, the two suffered from epilepsy with equal frequency, and the difference in the frequency of their affection depended upon the fact that the mother suffered from insanity much less frequently than the father. Of 394 cases of inherited epilepsy, the father was epileptic in 45, and the mother in 46 cases; the father was insane in 21 cases, the mother in only 11. In some cases the family tendency, as evidenced by the number affected, was very strong. In one case no less than fourteen members of the family suffered from epilepsy—the patient's mother and maternal grandmother; her mother's sisters and mother's brothers, six; four of her own sisters, and five of her sister's children" (*Lancet* and *Brit. Med. Journ.*, Feb., 1880).

Dr. Gowers obtains no evidence in support of the view that rheumatism or phthisis favour the occurrence of epilepsy. In eight of the cases the patients were the subjects of well-marked inherited syphilis. In all of these cases the attacks had the aspect of idiopathic epilepsy, cases in which there were symptoms suggestive of local brain-disease being excluded. In only two of these cases did the attacks begin in infancy. In all the others they commenced towards the end of or after childhood. Inquiries were made in a considerable number of cases for consanguinity of parents, but in no instance was it met with.

(3.) *The influence of age on the occurrence of epilepsy, as evidenced by the date at which the first fit occurred in* Dr. Gowers' series of 1,450 cases, is as follows:—

"If we take, first, decennial periods, we find that, under ten years of age, more than one quarter of the cases commenced, 29 per cent. Between ten and twenty, nearly one-half of the total number, 46 per cent., began. In the next decennial period, twenty to thirty, the number falls to a seventh, 15·7 per cent. Between thirty and forty, only 6 per cent. began; between forty and fifty, only 2 per cent.; between fifty and sixty, only 1 per cent.; and over sixty, only one-third per cent. of the total number. Thus, just three quarters (75 per cent.) of the cases commenced under twenty years. A large number of cases, no less than 12½ per cent. of the whole, commenced during the first three years of life. In this group no cases of simple infantile convulsions are included, only such as, beginning in infancy, continued as chronic epilepsy. There is a difficulty in ascertaining the exact date of commencement in all cases. Of about one-third all that could be learned was, that they began in infancy. The number of cases is largest in the first year (5½ per cent. of the whole), and falls rapidly to three years of age, and then more slowly until five, when the minimum for the early period of life occurs, only 1·7 per cent. commencing. From this there is a considerable rise at seven, the commencement of the second dentition, then a fall at eight, and from this the numbers increase slowly at nine and ten, rapidly at twelve, until the maximum is reached at fifteen and sixteen, at each of which 84 cases commenced, 5¾ per cent. of the total number. From this period, we have a rapid fall to twenty-one, when only 2 per cent. of the whole commenced. During the next eight years, the number of cases commencing in each year varied from 26 to 14. Between thirty and forty years, the annual number varied between fifteen and three, and between forty and fifty it varied from one to six. Between fifty and sixty, the annual number of cases was from one to three. Over sixty, the cases were—two at sixty-two, two at sixty-four, and one at seventy-one. The proportion in which the two sexes suffer varies considerably in the different periods of life. In the cases commencing under ten years, the females exceed the males by 6 per cent. In the second decennium, between ten and twenty, the excess of females amounts to 18 per cent. In the third, between twenty and thirty, there is a great fall; but the excess of females amounts to 12 per cent. Between thirty and forty, the numbers being still smaller, the relation is reversed; the males exceed the females by 16 per cent. Between forty and fifty, with a continued fall of numbers, the excess of males is still greater, amounting to 36 per cent. Between fifty and sixty it reaches 40 per cent., and over sixty males only suffer. The latest age at which epilepsy commenced in a woman was fifty-five, whereas, in men, cases commenced at fifty-eight, fifty-nine, sixty-two, sixty-four, and seventy-one. At each of the maximum periods, at infancy and puberty, the excess of females is very great. During the later period of childhood, the numbers are nearly equal. The excess of females lessens after puberty, and ceases by middle life."

(4.) *How far does an inherited tendency influence the age at which the disease commences?*—It has usually been stated that when there is heredity, the disease begins before twenty; and at an earlier age among boys than among girls. Dr. Gowers' analysis does not confirm these statements. On the contrary, it shows that the influence of heredity continues until a late period of life, and, although absolutely greatest in youth, it is relatively only a little less during adult age than during early life:—

"About 5 per cent. began in the first year of life, and the annual number fell to a minimum in late childhood, to rise to a maximum at puberty. This maximum, however, occurred at fourteen years of age—a year earlier than the maximum for non-hereditary cases, which was at fifteen and sixteen. The cases with heredity lessen gradually during life, but continue to occur to the last. The latest case of the entire series was a man who suffered from what was apparently idiopathic epilepsy commencing at the age of seventy-one years, and in him the disease was inherited, his father having suffered from fits, clearly epileptic, for many years."

(5.) *How far does age and sex influence the occurrence of the hystero-epileptic or co-ordinated forms of convulsion?*—Of 1,000 cases in which the form of convulsion could be ascertained, it was co-ordinated in 185, or 18½ per cent. of Dr. Gowers' cases.

"At every age the proportion of females he found greater than of males. Under ten, these hystero-epileptic cases constitue 15 per cent. of the male and 18 per cent. of the female cases. Between ten and twenty the difference is, as might be expected, much greater. They constitute 14 per cent. of the male, and no less than 26 per cent. of the female cases. Between twenty and thirty, the percentage of the males remains the same (14), and that of the females falls slightly (to 21). Between thirty and forty the male cases with hystero-epileptic symptoms fall to 12 per cent., while the females present a slight rise, and constitute 24 per cent. In the male cases commencing over forty none of this character occurred; and in women, although 29 cases of the series commenced over forty, only one presented these symptoms. Thus, cases with hystero-epileptic symptoms commence almost exclusively under forty. In males, they bear at each age nearly the same proportion to the cases of pure epilepsy, decreasing very slightly from a maximum in childhood to a minimum in middle life. In women, their minimum is in the first decade; their maximum is in the second; and the proportion after adolescence remains nearly double that of males during the period in which they occur. I think, however, that some surprise will be felt at the fact shown by these figures—which my actual observation of attack fully confirms—that, even up to the fourth decade of life, one-third of the cases presenting hystero-epileptic phenomena occur in males" (Dr. GOWERS' *Gulstonian Lectures,* l. c.).

(B.) *The Exciting Causes of Epilepsy.*—On this head Dr. Gowers' statistics are full of details.

"The first point which demands attention is the causation of the large number of cases which begin in the first three years of life. Of these, 180 in number, no information was forthcoming in about one-third. In 24 cases, although information was obtained, no cause could be ascertained. In 97 cases, however, a cause for the first fit was assigned. In two, it was exposure to the sun; in one, ascarides; in seven, an acute disease; in eight, a fall; and in eight, the first fit occurred soon after birth, the labour in several of these cases having been difficult, and in some the forceps employed. In the remaining 72 cases, 33 males and 39 females, the first fits occurred during dentition, and were attributed to teething. The percentage of neurotic heredity in these cases was nearly the same as for the whole of life — 34 per cent. Thus nearly two-thirds of the cases beginning in infancy, the conditions of origin of which could be ascertained, arose from the so-called dentition convulsions. It seems legitimate to ascribe to the same cause a similar proportion of the cases respecting which no information was forthcoming. If so, we have a total number of cases due to this cause which constitutes 7 per cent. of all the cases investigated. Few persons who have attended closely to the diseases of children can fail to be convinced of the truth of the opinion long ago urged by Sir William Jenner, and now widely accepted, that almost all convulsions associated with dentition are really due to the constitutional condition of retarded development, which we call rickets, to the irritability of the nervous system which accompanies this condition. The further details of the cases I am considering fully confirm this, for in a large number there was a history of late teething, late walking, and in many of crooked limbs. Even these facts probably do not represent fully the influence of rickets in the production of epilepsy. In many of the cases just described, the course of the attacks was continuous from infancy to adult life. In others, however, there were intermissions, especially during the period of childhood. In several, for instance, the fits ceased at seven, to recommence at ten or eleven years. In 27 other cases which commenced after the period of childhood was over, repeated convulsions had occurred during the first dentition, accompanied in many with other signs of rickets. It seems reasonable to ascribe to these convulsions of infancy a share in predisposing to the convulsions of later life; and the addition of these cases raises the number, in which a causal influence may with probability be ascribed to rickets, to nearly 10 per cent. of the whole. Whether rickets is or is not entirely preventible, there can be little doubt that in the degree in which it leads to convulsions it is always preventible by proper attention to the diet and hygiene of infancy. These facts, therefore, suggest that a considerable proportion of the cases of epilepsy are really within the range of preventible diseases. They suggest, also, that inherited taint exerts the same influence here as at other periods of life, predisposing to the occurrence of convulsions in backward children, and to the persistence of the fits, and that the management of children with such inherited taint should be a subject of extreme care. The statements made by patients as to the causes of their fits have of necessity to be received with caution, and the facts carefully investigated. An occurrence so striking as a fit is naturally assumed to be due to some cause at least discoverable, and so is often attributed to a remote and improbable antecedent. Of 1150 cases in which this point was noted, a reasonable cause for the first fit was given in 428, or 37 per cent., and in the remainder no probable cause could be ascertained. From this series the cases distinctly originating in dentition convulsions are excluded.

"An exciting cause is met with in males much more frequently than in females: in 44 per cent. of men (235 out of 537 cases), and in only 31 per cent. of women (193 out of 616 cases)—a difference between the sexes of 13 per cent. This can scarcely be accounted for by greater exposure to exciting causes, since some of these act more on the female sex; it seems to indicate a greater predisposition on the part of women (or greater nervous instability?). The proportion of cases in which an exciting cause can be traced in the different periods of life is as follows:—Under ten it is nearly the same for both sexes (males 41, females 40 per cent.); from ten to twenty there is a great difference between the sexes, excitants being traceable in 19 per cent. more males than females (males 47, females 32 per cent.) In the next two decades, 20–40, the percentage in men continues the same (at 42 per cent.); but in women it rises (to 35) in the first, and falls (to 32) in the second. In the cases which occur over forty years of age the percentage in each sex is about equal (40); but in the male cases commencing over fifty, an exciting cause could be traced in as high a proportion as two-thirds. Thus the most striking peculiarity is the large proportion of cases which commence in women between ten and forty years of age without any discoverable exciting cause to which they can be ascribed. When epilepsy is the result of a powerful original tendency, it often occurs without any apparent cause, and when the patient is in his best health" (GOWERS, l. c.).

Of all the immediate causes of epilepsy the most potent are *psychical—fright, excitement, anxiety.* The effects of such causes in its production are so well known that Raphael has introduced into his picture of the Transfiguration a boy falling into an epileptic fit. To such causes were ascribed

157 cases in Dr. Gowers' analysis, more than one-third of those in which a cause was given, and one-seventh of the whole series.

"Of the three forms of emotion, *fright* takes the first place, with 119 cases, equal to 10 per cent. of the whole. The relation of this cause to age is, however, very distinct. It is effective chiefly in early life, when emotion is the most ready, and is most powerful at the transition from childhood to adult life, while after middle life it is almost inactive. Of the 119 cases only three commenced after thirty years of age, and 100 commenced under twenty. Of these, the majority, 70, commenced between ten and twenty, only 30 cases under ten. The female sex is notoriously the more emotional, and accordingly the disease (as Dr. SIEVEKING and others have pointed out) resulted from *fright* in a larger proportion of females than of males, although the difference is, perhaps, less than might be expected—71 females and 48 males, 60 and 40 per cent. respectively. It is notorious also that the difference between the sexes in *emotionality* increases as life advances. In childhood one sex is as emotional as the other, but with puberty men become far less emotional than women. *The influence of fright as a cause of epilepsy* is in *strict harmony with this fact.* Under ten years of age, the sexes suffer equally. Between ten and twenty the males are to the females as 3 to 4; between twenty and thirty as 3 to 13, and over thirty the only cases due to this cause occurred in women. After seventeen only three cases occurred in men, while twenty occurred in women. The influence of fright is accurately recognised in popular phraseology, and is easily comprehensible when we consider how powerfully fright affects the nerve-centres. The stimulation of the motor centres shows itself in the sudden muscular actions, such as *the start of alarm*, which, no doubt, originates in the need for immediate action which danger often entails. The *tremor*, which often persists, indicates the enduring disturbance of the nerve-centres. In the organic nerves the vaso-motor centre is first stimulated and then inhibited, the sphincter centres are released from control, the sweat nerves are excited, and the heart's action is accelerated, perhaps, by inhibition of the vagus, and the last effect is the most constant, the most ready. The exact form of fright varied, of course, in different cases, but the list teaches many lessons. In no less than eight cases the cause was some *stupid practical joke*, as a pretended ghost. In two cases children were shut up in dark cupboards. In five cases the fright was from an alarm of fire, in six from burglars. In six cases the patients had watched other persons in fits. Three resulted from an alarm during severe thunderstorms. One case was that of a soldier who had his first fit a few hours after being terrified, while on sentry duty, by the unexpected appearance of some goats. Of 76 cases in which the interval between the fright and the fit was noted, the fit occurred *immediately* in 28, or one-third; *within a few hours* in 16. The interval was between a day and a week in 19, and it was more than a week in 13.

"As might be anticipated, in a considerable number of cases the fits were of the hystero-epileptic type. Of 90 cases, in 61, or two-thirds, they were purely epileptic; in 29, or about one-third, they were hystero-epileptic. The difference between the two sexes in the frequency with which hystero-epilepsy succeeds fright is not great. Of 51 cases in females, 18, or 35 per cent., were hystero-epileptic. Of 39 cases in males, 11, or 28 per cent., were of this type. A relation can be traced between the interval that elapses before the fit and the character of the attack.

The longer the interval the smaller is the proportion of hystero-epileptics. In the cases in which the interval was more than a week, only 18 per cent. were of this character; in the cases in which the interval was less than a week, but more than a day, the percentage of this form was 21. In the cases in which the interval was only a few hours, the percentage was 25, while in those in which the fit occurred immediately no less than 57 per cent. were hystero-epileptic.

"Other forms of sudden emotional excitement caused the first fit in nineteen cases—nine males and ten females; and these cases also occurred during the emotional period of life, under thirty years of age. *Anxiety* was the only traceable cause in twenty-nine cases, and in these males preponderated, sixteen males to thirteen females, and seven occurred over thirty years, six of these being men.

"Next in numerical importance among the exciting causes of epilepsy are *traumatic influences, blows* and *falls on the head.* To these, after the elimination of doubtful instances, 65 cases were due. A third (21 cases) occurred under ten years, rather more than a third (25 cases) between ten and twenty. Males are affected by this cause more than females, the numbers being 39 to 26. This is clearly due to the risks of occupation, for in the cases commencing before ten years the females were in excess. Between ten and forty the male cases in each decennium were rather more than twice the number of the females. Over forty the only cases were in men.

"*Hystero-epilepsy* resulted from a traumatic cause in only a small number of cases—6 out of 44; and in estimating the significance of these, it must be remembered that *traumatic causes often involve intense emotional disturbance.*" [As also does a narrow escape from almost inevitable death by accident.]

"In two-thirds of the cases the injury was a fall on the head, in one-third it was a blow on the head. In most of the cases the patient was stunned for a time, but in only a fifth (eight cases) did the first fit occur immediately, in the rest an interval elapsed; in one-third of the cases the interval between the injury and the fit was more than a day and less than a week; in another fifth the interval was between a week and a month, and in about the same proportion the interval was more than a month. *Exposure to the sun* was given as the cause of the first fit in a considerable number of instances, and in 27 it seemed probable that the statement was correct. From this cause, as may be expected, males, from their greater exposure, suffer most: 20 were males, and only 7 females. The cases were pretty equally distributed through life, but the cases in females were relatively most numerous in childhood, and decreased in adult life, to cease at thirty. The form of convulsion was almost always *pure epilepsy*, only one case presented *hystero-epileptic symptoms.* In ten of the cases there was a distinct attack of *sunstroke.* In ten the fit occurred immediately; in several others in which an interval elapsed, this was marked by some form of nerve disturbance, such as *tremor. An acute disease* was the apparent cause of the first fit in 37 cases (14 males and 23 females), and in more than half (19 cases) the disease was scarlet fever; in a quarter of the cases (nine) it was measles; in five cases it was continued fever, called typhus, typhoid, or low fever; in one case it was rheumatic fever; and in one a severe attack of diarrhœa. The remarkable influence of *scarlet fever* in the causation of epilepsy demands further consideration. The cases due to this all commenced during early life, under seventeen. It may be thought that the first fits in these cases were due to uræmic poisoning, but this is not supported by the particulars ascertained regarding fourteen cases. It is true that in three of these all that could be learned was that the first fit occurred during scarlet fever, accompanied with dropsy, and in two others that the fits occurred after an attack of scarlatinal dropsy was over. But in two other cases in which the scarlet fever was succeeded by dropsy, the first fit occurred after the fever, before the dropsy. In six cases there was no dropsy; in two of these the first fit occurred after the fever was over, in one during convalescence, and in one a month later. In three cases the first fit occurred during the height of the fever, and in one convulsions ushered in the disease, persisted throughout the attack, and continued afterwards as permanent epilepsy. In only one case did ear disease also result from the fever. The urine was examined in most cases, but no albumen was found. These facts suggest that the first fits were due to some peculiar effect of the scarlet fever poison upon the nervous system. It may be remembered that the scarlet fever poison has a very wide-spread action in the system, and that, after scarlet fever, optic neuritis, leading to subsequent atrophy, may occur, without renal or coarse cerebral disease. *Intestinal worms* rank low as a cause of persistent epilepsy, but were noted in six cases, the convulsions continuing long after the worms had been expelled. In five cases the first attack was attributed to digestive derangement. In one case asphyxia, and in nine immersion in the water, caused the first fit. Certain *toxæmic influences* were the apparent cause of the disease in several cases. Of these, *chronic alcoholism* was the cause in a comparatively small number—thirteen cases only. In some the first fit occurred during intoxication, and in a few the attacks recurred after each alcoholic excess. Six patients were the subjects of *chronic lead-poisoning.* In two of these there was also *chronic renal disease.* In one of the others the epilepsy had existed from infancy, but became much more severe when the *plumbism* was developed. In the remaining three the lead-poisoning was the apparent cause of the attacks, which resembled, in character and course, those of idiopathic epilepsy. It is well known that acute lead-poisoning may be accompanied by convulsions; that chronic lead-poisoning may cause chronic convulsions precisely like those of ordinary epilepsy, has been pointed out by others, but is not, I think, generally known. The list of nervous disturbances which may result from lead is a long one, and includes acute and chronic mental derangement, neuralgia, brachial spasm, as well as palsy, and muscular wasting and optic neuritis. It is known that lead is to be found after death in the brain in considerable quantity. I have mentioned that in two of the cases of lead-poisoning there was also consecutive renal disease. In two other cases of chronic renal disease, convulsive attacks, resembling idiopathic epilepsy, brought the patients under treatment. In one, the absence of any coarse changes in the brain was proved *post-mortem.* It appears, therefore, that such convulsive attacks may occur and recur for months without other sign of uræmic mischief. Several cases in which chronic mental derangement resulted from Bright's disease have also come under my notice. I have met with no case which suggests that *tobacco-smoking* exerts any influence in the production of epilepsy; but in one case the fits were apparently due to the patient, a lad, having shortly before commenced working in a *tobacco-factory,* where his occupation caused frequent nausea. In one case the first fit occurred after the administration of *chloroform;* and, in one, attacks, which had ceased for many years, recurred after the inhalation of *nitrous oxide. Of sexual processes* retarded or absent menstruation coincided with the first fits in a large number of the cases which commenced in girls between fourteen and seventeen; but the difficulty in determining the exact causal relationship between the two conditions is very great. Epilepsy once set up in such cases, the subsequent establishment of regular menstruation appears to exercise very little influence upon the fits."

Of other exciting causes "there are certain vices," writes Sir Thomas Watson, "which are justly considered as influential in aggravating, and even in creating, a disposition to epilepsy. Debauchery of all kinds, the habitual indulgence in intoxicating liquors, and, above all, the most powerful predisposing cause of any, not congenital, is *masturbatian*—a vice which it is painful and difficult even to allude to in this manner, and still more difficult to make the subject of inquiry with a patient. But there is much reason to be certain that *many* cases of epilepsy owe their origin to this wretched and degrading habit; and more than one or two patients have voluntarily confessed to me their conviction that they had thus brought upon themselves the epileptic paroxysms for which they sought my advice," (Lect. xxxvi.) Sir Charles Locock attributes the great increase of the disease during the later years of his experience to this cause (*Med. Chir. Society's "Report;" Medical Times and Gazette*, May 23, 1857.) On the other hand, there is a want of direct proof that epilepsy and masturbation have any special relation to each other. On this point Dr. Reynolds observes "that the one is a tolerably prevalent disease, and the other a very widely distributed vice. There are multitudes of epileptics with regard to whom no such suspicion could ever be entertained; and there are much larger multitudes of masturbators who have never become epileptic. When, therefore, we find the two elements combined in the same individual, it is necessary to observe some caution in our attempt to interpret their relations." The influence of excessive venery or of masturbation in the production of epilepsy has, no doubt, been overstated. The serious nervous disturbances to which both of these habits give rise, is of a different nature from those of epilepsy. On this point Dr. Gowers' analysis shows that, in boys who are epileptic, masturbation is unquestionably common; but here again it is most difficult to decide to what degree it can be regarded as a cause of the disease, and it was distinctly the cause in only a small number of cases.

With regard to other exciting causes, Dr. Gowers states that:—

The first fit occurred in seven cases *during pregnancy*, without other obvious cause, and in five cases the disease commenced *after parturition.* No facts which have come under my notice suggest that acquired *syphilis* is a cause of idiopathic epilepsy. There remain for consideration two conditions, occasionally associated with epilepsy, in some cases causally, in others probably only by coincidence, or as the effects of a common cause—these are *heart disease* and *chorea.* An abnormal condition of the heart was noted in 93 cases; but this does not represent the proportion of the whole in which abnormality existed, since in many its condition was not noted. I have, however, rarely failed to examine the heart in epileptics, and I think that the proportion of cases of valvular disease is not far below the mark. In eight cases, there was extreme frequency of action; in nine, there was irregularity; in thirteen, all young persons, there was reduplication of the first sound. In thirty cases there was valvular disease; mitral regurgitation in twenty; mitral constriction in seven; aortic regurgitation in three; and aortic constriction in one. There was obvious dilatation without valve disease in twenty, and considerable hypertrophy without renal disease in two. The cases of irregularity and reduplication were all observed within a comparatively short space of time, and I am sure that, had my attention been earlier directed to their frequency, I should have found them in a larger number of cases. In a few cases it seemed that the heart disease and epilepsy were in accidental association. In some cases, the cardiac condition, especially simple dilatation and reduplication of the first sound, may have been a consequence of the repeated strain to which the heart had been subjected during the attacks. But in other cases, especially of valvular disease, there was reason to believe that the heart disease existed before the epilepsy, and in many there was no other etiological condition to which it could be ascribed. In twenty cases the patients had also suffered from *chorea.* The relation between the two diseases was as follows:—In eight cases, epilepsy existed before the chorea. In twelve, the chorea occurred first; in four of these, the fits began at the time of the chorea, and persisted afterwards. In only two of these was there heart disease. In five cases the fits occurred long after the chorea, the interval being four, six, twenty-seven, thirty, and forty years respectively. In one case, peculiar choreic convulsions occurred at the height of the chorea, but passed away as the disease lessened, and did not recur. It seems probable in the cases in which the fits immediately succeeded the chorea, and possible in the cases in which an interval elapsed, that the impaired nutrition of the motor centres during the chorea may have left a predisposition to further disturbance under the action of some other exciting cause.

There are also cases referred to causes of an eccentric or peripheral nature, such as to uterine or ovarian irritation, which are thus said to act upon the brain through the medium of the nervous system, in some way as yet unknown—*reflex epilepsy.*

Dr. Todd developed a theory of the disease, suggested by its occasional occurrence with renal affections. He held that the peculiar features of an epileptic seizure are due to the gradual accumulation of a morbid material in the blood, until it reaches such an amount as to operate upon the brain, as it were, in an explosive manner. In other words, the influence of this morbid matter, when in sufficient quantity, excites a highly polarised state of the brain, or of certain parts of it, and these discharge their nervous power upon certain other parts of the cerebro-spinal centre, in such a way as to give rise to the phenomena of the fit. This theory assumes that the essential derangement of health in epilepsy consists in the generation of a morbid matter which infects the blood, and it supposes that this morbid matter has a special affinity for the brain, or for certain parts of it, just as *strychnine* exercises a special affinity for the spinal cord. According to this theory, the disease ought to have found a nosological place amongst the *constitutional diseases.* But to give a definite character to such a humoral theory, it were necessary to discover some morbid matter in the blood in every case of epilepsy, which has been only partially discovered in the connection of convulsion with renal disease, when the excretion falls below a certain point and urea accumulates in the blood. Nevertheless, this is the essence and primary idea embodied in the theories of Robertson and Hughlings Jackson (see page 156, *ante*), as to the occurrence of the convulsions.

(*b.*) *Morbid Anatomy.*—It is rather by inductions based on minute clinical analysis, and a study of individual cases with a statistical analysis of the phenomena of numerous cases, that we can hope to gain an intelligent knowledge of any changes which may underlie such an apparently functional disease as epilepsy. Most careful *post-mortem* examination in prolonged cases has generally failed to reveal any constant lesion with which symptoms can be connected. From pathological anatomy and experiment we have learned something of the coarser pathology of convulsions; but our knowledge has been rather built up on careful clinical observation and analysis of cases (as in the model records of Dr. Hughlings Jackson), combined with the well determined facts in physiology. How successfully numerical analysis may be applied to elucidate the pathology of convulsive diseases may be seen in the works of Drs. Reynolds and Sieveking; and in the recent exhaustive analysis of Dr. W. R. Gowers, as set forth in his Gulstonian Lectures of Feb., 1880, from which so much precise, suggestive, and trustworthy materials are here made use of. The tendency of modern pathology has undoubtedly been to connect the epileptic seizure with a variety of anatomico-pathological lesions and conditions of a variable and inconstant kind. Epilepsy has thus been regarded as an intrinsic disease of the brain, because its most constant and marked groups of phenomena are referable to the functions of the central parts; while the loss of consciousness, associated with excessive mobility, suggested the *central ganglia* or *medulla oblongata,* as parts where, in future, morbid anatomy may yet discover a lesion.

On the other hand, epilepsy in very many cases has been regarded as "a disease of the whole man, and not of any one organ or system of organs alone;" and when lesions of a more or less constant kind were found in the brain, they were regarded as standing in the same relation to epilepsy that morbid states of the kidney do in relation to the whole phenomena of Bright's disease regarded as a *constitutional disease.* Epilepsy, therefore, would be regarded in this respect as a *constitutional disease,* with intervals of apparent freedom, and with times at which the disease culminates in the characteristic phenomena. In order fully to appreciate the nature of epilepsy, as Dr. Sieveking pointed out, a careful study of the general condition of each patient is necessary, and especially a study of the phenomena which may show themselves in the intervals of freedom from the paroxysms. Nutrition changes in epilepsy may be connected with a constitutional state of ill health, such as gout, scrofula, or the like, the paroxysms having a remote origin in the state of the blood; and when once set up, they bring the *medulla oblongata* and *upper* part of the spinal cord into that condition of excessive and perverted functional activity which maintains the epileptic state. Arterial spasms, and the consequent cerebro-spinal anæmia, is believed to be the first step of this disordered movement. The pale face, the loss of consciousness, the contraction of the muscles of the chest and larynx at the commencement of the fit, have all been regarded as pointing to this cerebral condition—

mainly on the assumption that pallor (or vaso-motor spasm) of the face is evidence of pallor or vaso-motor spasm of the brain.

By clinical observation and experiment it has been also presumed by Niemeyer and others, that the excitement of the motor nerves, of which the convulsions are the exponent, proceeds from the *medulla oblongata* and the portion of brain lying on the base of the skull:—(1.) By the interruption of the functions of the hemispheres which accompanies the convulsions. It is not probable that motor impulses proceed from the hemispheres at a time when the irritability of the other ganglion-cells and nerve-fibres is extinguished. (2.) Convulsions, similar to epileptic convulsions, can be excited by continuous excitement of the basilar portion of the brain by means of the induction apparatus, while no such result is obtained by a like irritation of the various parts of the hemispheres. (3.) Küssmaul and Tenner, in their experiments upon animals, could still produce convulsions of a decidedly epileptic character after extirpation of both hemispheres. (4.) Schroeder von der Kolk has found that, in all bodies of epileptics where the disease has been of long standing, besides numerous inconstant lesions, there was always a dilatation of the arterioles and capillaries of the medulla, with thickening of their walls. He believes that epileptic convulsions depend mainly upon an increased afflux of arterial blood to the medulla oblongata. There is no doubt, moreover, that the morbid irritability of the medulla which occasions epilepsy may arise without any increase or diminution of its supply of blood, merely from the improper character of its nutriment and from the admixture of certain materials in the blood. It must also be admitted that the *medulla oblongata* may be thrown into an irritated condition by the transmission of a morbid impression from remote regions of the nervous system, whether central or peripheral. It is well known that *neuromata* and *cicatrices*, or *tumors* pressing upon peripheral nerves, have sometimes occasioned epilepsy, and that the epilepsy has ceased after section of the affected nerve, or after removal of the cause. Perhaps, also, cerebral tumors and other diseases of the brain and spinal marrow may induce epilepsy in a similar manner, by gradual transmission of a morbid irritability to the *medulla oblongata.* This supposition has received strong support from the result of recent experiments by Brown-Séquard, in which dogs, whose spinal marrows had been injured, suffered from convulsions, although not immediately, but some time after the receipt of the injury.

It has been affirmed that in fifteen out of twenty cases in which the brains of epileptic patients have been examined, the structure of that organ has been in every respect healthy. Even when the patient has died during the paroxysm, the brain has in many instances been found ongested only. Epilepsy has been therefore regarded as a functional disease, the particular seat of lesion not being determined.

But although epilepsy may exist without any obvious disease of the brain or of its membranes, it must be admitted that the brain and its membranes are occasionally found in various states of disease. Thus, the membranes may be inflamed, thickened, or ossified, and with every variety f exudation; or the substance of the brain may be indurated or softened —the seat of abscess, of cancer, of tubercle, or of other structural disease. Any such structural disease is then considered to give rise to the epileptiform attack. The epileptic state was shown by T. and C. Wenzel to have been invariably found associated with a morbid state of the *pituitary* body in the *cella turcica*—a spot of the encephalic region very rarely examined in *post-mortem* examinations. On the other hand, Niemeyer states (very laconically) that "alteration in the appendages of the brain, which Wenzel mentions as a constant lesion in epilepsy, is absent in the majority of the cases." Since 1869 *post-mortem* examinations have been made on twenty-seven male and seven female epileptics at the Hildesheim Asylum, especial attention having been directed to the state of the *hippocampi.* In only six cases was any change, consisting of abnormal hardness or considerable shrivelling of one or both *hippocampi,* found to have occurred (HEMKES, quoted by COBBOLD, in *London Med. Record,* March 15, 1878). In 1868 Professor Meynert described twenty cases of epilepsy in which pathological changes had been found in the cornu Ammonis, and expressed his opinion that such changes would be found to be very common in that disease. Charcot has since shown that such is not the case; and now Hemkes confirms his opinion; so that these changes have no real significance.

Finally, as regards (*a.*) *Causation,* and (*b.*) *Morbid Anatomy* of epilepsy, Dr. Hughlings Jackson writes that:—

> "There is a tendency to fall back on the nearest most striking event, and to explain away the symptoms by fright, by temporary dyspepsia, by overwork, and so forth. We have not yet discovered the cause of epilepsy proper in any sense of the word 'cause,' nor is there any morbid anatomy of it. We note carefully the facts of the above and other so-called causes, but until we find some morbid anatomy, we are not justified in anything more than stating, as an hypothesis, that epileptic fits occurred from this or that condition or circumstance. And when we have found the morbid anatomy, we shall then have to discover how fright, dyspepsia, &c., produce the unstable condition of nerve-cells, on the discharge of which the first and every subsequent fit depend. The hypothesis is stated that such causes as fright are simply 'exciting causes' of the first discharge of nerve-cells which have, by prior pathological processes, reached a high degree of instability.
>
> "As regarding pathology, there is a negative pathology: *epilepsy proper, slight* or *severe,* is rarely owing to gross organic disease, whilst *epileptiform seizures* frequently are. The changes in epilepsy proper are minute, as is evident enough by their having never been found. When opportunity serves, we ought to look into the state of each tissue of the part. For here comes the great question, Is the change primarily arterial and secondarily nervous; or is it a change beginning first of all in the nerve-cell? There are many nervous diseases which are not nervous at all in the sense that the nervous elements are first to go wrong. In most cases of nervous diseases of which there is an anatomy, the nervous elements are quite innocent; they suffer because their arteries are blocked up, or break because tumors grow in their connective tissue, &c. This leads to the question of hereditariness, and thus to the question of diagnosis of epilepsy. It is not denied that epilepsy is a disease of some nervous organ. Whatever view anyone takes of the seat of epilepsy, he believes some nervous centre or centres to be affected. But now comes the critical question. There being several ingredients in any nervous organ, which one is primarily affected? That the nerve-cells are changed no one denies; but, repeating the above question in two questions, we ask, Does the

change begin in the nerve-cells of the centre, or do these suffer secondarily from a change beginning in some non-nervous element of the centres? The answer can only be hypothetical in cases of epilepsy proper, for the simple reason that as yet there is no morbid anatomy of epilepsy proper. Let us see how the two questions stand with regard to current notions of inheritance of nervous diseases. Supposing there were nervous diseases in many of the patient's near blood-relatives, we must be careful how we interpret this evidence as showing that his epilepsy is a nervous affection. We must consider what sort of pathology these 'nervous diseases' have. Are they nervous in the sense that the morbid process begins in the true nervous elements of the centres diseased, or only in the sense that it begins in the non-nervous element, and leads to damage of the true nervous elements? The fact that his relatives had hemiplegia or apoplexy from cerebral hæmorrhage, is simply no evidence that he inherits a disease of the nervous system in the sense that the changes begin in the nervous elements of nervous organs, for the very plain reason that the pathological processes causing paralysis and apoplexy are of a non-nervous kind; if they have a bearing, as I think they have, they point unmistakably to arterial or cardiac changes. But then the nervous affections in the epileptic's family may be such as chorea, neuralgia, migraine, or epilepsy proper. Since nothing is decided as to the pathology of any one of these affections, we cannot possibly say that they point to an inheritance of epilepsy, or tendency thereto, in the sense that they point to changes *beginning* in nervous elements. The hypothesis in possession is that epilepsy is owing to changes beginning in the nervous elements of nervous organs, and it is supposed to be supported by the fact that there are often in the patient's family persons who have that or some other neurosis—that is, some other nervous affection of which there is no morbid anatomy. Why is it that we speak with most confidence of the inheritance and interchangeability of those very diseases of which there is no morbid anatomy? The occurrence of these neuroses cannot be adduced as evidence that epilepsy is a disease primarily nervous. I wish to put forward, again, another hypothesis—viz., that the cells suffer secondarily, as a consequence of arterial disease; that there is thrombosis or embolism of small arteries in most cases of epilepsy proper. I have held this opinion since 1864, as regards those epileptiform seizures in which there were no signs of gross organic disease. It first occurred to me on finding epileptiform seizures in patients who had valvular disease of the heart. I have since applied it to epilepsy proper. It may be said, 'Your statement is only an hypothesis.' So is any other view which can be taken of a disease which has no morbid anatomy" (l. c., Jan. 25, 1879).

Amidst so much uncertainty and contradictory statements regarding the causation and morbid anatomy of epilepsy, it is gratifying to look forward to the extension of knowledge which we may confidently hope to acquire in the new era in the study of this disease, which may be said to have commenced with the investigation *of the modes of onset of* those attacks which begin deliberately, combined and compared with that of the subjective sensations, by which attacks commence that are less deliberate in their onset. This era was opened up by the investigations of Dr. Hughlings Jackson, already so often referred to, and by Lockhart Clarke, Alexander Robertson, of Glasgow, and Broadbent, of London, supplemented and illustrated by the experiments of Ferrier in this country (see pp. 84, 154, and 156, *ante*).

Scientifically, epilepsies are to be considered on the hypothesis that the paroxysm of each is dependent on a sudden temporary excessive discharge of some highly unstable region of the cerebral cortex. There is, in other words, in each epilepsy a "discharging lesion" of some region of the cortex cerebri (HUGHLINGS JACKSON). This "discharging lesion" results from a group of cells whose functions are excited to such discharge when their instability has been raised far above the normal—the term "discharge" being used as a short expression for the sudden liberation of nerve force, without any theory as to the form of physical or vital force thus liberated. It is in the study of epilepsies as departures from normal states that the investigations of Hitzig and Ferrier have so much value. The *clinical* and *medical* value of these researches are incontestable, whatever interpretation may be placed upon them by physiologists, psychologists, phrenologists, and others who may, for many and varied reasons, be interested in them, and value them for the facts which they establish. By faradic or galvanic excitations of the *cortex cerebri* in dogs and other animals (*i.e.*, by artificial rousing up or excitement of the functions of particular parts of the encephalon in the lower animals by these methods) convulsive seizures can be produced which are as closely like epileptiform seizures in man from disease in the same cerebral region as we could expect, considering the difference between the natural movements of men and animals. Dr. Hughlings Jackson very strongly urges the importance of Ferrier's investigations of the sensory regions of the brain, already fully referred to in previous pages (p. 57, *et seq.*) His conclusions harmonise so well with clinical observation that they are of inestimable value in giving method to investigation. "It is quite certain," says Dr. Hughlings Jackson, "that there are paroxysms, consisting chiefly of sensory symptoms, which pair off with unilaterally beginning convulsions, and which are strictly analogous to them. At any rate Ferrier has given a new starting point for the methodical and the scientific investigation of a most important class of paroxysmal affections which are, in a scientific regard, epilepsies. There are also cases of epilepsy in which, although the paroxysms are for the most part motor (ordinary epileptic convulsions) the '*warning*' of the fit is by some crude sensation—*e.g.*, by '*noise in the ear*,' or by some '*taste*' (this is very rare indeed), or by '*coloured vision*,' or by a '*strong smell in the nose*.' In each patient the '*warning*' is always the same crude sensation; and very often the patient has, besides severe fits heralded by his crude sensation, slight paroxysms consisting of little more than that 'warning.'" The warnings also take the form of mental phenomena—ideas, for example. (HUGHLINGS JACKSON, *Lectures on the Diagnosis of Epilepsy*, Harveian Society, *Brit. Med. Journ.*, Jan. 11, 1879). It is here assumed that the whole of the chief organ of mind, the cerebrum—is, like all other nervous centres, made up of hystological nervous arrangements, representing functionally "impressions and movements." The whole of the brain has this kind of anatomico-physiological constitution. The *organ of the mind* (not MIND itself) is thus constituted (see p. 65, *ante*). "Of what other materials can the *organs of mind* be composed than of processes representing both movements and impressions?" There are thus numerous epilepsies, the kind of paroxysms differing according to the particular region of the cortex affected; and since many regions (if not any region of it) may be affected, the number of different (scientific) epilepsies is great (HUGHLINGS JACKSON, l. c.)

Practically, at least two forms of epileptic seizures are to be recognised, namely—(1.) *Lapses of or defects of consciousness only* (*epileptiform seizures*); (2.) *Lapses of consciousness, with the accompaniment of more or less convulsive movements—epilepsy proper. As to Consciousness,* it is lost at first, or very soon, in the paroxysm of *epilepsy proper;* and late or not at all in the *epileptiform* seizures. To say that an epileptic paroxysm begins with loss of consciousness is equivalent to saying that the epileptic discharge begins in some part of the highest of the cerebral centres—*i.e.,* "in the latest centres," and, anatomically speaking (see pages 62 to 82, *ante*), the "latest developed centres" (both individually and in the series of organisms); physiologically speaking, "in those centres which are the seat of the activities during which our present highest consciousness arises" (HUGHLINGS JACKSON). *As to Spasms or Convulsions,* they begin more nearly bilaterally in epilepsy proper—are more nearly universal at once; are more rapid and sooner over. In the epileptic seizures they begin very locally on one side—hand, side of face, or foot; and become universal more gradually, the extension of spasms being comparatively slow, especially at the outset.

As to Seat of Change, the hypothesis of Dr. Hughlings Jackson regarding epilepsy proper is that the change exists in some part in front of Hitzig and Ferrier's region, or behind Ferrier's sensory region—*i.e.,* on disease in some part of the highest cerebral centres, and that lesions of the convolutions stand incomparably first as a cause of convulsions. The spinal cord acts only as a conductor. The over-acting grey matter is to be sought for above it in the skull. The teaching of *experiment* is that both the cortex and the medulla may originate convulsions; the teaching of *pathology* is that epileptiform convulsions have their origin at the surface of the brain (WILKS). But Dr. Gowers "doubts whether the pathological facts alone, or in conjunction with experiment, quite warrant the conclusion that epilepsy is a disease of the convolutions." It has also been shown by Dr. Burdon-Sanderson (page 64, *ante*), as probable, that the convulsions which occur, when the surface is irritated, may depend on the discharge of motor centres more deeply seated, though connected with, and excitable from, the surface regions. Hence these deeper structures at the level of the corpus striatum, or even lower still, may be the parts which discharge in epilepsy. And if the over-action of motor grey matter is the only certain element in the convulsion, such over-action may disturb the centres above as well as the centres below. Thus the loss of consciousness may be the effect of an upward discharge, just as the muscular spasm is the effect of the downward discharge, as Dr. Robertson of Glasgow has suggested; and according to Nothnagel there exist in the medulla oblongata, adjacent to the centres which regulate the respiratory movements and the state of the vessels, structures capable of giving rise by their action to general convulsions, "the convulsive centre," and this he regards as the seat of the disease in epilepsy. Dr. Gowers shows how clinical experience carries us further than pathology and experiment towards a definite conclusion, especially from the study of the modes of onset of the convulsions; and shows that in some cases the seat of the disease must be higher than the medulla oblongata. These are the cases in which the auræ refer to the special senses, or consist in mental processes. Such special-sense warnings are found present in one-fifth of the cases—*i.e.*, the discharge commences in a special sense centre, and therefore originally in the hemispheres. Those commencing with an intellectual auræ—an idea—must emanate from the highest of all the cerebral centres, that which constitutes "*the anatomical substratum of intellectual processes*," according to the phraseology of Spencer and Jackson.

As to Abnormal Physiological Condition of Centres.—In each form of epilepsy there is of necessity increased instability of cells, since there occasionally occur excessive liberations of energy; in each there is a "discharging lesion, which may happen in any of the grey matter of the encephalon which subserves sensori-motor processes." An epileptic is a person certain of whose cortical nerve-cells have become highly unstable; and in their discharge, the movements or impressions they represent are excessively developed. There is, then, no essential difference in the abnormal physiological condition in cases in the two types; yet the paroxysms of epilepsy proper are more "intense" (more spasms in a short time) than those of epileptiform seizures, and this possibly depends in part on the fact that the highest centres have most small cells.

As to Pathology.—In each there is increased nutrition; the excessive liberation of energy in the paroxysm of necessity implies increased taking in of nutrient substances having potential energy; increased nutrition is the other side of high instability. We have as yet little more than this very general, but inevitable, inference as to pathology in either. How the increased nutrition is brought about we do not know in most cases; in some cases, it is determined by tumor.

As to Morbid Anatomy, we can say that epilepsy proper is rarely caused by gross organic disease; but epileptiform seizures are often so caused, syphilitic gross disease in particular. Sometimes, however, in cerebral tumor there are attacks difficult to distinguish from the epileptic vertigo of epilepsy proper.

Of course, the gross organic disease only "causes" the seizures in the sense that it leads to high instability of cells in its neighbourhood; it acts like a foreign body. The probability is that it leads to the hyperphysiological condition of instability, by producing a subinflammatory change, and thus increased nutrition. This difference as to morbid anatomy in the two seizures can only mean either that gross organic disease most often occurs in some particular places, or that in some particular places it more easily leads to instability of nerve-cells. Thus epileptic or epileptiform attacks without loss of consciousness point to gross organic disease, such as tumor of the brain, as Bright and Wilks long ago pointed out.

The conclusion arrived at by Dr. Gowers is—"that all the phenomena of the fits of idiopathic epilepsy may be explained by the discharge of grey matter; that the hypothesis of vascular spasm is as unneeded as it is unproved; that there are no facts to warrant us in seeking the seat of the disease elsewhere than in the grey matter in which the discharge commences; that this is in most cases within the cerebral hemispheres, although possibly, in some instances, lower down in the medulla oblongata; that epilepsy is thus a disease of grey matter, and has not any uniform seat. It is a disease of tissue, not of structure."

Symptoms.—Epilepsy may be grave or slight, so slight that it may be but a sudden and very transient loss of consciousness, closely followed or accompanied by but little spasm, and that of small muscles, signified by pallor of the face (due to spasm of facial arteries), slight spasm of the eyes, hands, and the like. Such spasms, while limited in degree, are still widely scattered, affecting parts in many different regions.

"There is even a kind of spasm which is not obvious. A '*vacant*' look may depend on slight rigidity of the facial muscles. A patient is sometimes said 'to look vacant,' or 'look fixedly.' The so-called 'vacant look' in a slight seizure may signify slight, even-spread facial spasm. It may also signify relaxation of the facial muscles. In some seizures there is at the onset a real expression, which cannot be put down either to a wave of facial spasm, or to a relaxation of facial muscles. There is a very definite emotional expression; one of fear is common. We not uncommonly hear a mother assert of her epileptic child's slight attacks, 'She looks frightened, and runs to me.' There is also the antithetical expression of indignation in other cases. There are minor degrees of each emotional expression, as *astonishment*, '*looking startled*,' &c. It is important to notice these expressions; for possibly they will help us to account for the 'form' of the actions which in some cases follow seizures. Again, there may be more spasm in slight seizures than we see. If we look at the face and limbs only, we may see very little, although the respiration is altogether suspended, as is shown by dark lips; we infer that there is spasm either of the glottis or of other parts of the respiratory apparatus. The slightness of spasm in some slight seizures is occasionally shown by the persistence of movements, which widespread spasm would render impossible. In slight seizures, the patient may go on walking apparently well; he may sway about, but, so far as he does walk, so far is spasm absent from the parts concerned in locomotion. Then, on the other hand, there are conditions which are, or simulate, loss of power. If the attack be sudden, the patient falls down; in some cases he drops what is in his hands; the head drops when the patient is sitting; it is not pulled down by spasm; he may look like a person going off to sleep in a chair. These conditions are not easy of explanation; to say that a man falls or drops things because he loses consciousness is an explanation which explains nothing, and may pair off with such an 'explanation' as that chorea is due to 'disorder of volition.' He may fall, either because some muscles overcontract and destroy his equilibrium, or because he loses power in some. There are certain movements occurring in slight epileptic seizure which should be distinguished from spasm, either tonic or clonic. A common one is a movement of mastication; another is a swallowing movement; there is such an one as rubbing the hands; and there is sometimes writhing of the arms during suspended respiration. To call them movements is to tacitly deny them to be spasm" (HUGHLINGS JACKSON, l. c.)

The following are some of the names which patients or their friends give to slight epileptic seizures :—"*sensations*," "*turns*," "*spells*," "*thinkings*," "*stupidities*," "*reveries*," "*forgetfulness*," "*faints*," "*giddiness*," "*becoming absent*," "*quiet faints*," "*comes over queer*," "*feels as if I was like lost—as if I were in some strange country*," "*dropping off to sleep in the most extraordinary way*," "*in a deep study*," "*sitting as if he were thinking*." The slightest of these so-called "*sensations*" (or other names) are often the sole seizure for weeks, or months, or years before the severe fits come on. Of such slight "*fits*" no notice is taken by the patient, who may say nothing of them till he is questioned when severe fits follow them. He dates his illness from the first severe fit; and if the slight and severe are mixed up together, he only takes notice of the severe ones; and calls the others "*symptoms*" or "sensations"—"*queer sensations which he can't describe*." Such sensations are really slight, incipient, or abortive fits. In such slight paroxysms no special investigations can be made. Thus there may be epilepsy proper, *without* loss of consciousness. There may be defect of consciousness only,—the patient may not know at the time where he is; he may hear people talking, but cannot tell what they say. He is defectively conscious to the extent of being, in common language, "*confused*." He has become so suddenly, and remains so for a few seconds only. "To be confused" and "to be defectively conscious" are thus synonymous expressions. There is also other evidence of an epileptic patient not being absolutely unconscious in a paroxysm. His words may not only be incoherent; but if he is asked a question he replies to it vaguely, or he may reply quite correctly. According to the report of the patient, there may have been no affection of consciousness at all; but a common precursor of the affection of consciousness in a paroxysm of epilepsy is a strange sensation at or near the epigastrium—a sensation "*in the chest*," or "*at the heart*." Children may call it "*pain*" or "*stomache-ache;*" by adults it is variously described as "*a sinking*," or "*nausea*," frequently accompanied by fear. For months such sensations may represent all the paroxysm; and the fact that such paroxysmal sensations are epileptic in nature is only shown by the future of the cases, when undoubted epileptic-attacks occur with the usual convulsions and characteristic tongue-biting, each paroxysm being preceded by the peculiar and familiar sensation. What was once all the paroxysm, now becomes the "warning" or inception of the severe fit. The so-called *epileptic vertigo* is another form—not merely a "confusion of the head," but a sensation of movement of external objects (see p. 142, *ante*). The sensation of turning of the body itself. At the beginning of some severe attacks the patient does actually turn to one side, "*turns right round*." *Epileptic vertigo* is therefore a slight, but actual epileptic seizure commencing in this particular way—the defect of consciousness being slight and transient; and according to the patients' and friends' account there may have been no such defect. Thus there may be no affection of consciousness, nor defect of consciousness, or, at most, a very temporary loss of consciousness. It is only the paroxysmal character of the future seizure, and the occurrence of these phenomena at different periods of the same case, in each of the three degrees, which warrants the association of all in the one name of "*epilepsy*." There are therefore many degrees of seizures, from the most transitory and trifling sensations to severe universal convulsions—an apoplexy quite undistinguishable at the first from coma or apoplexy produced by fatal cerebral hæmorrhage—followed by paralysis and coma. It is also common for the patient to have very partial convulsions before widespread or universal ones. Dr. Hughlings Jackson notes the case of a patient who had, first, attacks of spasm beginning in her right index finger, and “working up” to the elbow; months later she had severe convulsions, with loss of consciousness, attended by much spasm. A patient also may have spasm, to him apparently limited to the big toe, and at other times widespread convulsion. Another patient used to show his twitching hand to his fellow workmen “as a curiosity” before he had severe seizures which appeared to him of more moment. The slight seizures are often, at the beginning of a case of epilepsy, so slight and transient, that the patient takes no notice of them. It is only when a convulsive seizure comes on that he finds out what his slight transitory mental confusion meant. It is upon such a basis of degree that cases of epilepsy are classified—namely into *slight* and into *severe;* or, using the French names, into *le petit mal* and *le grand mal,* which being interpreted means “little fit” and “big fit.” These terms refer to quantity of manifestation, especially to range of spasm—not to gravity of the case. The severe cases are characterised by much spasm, and often universal convulsion, with tongue biting. The type of the slight cases is sudden and transient loss of consciousness, closely followed or accompanied by but little spasm, and that of small muscles, such as is signified by pallor of the face (spasm of facial arteries), slight spasm of the eyes, the hands, and the like. In some of these cases there is apparently an universal slight stiffness—a miniature universal convulsion. Possibly also the “*tremor*” described by some patients may be a miniature universal clonic convulsion. But the most striking effects are manifested in those muscles or parts the movements of which require little force for the displacements they have to effect. These slight seizures are said to be the worse for mind; and this accords with the hypothesis that the currents are developed by discharges of some parts of the highest centres, or are chiefly limited to the highest centres. Although the spasm may be limited in degree, it may affect parts in many different regions, and in this sense it may be widely scattered; while by contrast the spasm in the severe cases is very great, not simply widely scattered, but universal—all the “visible” muscles, small and large, being affected (HUGHLINGS JACKSON, *Brit. Med. Journ.*, Feb. 11, 1879).

Phenomena of the Attacks of (1.) *Simple Epilepsy.*—The attack may commence without any previous warning. These warnings are known by the term “*aura*.” They comprise all the premonitory symptoms which may prognosticate the approach of a fit. The fits in idiopathic epilepsy are so rapid in their evolution, and so similar in most cases in their developed characters, the opportunities for watching them are so rare, that mere observation of attacks or fits proved for a long time unproductive of much addition to our knowledge. It is the study of *the modes of onset* which is now necessary to be diligently prosecuted. As Dr. Gowers writes :—

> “The *aura* of an attack was formerly regarded as a change in the

nerves of the part, to which it was referred, exciting the fit. The *aura* is now known to be merely the impression made upon the consciousness by the commencing action in the brain, or by the effect of this on the periphery. The fact that an aura commencing in a limb may be arrested by a ligature round the limb above the part to which the sensation is referred, was once thought to furnish evidence that the process of the convulsion originated at the periphery. It is now well known that the ligature will arrest a fit which is due to a cerebral tumor as effectually, and more frequently, than one of any other kind. The fact that the aura is the result of the commencing change in the brain renders its study of even greater importance than if it originated at the periphery. By its means we gain information (to be obtained in no other way) as to *the function of the brain first deranged—i.e.*, as to *the functional region of the brain in which the process of the fit begins.* In some cases, at least, this may be regarded as the seat of the disease. We learn also something of the form of normal cerebral action, which disease modifies rather than subverts. The *aura* of an attack, using the term in its widest meaning, as signifying the subjective commencement of an attack, may consist in a consciousness of motion or of sensation, or in a mental state. That every structure of the brain concerned with sensation proper is connected, directly or indirectly, with a part concerned with motion, may be regarded as a proposition scarcely needing proof. A discharge such as occurs in an epileptic fit, taking place in either of those related structures, may lead to a discharge in the other. The two may act so simultaneously that the resulting motion and sensation may occur together. The patient may feel a tingling in his hand at the same moment as the fingers begin to twitch. Or the discharge in one may lead the way—be followed by the other at an interval. The twitching or the tingling may come first.

"It is therefore of importance to determine, in any given case, with which the attack commences, the motion or the sensation—*i.e.*, whether the motor or the sensory centre leads in the discharge. By so doing we are able to understand better the association of auræ and their progress. It is necessary, however, carefully to discriminate the form of the sensation, because the consciousness may be affected directly by the action of a motor centre, although no movement results (Dr. JACKSON). There are motor sensations as well as sensory sensations, and a fit may begin by a sense of motion before any actual movement occurs. The word 'sensation,' however, will here be used, when unqualified, to signify a sensation proper, such as numbness, tingling, and the like.

"In what proportion of cases is consciousness lost so early that the commencement of the fit is unfelt? Of 1,000 cases in which the presence or absence of a warning was noted, it was always absent in 495, while some aura existed, at least occasionally, in 505. Thus, roughly speaking, loss of consciousness precedes, or accompanies, the first symptoms in half the cases; in the other half the patient is aware of the commencement of the attack. We may consider what facts regarding the modes of onset these 505 cases supply. The sensation or motion with which the attacks commence may be referred to almost any part of the body, limbs, head, trunk, organs of special sense, and to many viscera. We must therefore infer that the process of the fit may commence in any part of the brain in which these various parts are represented. We may, for the present purpose, classify most of the auræ or modes of onset into seven groups: (1.) The unilateral auræ, a motion or sensation in one side of the tongue, face, trunk, or in one arm or one leg. (2.) Certain general auræ; bilateral sensations in the limbs, tremors, starts, malaise, faintness, &c. (3.) Auræ referred to certain organs, mainly to those to which the pneumogastric nerve is distributed, and to this group belong most of the visceral warnings. The most common is the well-known epigastric sensation, and others are a feeling of choking, dyspnœa, nausea, and cardiac sensations. (4.) Vertigo and other allied sensations. (5.) Certain sensations in the head, pain, &c. (6.) Psychical auræ, the consciousness of an emotion or an idea. (7.) Special-sense warnings; some of these are strictly unilateral, but it is convenient to consider them together. This classification is clinical, and as such is adopted as affording facilities for the determination of facts apart from theories.

"First, of *the unilateral peripheral auræ* present in 17 per cent. of those with warnings:—Of these, the commencement was in the arm in more than half; in the leg and face in smaller and nearly equal numbers; in the tongue in five, and in the side of the trunk in only two cases. Such unilateral commencement is common in seizures due to organic brain disease, and the organic disease causing them is usually on the surface of the brain, in the region stimulation of which causes movements in the parts first affected in the fit. Such stimulation, indeed, as Ferrier has shown, may cause convulsions beginning in the part. But from the series of cases now under consideration, all those presenting symptoms suggestive of organic brain disease were excluded. In five cases only the aura was referred first to the tongue, in two others the tongue was affected secondarily. In one the commencement was by a movement, in one by a beating, in the rest there was merely a sensation; in three tingling, and in one a sensation of something crawling on the tongue. In each case the convulsion was right-sided, and in each case in which the side of the tongue affected was noted, this was also the right side. Hence it seems probable that the sensori-motor processes for the tongue predominate on the left side of the brain, a fact also suggested by the great impairment of the movements of the tongue frequently noticed in right hemiplegia with aphasia. The association of movement of the tongue is twofold, one in its use for speech, one in its use in mastication and deglutition. We may trace this double association in the combinations of the aura. Tingling in the tongue was associated in one case with twitching in the lips, in another with a lateral movement, or sense of movement, in the jaw. The sensation of something crawling on the tongue, in one case, was followed by a feeling of sickness, then of something rising in the throat, and then by palpitation of the heart. The seizure commenced in the face in sixteen cases, and on one side as frequently as on the other, but much more frequently by a motion than by a sensation. In eight cases the mouth was drawn to one side, no doubt by the zygomatic muscles. Commencement in the lips was rare. In only two cases was there an initial contraction in the orbicularis of the eyelids, and both were associated with a sensation in the hand, and were right-sided. The cases in which the attacks commenced in the arm were forty in number. In about three-quarters of the cases the commencement was in the hand. Of those beginning by spasm, twitching, &c., the first motion was, in most instances, either in the hand as a whole, or in the arm as a whole. In only one case was there initial movement in the fingers, and this was clonic spasm in the common flexors. In fourteen cases the attack began by a sensation—'numbness,' 'tingling,' 'pins and needles,' &c.; and in these the commencement was, in most, in a definite part of the hand—the forefinger in three cases, never in the thumb, twice in all the fingers, once in the middle finger, once in the palm, twice in the back of the hand, three times in the hand as a whole, and twice at the wrist. It never began higher up the arm. In five other cases the sensation with which the fits began was less simple, and in several was certainly a motor sensation. One patient always felt 'as if his arm were drawing up,' and would beg that it might be held down, although there was no motion in it. The feeling was accompanied by severe pain, 'as if the arm would break.' In another case there was a somewhat similar pain, 'as if the arm were withering up.' In a third there was a feeling of stiffness, and in a fourth a feeling of twitching in the first three fingers, apparently without movement, and in a fifth there was a sensation in the back of the arm 'as if the nerves were being drawn.' The attacks commencing with motion, or with the sensations last described, probably motor, began in the right and left hands in an equal number of cases; but those commencing with a simple sensation began in the left hand twice as frequently as in the right. In three-quarters of the cases consciousness was lost before the seizure had extended beyond the arm. But in nine cases, after ascending the arm, the aura passed to the head, and in four to the mouth. In two cases, after passing up the arm to the shoulder, the aura passed down the side to the leg, and down to the foot. In no case, after passing to the head or mouth, did the sensation afterwards pass down the side to the leg; but in two cases, in which it passed to the mouth, it afterwards commenced again in the foot, and passed up the leg.

"The seizure commenced in the leg in fourteen cases, and in ten of these in the foot. In only five did it distinctly begin as a movement, and this began in the toes once only, in the foot as a whole twice. In one case the movement, twitching, began in the hip, and passed down the leg. In five cases the commencement was by a simple sensation, once in the great toe, once in the sole, once in the dorsum, once in the foot as a whole, and once in the leg as a whole. A motor sensation, without movement, was described in one case: the patient suddenly felt as if the leg were bent up under the other, and that he must stretch it out. Of the cases in which the commencement was in the foot, consciousness was lost before the aura had extended beyond the foot in two cases; in six the aura extended up the leg and thigh; in three of these it passed on up the side to the head. In two cases, after passing up the side, instead of going to the head it passed down the arm to the hand, and then consciousness was lost. In no case in which it passed up to the head did it afterwards deliberately pass down the arm; just as in the cases in which the aura commenced in the arm, if it ascended to the head it never afterwards passed down the side. The passage of the discharge to the region in which the head is represented, appears to be closely connected with loss of consciousness. In two cases the sensation, commencing in the foot, passed up the side, and when it had got to the level of the hand it recommenced there, and then passed up the arm. There are thus two modes in which the arm is involved secondarily to the leg, just as there are two modes in which the leg is involved secondarily to the arm—one extension by continuity, passing from the one limb to the other by the trunk, and passing down the limb secondarily involved; and the other by separate commencement in the extremity of the second limb, and th passage of the aura up both. It seems probable that in the cases i which it passes from one to the other by continuity through the trunk the discharge in the sensory centre takes the lead, and determines th course of the aura, although it may be closely, even instantly, followed by the motor discharge. The representation in the brain of the cutaneous nerves must be as continuous as the skin in which they are distributed. The passage of the aura by continuity up the arm, down the trunk, and down the leg, or *vice versâ*, is intelligible on this theory, but scarcely on the theory that the discharge in these cases begins in the motor centres. This view is supported by a case lately seen, in which the seizures began with a sensation in the foot, without motion. The sensation passed, as such, up the leg and side, and *down* the arm to the hand, and not until it reached the hand was spasm added; the hand began to twitch, and the movement passed *up* the arm, and then consciousness was lost. In several cases in which the seizure began in the mouth and face, the aura passed afterwards down the arm first, and down the leg afterwards. No aura *beginning* in the tongue, face, or leg was associated with one in the special senses. In three cases of attacks commencing in the arm there was a subsequent special-sense aura, but in each of these the seizure in the arm began by a sensation proper, not by a motion. This seems to indicate that, as might be expected, the special-sense centres are connected with the sensory-limb centres, rather than with the motor-limb centres. The only case in which a special-sense aura was in any way associated with the lower extremity was one in which the seizures began with a pain in the back of the hand, and this pain as such passed up the arm and down the leg. When it reached, still as pain, the middle of the thigh, a flash of light appeared before the eyes. The pain went on down the leg, and when it reached the foot consciousness was lost. In only two cases did the attack commence in the side of the trunk, and in one the sensation was described as 'burning.' In two cases a left-sided aura was associated with palpitation of the heart. In one patient, a girl, a singular relation existed. The fits began with a feeling of numbness and beating in the right great toe, which passed up the leg, and when it reached the groin, the patient felt a need to micturate. If she could do so the aura ceased; if she could not she suddenly felt as if turning over and over, then a peculiar psychical condition occurred; she lost consciousness, and passed into a severe epileptic fit. I have met with another case in which micturition sometimes arrested a commencing fit. I have also seen a case in which the act of micturition sometimes brought on a fit. The shiver, which in many children and some adults, normally attends the act, indicates the associated affection of higher motor centres, and these facts suggest that the passage of urine during epileptic fits is not merely the result of loss of consciousness, but is really part of the convulsive action.

"The mutual relation of the motor and special sense centres in the surface of the brain, ascertained by Hitzig and Ferrier, enable us to understand something of the progress of seizures. They do this whether we regard epilepsy as a cortical disease or not, because from Burdon-Sanderson's researches the relation is similar in the lower centres. For instance, a fit beginning in the face or tongue, if it involves the limbs, takes the arm before the leg, and we find that the arm centres intervene between those for the face and tongue, near the fissure of Sylvius, and those for the leg in the upper part of the hemisphere. Again, in a fit beginning with twitching of the angle of the mouth, rotation of the head commonly succeeds, and the centre for rotation of the head is situated in front of, and contiguous to, the centre for the action of the zygomatic muscle. But in other cases it is difficult to understand the progress of the aura from the experimental facts. For this there are probably three reasons:—First, we know little of the degree of structural association or separation of these centres, the cerebral sulci, of course, affording no information on the matter; secondly, the centres represent in many cases complex movements, and fits begin usually with simple movements; thirdly, we are entirely ignorant of the mutual relations of the sensory centres for the limbs, which Ferrier's experiments locate on the inner aspect of the temporo-sphenoidal lobe. The facts of many auræ make it probable that these, rather than the motor centres, lead in the discharge, and determine the course of the commencing fit, and I think that thus the progress and association of many auræ may be explained.

"A rare but curious condition sometimes attends the onset of a fit which begins unilaterally—what may be termed the alteration of the aura. In some right-sided convulsions from left meningeal hæmorrhage, which I once watched, the rotation of the head to the right, which accompanied the fit, was preceded by a rotation to the left, and this itself was preceded by a slight initial rotation to the right—a double alteration. I have obtained very few indications of this phenomenon in idiopathic epilepsy. In one case a left-sided fit was preceded by shaking and trembling of the limbs of the right side; in another case the attacks began with a sensation in some left cranial nerve, followed by a sensation around the right wrist.

"The great majority of fits which begin unilaterally are epileptic. Of seventy-six in which the character of the fit could be ascertained, only nine (or 12 per cent.) presented hystero-epileptic or co-ordinated symptoms. They constitute the larger proportion of cases (one-fourth) in which the auræ consist of cardiac sensations" (Dr. Gowers, l. c.).

Dr. Hughlings Jackson has also given examples of all the varieties of the aura epileptica, forming in reality the first part of the disease; and besides the defect or loss of consciousness, he notices also a positive and a negative element combined in that "dreamy condition," in which the patient, during a slight and obvious fit, becomes vague as to his present surroundings, and at the same time, or in instant sequence, he has a "dreamy" feeling of some apparently former surroundings, a "rising up" of formerly organised mental states. These constitute the phenomena of "*epileptic dreams;*" and there may be also *actions* which are epileptic without any "dreamy state." There may be *epileptic somnambulism,* in which the actions become more or less elaborate. Thus an epileptic, after a slight fit, took his boots off in church; a patient of Dr. Hughlings Jackson began to undress himself at a wharf: his fellow servants stopped him when he had got one leg of his trousers off. There are thus two classes of cases whose symptoms are to be distinguished, namely—(1.) Those in which there are "*dreamy*" states and *no actions;* (2.) those in which there are "*actions*" and either no dreams, or no remembered dreams attending the actions. In addition to these phenomena there are other symptoms which confirm the existence of the epileptic state, such as subsequent complete loss of consciousness, involuntary passage of urine—the constant occurrence of a metallic taste in the mouth preceding the attacks, or forming the first part of them. *Visceral aura* are also of much practical importance to be studied. The passing of urine and of fæces most often occurs in slight cases; such an occurrence is a valuable help in diagnosis.

The essential difference betwixt the two kinds of seizures is chiefly as to the degree of evolution of the centre, part of which has become unstable. In the severer forms of epilepsy proper the convulsions are formidable. They consist of first, tonic, and then of clonic spasm; but there is no abrupt transition from the one to the other :—

> "The clonic spasm is developed by interruptions in the tonic spasm, and these interruptions may be detected by the hand before they are visible to the eye. The clonic spasm is, indeed, only a condition of tetanus in the physiological sense of the word. Slight intermissions probably exist, but are too frequent to be recognised. As they become less frequent, they increase in degree, until they become complete. The muscular contractions are thus separated; as the fit ceases, they do not become slighter, but less frequent, and the last jerk is often as intense as any which preceded it. In partial epileptiform seizures the spasm is usually clonic throughout; and, in rare instances, in tolerably severe attacks all the spasm was clonic. There is one class of epileptic attacks in which there is only tonic spasm—general rigidity. They constitute, however, a special group, and are on the side of epilepsy towards hystero-epilepsy. In the spasm of an epileptic fit, flexion predominates slightly over extension, and more in the distal than in the proximal parts of limbs, at least in the arms. The hands are said to be usually tightly clenched. Sometimes this is the case; but a much more common position is with the wrist strongly flexed, and the fingers in the inter-osseal position—that is, with the metacarpo-phalangeal joints flexed and the others extended, the thumb being within the fingers, and all close together. It is an exaggeration of the posture seen in tetany" (GOWERS, l. c.).

During the fit the hair stands on end, the forehead is wrinkled, and the brow is knit. If the eyelid be opened, the eye is seen to be injected, sometimes convulsively agitated, at other times in a state of strabismus, and sometimes fixed: more commonly the eyelid is quivering, and half open, so as to show the white of the lower portion of the conjunctivæ. The face is red, or livid and swollen, the teeth generally clenched, and the lips covered with foam; sometimes, however, the mouth is open and the tongue thrust forward; and should the masseter muscles act spasmodically, it may be bitten through, or otherwise much injured, and the foam consequently be mixed with blood. The force with which the jaw closes is so great that teeth have been known to be broken and the jaw luxated. The limbs, also, are violently convulsed, thrown about in every direction, and with such power that it often requires three or four persons to prevent the patient seriously hurting himself. In these convulsions, also, the hands are strongly clenched, the body may be arched backwards (*opisthotonos*); and on the muscles relaxing the patient may fall to the ground with great force. While the limbs and trunk are thus powerfully agitated, the muscles of the chest are often spasmodically fixed, so as hardly to permit the acts of respiration. The functions of organic life are also implicated in this scene of tumult. The pulse is generally frequent, sometimes hard and intermittent, and at other times scarcely perceptible, although the heart's beats are strong and tumultuous. A rapid and irregular pulse is diagnostic of muscular weakness—loss of contractile power of the cardiac organ; the symptoms of which are to be ascertained by the *tactus eruditis* and the pulse tracings, rather than by any alteration in the quality of the heart's sounds. The respiration is stertorous, the stomach and bowels troubled with borborygmi, the skin bathed in sweat, while the urine, semen, or fæces are occasionally emitted, not as a result of a loss of consciousness, but as really part of the convulsive action. Blood sometimes flows from the eyes, ears, or nose, frightfully expressive of the violence of the attack.

When the paroxysm has reached its crisis the muscles relax, the convulsions subside, the respiration becomes more free, the pulse more regular, and the countenance more natural; and at length the patient falls into a heavy sleep, from which he awakes sometimes in good health, but more often shaken, exhausted, and suffering from severe headache, which lasts some hours or even days. In neither case, however, has he the slightest consciousness or remembrance of what has passed. In other instances the termination of one paroxysm is but the beginning of another, and the succession is occasionally so continued that the attack, with short intermissions, may last twenty-four or forty-eight hours, or even longer.

When *children*, from teething or other causes, are seized with epilepsy, the attack is often preceded by a spasmodic affection of the larynx, causing the whooping or crowing sound so well known to every practitioner; but it may, and often does, take place without any warning. In the former case the child perhaps is in his best health, but on awakening is seized with the characteristic whoop, often accompanied by a spasmodic flexion of the thumb against the palm, or the fingers are clenched, or the toes bent. These symptoms may recur a varied number of times, till at length, with or without this warning, the eye is seen staring, fixed, or convulsed; the face and extremities pale or livid; the hand clenched, the body rigid, and the head and trunk curved backwards. The fit is now formed; and the *fontanelle* is found distended and pulsating. These symptoms generally last only a few minutes, when a strong expiration takes place, a fit of crying succeeds, and the child, much exhausted, recovers its consciousness, and after a short interval generally falls asleep.

The duration of the paroxysm in children seldom lasts more than a few minutes. In the adult it often does not exceed that period; but in many cases it lasts half an hour to two hours, while in others the greater part of the day passes before the paroxysm terminates. It seldom happens that the paroxysm occurs but once. In the mildest case in the child it is commonly repeated three or four times in the course of the first three or four years of childhood, while in other cases it will occur three or four times in the day; and in severe cases the child is hardly out of one fit before it falls into another, till at length they gradually subside. In the adult the frequency of the fit varies extremely in different patients. In some instances there is an interval of several years; at others it returns annually, or every six months, or mensually, weekly, or even daily; while others will have twenty or thirty fits in the course of the same day.

Some special points regarding the symptoms of epilepsy have been the subject of statistical analysis by Dr. W. R. Gowers in the Gulstonian Lectures already noticed. They illustrate the following topics:—

(1.) *The relative frequency with which fits occur by day or by night* (using the terms as synonymous with the sleeping and the waking states). The investigation of this point in 840 cases yielded the following conclusions: The attacks occurred only, or almost only, at night in one-fifth; only, or almost only, by day in rather more than two-fifths. They occurred by both day and night in rather less than two-fifths. Fits occurred in the night, partially or only, in rather more than half the cases; by day, partially or only, in four-fifths of the cases. In 1 per cent. the attacks occurred only while going to sleep; in one-half per cent. only on waking up out of sleep. In 5 per cent. the attacks occurred only in the early morning. A much larger number had their first attack in the early morning; and many of these patients were in the habit of early rising, a habit which, it thus appears, should be practised with caution by those predisposed to epilepsy. Between the cases of *epilepsy* and those with *hystero-epileptic* symptoms, there was no difference in the frequency with which the attacks occurred in the day or in the night. In *epileptics*, it is somewhat more common for the first fit to occur in the day than in the night—rather more than half commenced in the former, rather less than half in the latter. In *hystero-epileptics*, it is much more common for the first fit to occur in the day than in the night. Two-thirds commenced in the day, one-third in the night. Nevertheless, in more than half these cases some fits subsequently occurred during the night—that is, during sleep. When the first fit occurred in the day, the subsequent fits occurred *only in the day* in half the cases, *only in the night* in one-seventh; by *both night and day* in one-third. When the first fit occurred during the night, the subsequent fits occurred only at night in about two-fifths of the cases; by day and night also in about two-fifths; and by day only in one-sixth. If a patient who has his first fit in the day has subsequent fits, the probability is as six to one that some of his attacks will occur in the day; the probabilities are equal that his attacks will, or will not, be confined to the day; and they are equal that he will, or will not, have some attacks in the night; while the probability that he will have attacks only in the night is but one in seven. If a patient who has his first fit in the night has subsequent attacks, the probabilities are equal that these also will occur only by night, and that they will occur both by night and by day, being in each case two in five. The probability is as five to one that they will not be confined to the day—that is, in favour of some fits occurring in the night. Frequently, fits which have recurred for a time in one condition, during sleep, waking, or both, change their time. The conclusions deducible regarding these are as follow: 1. When fits which have occurred only by night commence to occur by day, they commonly continue also during the night. 2. When fits which have occurred only during the day occur during the night, they commonly cease by day. 3. Attacks which have occurred by day and night often cease to occur in the day and continue at night, but very rarely cease by night and continue by day.

(2.) *The relation of attacks in women to menstruation* is a subject on which various opinions have been expressed. This point has been investigated in sufficient detail in 82 cases. In one-twelfth, no attacks occurred at the time of menstruation; in one-third, there was no difference at these imes; in more than half, the attacks were worse *at the monthly periods;* most frequently they were worse *before the period;* next in frequency they were worse *during the period*, and much less frequently *after the period.* n ten cases, the exacerbation was variable, either before, during, or after;

in two cases, the attacks were worse at the menstruation period soon after the patient became subject to fits, but not after the disease had existed for some years. In two cases, fits never occurred at the periods when menstruation was regular, but they did when it was irregular.

(2.) *Phenomena of Hystero-Epilepsy.*—A description of all the remarkable features of this combined affection, and most of our recent knowledge concerning it, are mainly due to the labours, expositions, and demonstrations of Professor Charcot at the Salpêtrière.

Hystero-epilepsy is a nervous disease, chiefly occurring in women, but, as Dr. Gowers' statistics show, also in men (page 229, *ante*). It is of great rarity, affecting women especially during the child-bearing period of life; sometimes, though rarely, occurring before the actual commencement of menstruation, and continuing after its cessation. It is associated with *hyperæsthesia* in one or both ovarian regions, and is usually attended by *hemianæsthesia*, and more rarely by *anæsthesia* (see page 218, *ante*) of both sides of the body. There is some, if not complete, loss of tactile sensibility, and usually absolute insensibility to pain, *analgesia* (see page 222, *ante*), of skin, and all other sensitive structures on the affected side; the muscular sense being, however, nearly always preserved. There is some degree of paresis of the affected limbs. The loss of sensitiveness may or may not affect the organs of special sense; it frequently does so affect the eye, which becomes affected with a remarkable form of colour-blindness, *achromatopsia* (see page 222, *ante*). It is characteristic of the hemianæsthesia of hystero-epilepsy that by various means—action of magnets, solenoids, metallic plates, wooden buttons, and mustard leaves—it may be momentarily diverted from the affected side, which then regains its normal sensibility, and transferred temporarily to the opposite side of the body.

The essential and pathognomonic sign of the disease is the occurrence of attacks, which present remarkable phenomena in a definite order; at first epileptiform, then affecting the mental functions of the patient, who by gesture and actual utterance reveals to the spectator various phases of emotional activity. The convulsive phenomena are distinguished not only by great intensity; but the form they assume are those of *epilepsy* mingled with the clonic convulsions of hysteria. It is this mingled form which is the only form of convulsive disease met with in these patients. In this respect the cases differ from ordinary hysterical patients. In this form of disease, hysteria is present in combination with epilepsy, so as to constitute a mixed form of illness, as the name implies. It is, in fact, a hybrid disease, in which some of the features of epilepsy are to be recognised together with those of hysteria. Epilepsy is usually the primary disease, upon which, at the period of puberty, hysteria becomes engrafted; under the influence of certain causes and of moral emotions in particular At other times epilepsy is superadded to hysteria. But it is to the form in which hysteria and epilepsy are coeval that Professor Charcot desire to restrict the term of *hystero-epilepsy.* They both develop at the sam time, and the crises do not remain distinct—the two convulsive forms alway show themselves combined, though in varying proportions, without bein at any time completely dissevered. These crises are called "seizure fits (*attaques-accès*). The hystero-epileptic seizure, besides occurring sponta eously, can usually be induced with ease by some modes of peripheral irritation.

The various phenomena of the hystero-epileptic seizure have been divided into three or four stages, as in the following description from a typical case related by Professor Gamgee, as witnessed at a demonstration given by Professor Charcot:—

> "In *the first stage* following the application of peripheral irritation there is no perceptible latent period: its features were the following:—The head was thrown violently backwards, the limbs and body became rigid, the respirations infrequent and stertorous; in a few seconds, the tonic spasms were succeeded by clonic spasms affecting the muscular system. A slight remission, lasting for a very few seconds, occurred, which was spoken of as a kind of *entr' acte*, and then commenced the second stage. This first stage may be termed the *epileptiform*. *The second stage* was characterised by extraordinary movements affecting the whole trunk. The back being somewhat arched, as in opisthotonos, the body was thrown with great violence and astounding rapidity alternately on to the occiput and heels. This stage, which, like the first, is of very brief duration, is denominated the '*phase des grands mouvements;*' during its continuance occur the first hallucinations, to be afterwards referred to. The violent movements cease almost instantaneously, and then follows—*The third stage*, or stage of emotional attitudes (*phase des attitudes passionelles*). During this stage the patient assumes successively the expression of face, the attitudes, and the gestures which portray varied emotions, intense and varied—distinguished in the order in which they occurred by the following letters:—(*a*.) No sooner had the great movements ceased, than, raising herself into a sitting posture, with clenched fists and menacing expression, the patient presented the most startling picture of one threatening; but almost instantly the picture changed to (*b*.), when the whole expression and attitude portrayed cowering, abject fear. Of no longer duration than (*a*), (*b*) was followed by stage (*c*.) The patient now assumed an expression of absolute beatitude. It is impossible to describe the look. It was the expression which some of the old masters have impressed upon their saints and martyrs. But now occurred a change no less striking than the preceding. (*d*.) The expression of saintly happiness was succeeded by one of intense joy; the patient sees one whom she loves; she beckons to him to come, to come quickly; he has come. . . . Then succeed gestures which stamp this as the *phase of lubricity* of the stage of the emotional attitudes. (*e*.) Again fear takes possession of the patient; at first it is rats which she sees, and which she appears to fear the attack of, which evoke passionate exclamations of dread and disgust; then it is obviously the fear of some human being which oppresses her, and causes her to beg for mercy. (*f*.) There is no longer fear. The patient hears the strains of music; she is pleased; she herself begins to hum the tune, but only for an instant, for (*g*.) her singing is followed by weeping, which is broken by reproaches addressed to her parents as the causes of her misery. This last phase, the stage of passionate attitudes, may be made to constitute a fourth stage, or a stage of recovery, in which hallucinations persist for a time."

These phases do not always follow in a regular manner. They get entangled occasionally, so that now one and now another predominates. This *hystero-epileptic acme* or seizure may be prolonged over a considerable time—*e.g.*, forty-five days and sixty days, and this without affecting the general health; while neither *obnubilation* of the intellect nor dementia are ever the consequences of these seizures. While there is great elevation of temperature, without necessarily a fatal result (105·8° Fahr.), in the *major acme* of epilepsy; in the *major acme* of *hystero-epilepsy*, on the contrary, the temperature rarely exceeds the normal standard.

Foci of hyperæsthesia, on the other hand, are also to be found in such patients; and these foci have this singular property that it is possible, by pressing on them, to cause the production of the hysterical fit. These are termed *points épileptogènes*, or rather *hystéro-épileptogènes*, and their existence is suggestive of the experiments of M. Brown-Séquard on the guinea-pig, where pressure on a particular part, after the operation, gave rise to a paroxysm which he terms epileptic, but which Professor Charcot regards as *hystero-epileptic*. All the patients have these points, but all do not have them in the same place. They have sometimes been discovered by chance, sometimes by the revelations of the patient, who generally preserves the secret and protects the part. One of these patients, for instance, was observed always to carefully face the physician and others, and it was found that she had a sensitive zone in the back, between the shoulders. It was sufficient to rub this area once or twice with the hand to cause the production of the fit. In another, the sensitive zone is situated below the axilla and behind the breasts, on both sides; whilst in a third, it is placed lower down, a little above the waist. The *ovarian hyperæsthesia* is evidenced, firstly, by a great tenderness in the region of one or both ovaries; it is usually on the side on which *analgesia* exists that the ovary is hyperæsthetic; secondly, by the hystero-epileptic convulsions frequently commencing by an *aura* which seems to take its origin in the hyperæsthetic ovary; thirdly, by the extraordinary fact that, at any stage, an attack of hystero-epileptic convulsions may with certainty be instantly cut short by firm pressure made with the fingers or fist in the region of the hyperæsthetic ovary. This inhibitory power of pressure upon the ovary is as surprising as is the influence of definite peripheral irritation in inducing the attack. At the instant that pressure is made in the ovarian region, two results are said always to follow—firstly, the patient's mouth opens, and the tongue is spasmodically extruded; and, secondly, the convulsions cease. The patient can then always describe the hallucinations to which she was subject at the particular instant; and it is found that these hallucinations are always constant—*i.e.*, the same attitude corresponds to the same hallucination. The first, or epileptiform, stage is associated with no hallucinations, as is discovered by cutting the attack short during this stage. No evil consequences follow such fits, as one might expect, remembering that hysterical fits often succeed each other in rapid succession many times in the forty-eight hours, without leaving any other trace than the sensation of fatigue, and even that to an extent not so great as one might expect it to be after so much violent muscular exercise. In hysteria, in fact, we have a great deal of noise for nothing, with which some malingering is often mixed.

The state of catalepsy can in some cases be immediately induced by causing the patient to fix her attention on some object, such as a bright light. Thus, if she gaze for a moment at the electric light, she passes into a cataleptic condition, when her limbs remain in any position in which you choose to place them. The patient can then be made to assume any posture, however inconvenient, and will retain it as long as you please. In this condition you can study the influence of attitudes on the expression of the physiognomy. Thus, if we bring her arms and hands together, a smile appears, raising the corners of the mouth; on the contrary, if you place them in an attitude indicative of horror or denunciation, the physiognomy becomes solemn. The extinction of the light suffices to cause the patient to pass at once from catalepsy into lethargy—somnambulistic condition—in that state of suggestion or subjection which causes her to follow and obey. This is the condition into which animal magnetisers plunge hysterical persons, and, during its persistence, they cause them to perform mystical acts, such as working in the dark, describing absent persons, distant places, and the like. From this condition the patient can be brought round by one of two methods: by pressure on the ovary, for instance, or by blowing suddenly upon the ophthalmic region. She does not immediately resume complete consciousness and freedom, but may remain in a benumbed state for a quarter of an hour or more; this, however, soon disappears, especially in the open air, and the patient recovers her normal condition. At the conclusion of Professor Charcot's demonstration, thus reported by Dr. George Sigerson (*Brit. Med. Journ.*, Nov. 30, 1878), a singular incident occurred. As the crowded audience, composed of physicians and students of different nationalities, separated, one of them seized the occasion, whilst close behind a patient whose case has been referred to above, to press with his hand upon the sensitive area in her back. The patient fell immediately upon the tiled floor of the corrider in a state of lethargy. The reckless culprit escaped in the confusion; but his act, which naturally caused great annoyance in an institution where every care is taken to guard against accidents, remains to prove the reality of the phenomenon he appears to have disbelieved (Dr. SIGERSON, l. c.)

As seen in this country, cases of hystero-epilepsy present many variations, and differ somewhat in their character from that which they present abroad. They have long been described here as merely hysterical, but their severity and epileptoid aspect have caused them now to be more specially recognised. Dr. Gowers has occasionally found ovarian tenderness, and by ovarian pressure he has often made the patient feel ill, and caused choking sensations in the throat, but has never succeeded in bringing on an attack by ovarian pressure; nor of the many gentlemen who have practised ovarian compression at the Salpêtrière in Paris, and tried it on the hystero-epileptics at Queen's Square, London, has any one been more successful. Now and then Dr. Gowers has arrested a fit by ovarian compression; but the arrest has been apparently due rather to the pain caused than to any special influence, and in such cases the attack could be cut short much more readily by faradisation of the skin. Another point of difference from the disease as it occurs abroad is that, in the natives of this country it has never been accompanied by any erotic manifestations. The excessive violence of the muscular spasm is comparable only to the convulsions of epilepsy and tetanus.

It is in cases of this description that certain phenomena have, in recent years, been again brought prominently to notice, to become once more the subject of much controversy, experimenting, and observation, after having been long since regarded as unworthy of the consideration given to them. About 1830, Dr. Burg asserted that in certain nervous diseases plates of metal placed upon the skin have the property of altering general and special sensation, and cutaneous vascular supply. The metals generally used were gold, silver, iron, copper, and zinc. The susceptibility of patients to the influence of these metals was said to vary, so that a patient sensitive to one metal will be insensitive to another. The assertions of Dr. Burg regarding the influence of these metals were confirmed by a commission of inquiry, consisting of M. Charcot, M. Luys, and M. Dumontpallier, who were appointed by the Société de Biologie to investigate and report. A great number of experiments were made on hystero-epileptic and other patients by M. Charcot at the Salpêtrière; and a report was drawn up by M. Dumontpallier embodying the results of the experiments and observations of the commission, which was presented to the Société de Biologie on 14th April, 1877. The conclusions arrived at and set forth in the Report are:—"That in hysterical and hemianæsthetic patients, if small pieces of metal (gold, copper, or iron) be fastened to a bandage and bound round the arm, leg, forehead, and a piece of the same metal be placed in the mouth and on the mastoid process of the patient, on the anæsthetic side, sensation will, after the space of about a quarter of an hour, return on that side; the formerly diminished cutaneous vascular supply will be increased, the muscular power will be augmented, the lowered temperature will rise, and the senses of hearing, taste, and smell, which had been impaired or annulled, will regain their normal vigour. What, however, is gained on one side of the body is lost on the other, in an almost exact ratio: as general and special sensation, muscular power, cutaneous circulation, return on the anæsthetic side, there is a proportionate and identical loss on the normal side. In cases of hysterical and hemianæsthetic patients, these effects remain for about one or two hours, and then gradually fade; the power lost during the experiment on the normal or hyperæsthetic side is slowly regained as it is lost on the other, till the previous condition is re-established. In patients, however, in whom the anæsthesia is of organic origin, the effects produced are more or less permanent" (ALICE M. HART, *The London Medical Record*, Nov. 15, 1877, p. 440, see also Oct. 15, 1879, p. 385).

As to the cause of the phenomena thus believed to be genuine, many theories have been advanced. The description of these cases and experiments recall the memory of similar experiments and cases which were described (some of which I saw in Dundee) forty years ago. The exhibitions and experiments were common in all the large towns of the country, and the cases of hystero-epilepsy recently described now recal the memories of animal magnetism or hypnotism, of Mesmerism, of Darlingism, and of the performances of the girls O'Key at University Colleg Hospital in London. All of these experiments and exhibitions, howeve genuine in their inception, were so mingled up with imposture, that th discussion of such subjects came at last to be proscribed. The subjects were excluded from discussion at the French Academy some years ago; and however strongly we may have been impressed with the genuineness of the facts, they still remain surrounded by so many fallacies and so much suspicious data for impartial interpretation, that an attitude of the strictest scepticism and doubt is still the proper frame of mind to approach the investigation of such cases by experiments, before we can claim to possess approved facts of scientific purity. In one of two cases of hystero-epilepsy related by Dr. Thomas Inglis, Senior Physician to the Royal Edinburgh Asylum, it was shown that other applications quite different from metals were followed by similar phenomena to those produced by metals. In this case there was hemianæsthesia. Over the left anæsthetic arm a mustard-blister leaf was tied, and removed some hours afterwards, when the whole arm was found to be hyperæsthetic, while the right arm, which was previously extremely sensitive, had become almost completely anæsthetic. This transfer of sensibility, or "phenomenon of transfer," as it is called by Westphal, only lasted a few hours. The paralysis was benefited by the use of electricity; but the relief was only temporary, and it returned after the next epileptiform attack (*Brit. Med. Journal*, Oct. 12, 1878, p. 553). The now well-known regulation series of phenomena were equally demonstrated to follow the application of wooden buttons (A. H. BENNETT, in *Brain*, Oct., 1879). The same phenomena have been observed to follow feeble electric currents, as also the influence of magnets and solenoids at a distance. The alleged phenomena and their cause are thus still the subjects of discussion. The phenomena, and the causation of them, are alike denied by not a few, who regard the changes of general and special sensation to be but more recent examples of the artful cunning and resources by which hysterical women have before been known to deceive most accomplished and experienced physicians.

The phenomena which have been thus described have led to a further development of Dr. Burg's observations in the direction of what has been called "*metallo-therapeutics*." This system is founded on two laws:—(1.) That the patients have a certain aptitude for metals; that is to say, every patient seems to be influenced in a certain way by the external application of a metal, which, however, is not in all cases the same metal. (2.) That when a metal has acted by its application to the surface of the skin, and has brought back sensibility, muscular force, and temperature, this same metal must be given internally to obtain similar and continued results.

Admitting the phenomena as regards metalloscopy and metallo-therapeutics to be such as are described, we cannot hope, in the very infancy of our knowledge as to the nature of nervous force or power, that any explanation satisfactory to all minds can at present be given. "We cannot unravel the mysteries of a very intricate piece of mechanism by watching it; discoveries have never been made by any such method. Researches are instituted to solve simple questions, and thus step by step, by discovering new laws, we apply these in explanation of complex conditions which had hitherto been unintelligible. No one by looking at the brain or nerves can tell the nature of the forces operating; and, if he conjecture that they may be electrical, or of the nature of those forces which have lately been observed in the telephone or microphone, it must be remembered that we have gained a knowledge of these forces in the laboratory. It is probable that, if we could at the present moment be told the nature of nerve-force, the very terms of the expression would be foreign to us. We are, indeed, in the position of not knowing what kind of force is produced in the brain and cord, nor the mode in which it is transmitted. Our knowledge is almost limited to the facts that an act of the will can move our muscles, and that a galvanic current can do the same. We therefore consider, that every observable change in the nervous and muscular system must be due to one or other of these causes" (WILKS). "The facts show how little we *do* know" (CLAUDE BERNARD.)

At the same time there are certain phenomena which will always claim the attention of physiologists and physicians as having been veritably presented in a sufficient number of instances to entitle them to be considered as genuine and regular manifestations of the peculiar bodily and mental condition under consideration.* We owe to Dr. W. B. Carpenter an admirable exposition of the chief laws of the phenomena of *hypnotism*, or *artificial somnambulism*, and the successful application of them in explanation of the many cases of imposture and delusion as regards the influence of metallic and electric cures, which every now and then enlist the sympathy and secure the credulity of those who are not awake to the numerous fallacies which surround the investigation of such cases. The phenomena of hysteria, hystero-epilepsy, and many so-called "mesmeric" phenomena occurring in the cases described by Charcot and others, may be regarded as accepted and described long ago. Many are genuine, but some are imitated and produced at will; and the oftener they are repeated, the more easy are they to be reproduced. It is therefore all the more necessary to keep the fallacies ever before us, and to recognise the accepted phenomena apart from any theory as to their development. "The Effects of Attention on Bodily Organs," as enunciated by Sir Henry Holland, is a subject too little studied. On this basis Dr. W. B. Carpenter would rest his *rationale* of an immense number of abnormal phenomena that at first sight appear to have but little in common. This potent source of error has been quite ignored in the clinical experiments and observations of Professor Charcot and others, on the effects of magnetics, bobbins, buttons, mustard leaves, and metals on hystero-epileptic patients. The same phenomena of hystero-epileptics have recurred in successions of epidemics, from the Dancing Mania of the Middle Ages to the outburst initiated by Mesmer in Paris before the French Revolution, which has been since that time known as "Mesmerism." The varied phenomena of this condition are all traceable to that morbid excitability of the nervous system which is the special characteristic of the hysterial temperament, though by no means limited to the female sex. (See HECKER'S "Epidemics of the Middle Ages," *Sydenham Society.*) The characteristics of these epidemics are but exaggerated forms of what is continually presenting itself in individual cases to the general medical practitioner. Nothing is more common than a "*morbid intensification of some local pain, under the influence of the* ATTENTION *given to it by the patient.* This *attention* may be kept more alive and steadfast by the kindly sympathy of those around her, under the mistaken idea (not unfrequently shared in in former times by the doctor himself) that the pain is indicative of serious local mischief. Such sympathy, habitually shown to trivial ailments, at last culminates to such a crisis, that "if you only point your finger at any part of the body she will feel a pain there." Such an ailment is but the resultant of the sympathy which *had fixed her attention* on her own morbid sensations. It s not suggested that there is any simulation in these cases. The pains are to such patients as real as if they had proceeded from any serious disorder. The physician must not pooh-pooh them as "fancies," nor stigmatise them as "shams." If he should do so, he has not mastered heir *rationale.* The second point of importance in experiment on such ases concerns the effects of *directing the attention on* bodily organs or parts f the body. The effects become much more marked where there is a *definite expectation of* some particular result. In the performances of Mesmer he "crisis" (as it was then termed, corresponding to the present hystero-pileptic convulsion) was found to come on when the patients *believed* hemselves to be in magnetic communication with the *baquet,* whether hey were so or not. The influence of "expectancy" is all-powerful in he production of hystero-epileptic phenomena. The elimination of the nfluence of "expectancy" is absolutely necessary in experiments on ystero-epileptics, and all such hysterical or epileptic patients, otherwise the eality of magnetics, bobbins, buttons, mustard leaves, electric and magnetic gencies, is liable to doubt. If such an element of fallacy is not guarded gainst, it shows little acquaintance with the history of such cases or xperience in their results. Once a system of experimentation is estab-shed, the sense of "expectancy" becomes so keen, that every indication fforded by tone or manner, as well as inferences drawn from words or cts, acquire an extraordinary significance, which makes it difficult to xclude the influence. It is not suggested that patients intend to deceive, although it must be admitted that deception and malingering are extremely common among hysterical patients. (Dr. W. B. CARPENTER in *Brit. Med. Journ.*, December 14, 1878, p. 866.) Another important fallacy is to be guarded against—namely, as to *the proof of analgesia.* By merely touching the patient and asking if she can feel, it is clear that reliance must be placed solely on the veracity of the patient; and the same applies to other phenomena, such as achromatopsia and impairment of the special senses. Also, in carrying out repeated experimentation, it is necessary to let the patient know she is being pricked, and if not she soon finds it out. "Practice makes perfect" is an ancient and true adage, and one of Professor Charcot's patients had a residence of over thirty years in hospital. Experiments on such a patient had need to be well guarded and securely planned. There is also the fallacy of not seeing through inconsequential deductions and fallacious arguments from facts the authenticity of which is not in question, but where there is no causal connection between the data or parts of the syllogism.

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* These, as originally stated by Dr. W. B. Carpenter, are—(1.) A state of complete coma or perfect insensibility, analagous to "hysteric coma," and (like it) usually distinguishable from the coma of cerebral oppression by a constant twinkling movement of the eyelids. (2.) A state of somnambulism or sleep-walking, which may present all the varieties of the natural somnambulism, from a very limited awakening of the mental powers to a state of complete double consciousness, in which the individual manifests all the ordinary powers of his mind, but remembers nothing of what has passed when restored to his waking state. This condition is further characterised by the facility with which the thoughts are directed into any channel which the experimenter may desire, by the principle of "suggestion;" and, by the want of power on the part of the somnambulist to apply the teachings of ordinary experience to the correction of the erroneous ideas which are thus made to occupy the mind. It is a peculiar characteristic of this condition that the whole attention may be so completely fixed on one object that there is an insensibility to all impressions unconnected with it, although everything which bears upon it is fully appreciated. Hence the mind may be more readily played upon by the experimenter, and may thus be exclusively fixed upon any object which he may direct. In this manner a state of insensibility to pain may be brought about, nearly as complete as that which occurs in the comatose state, by causing the mind to be strongly and exclusively directed towards another object—"Reverie." (3.) Another frequent phenomenon is a remarkable exaltation of one or more of the senses, so that the individual becomes susceptible of influences which, in his natural condition, would not be in the least perceived—*e.g.*, sense of smell, of temperature, of the muscular sense, by which various actions that ordinarily require the guidance of vision are directly independent of it,—are alike common phenomena of the mesmeric or artificial, as of the natural, somnambulism. (4.) The muscular system may also be excited to action in unusual modes and with unusual energy. Cataleptic rigidity can thus be artificially induced, and a very extraordinary degree of muscular power may be called forth, even in very feeble individuals (CARPENTER'S *Physiology*, 3rd Edition, Appendix, p. 754).

Diagnosis.—An immeasurable responsibility is associated with the diagnosis of epilepsy; and, as already seen, the very slightest cue may be all which may be given to distinguish the epileptic state. It is especially to be distinguished from *apoplexy* and *hysteria;* and the following are the main grounds of diagnosis as given by Dr. Reynolds:—

1. *The Mental State* of the epileptic is thus far characteristic, that by far the greater number exhibit a deficiency of the powers of the will in relation especially to *Thought, Emotion, Sensation,* and *Mobility.* The mind is inclined to wander in a half-abstracted state, and without energy of purpose. There is little or no power of attention or concentration of thought, and there is a slowness of apprehension, with defective memory. The emotions and their expression are undirected and uncontrolled. The patient can only give unsatisfactory and often totally unmeaning accounts of sensations experienced. Something is felt to be wrong, but the place can hardly be fixed upon; and if the head, thorax, abdomen, or limbs are referred to, the patient is rarely able to express what he has experienced. A "working in my inside" is the comprehensive phrase commonly used to express their indescribable sensations. There is also a characteristic sluggishness and clumsiness of the voluntary movements. The walk and manner of the patient become ungainly. He rolls along rather than walks, stumbling over objects in his way in an unnecessarily awkward manner. The countenance tends to be dull, expressionless, and morose. These phenomena may be so slight as almost to escape detection, and may in many cases be overcome by a determined effort of will. Sometimes on the other hand, they are extremely well marked, and graduate into utter stupidity and dementia with paralysis. Epileptics are thus no really insane; but they are eccentric, suspicious, ill-tempered, pervers fretful, and difficult to get on with. They are shy, peevish, gloomy, an exclusive. Their intellect is below the average, and memory becom more and more impaired after repetition of attacks. Their judgment often incorrect, and they are generally unhappy (ALTHAUS).

2. The *Motorial,* and 3. *Sensorial,* phenomena are such as have be described under the head of *Symptoms.* The attacks may be distinguish into two groups, namely:—(1.) Those in which the loss of consciousn

is complete, associated with violent spasmodic movements. This group comprehends "*le haut mal*" of the French authors, and the laryngismal and tracheal epilepsy of Dr. Marshall Hall. (2.) Those in which one element predominates much over the other, even to its entire exclusion:—(*a.*) Attacks in which loss of consciousness being complete, there is little or no spasmodic movement. This class includes "*le petit mal*," or "*vertigo epileptiforme*," of the French, and the syncopal attacks of Dr. Marshall Hall; (*b.*) Attacks in which there is marked general or partial spasms of the muscles, somewhat of a tonic kind, but in which there is no appreciable loss of consciousness. Such seizures constitute the "abortive" attacks of Dr. Marshall Hall.

The following are forms of epilepsies after the pathology of Dr. Hughlings Jackson:—(1.) A sudden and temporary stench in the nose, with transient unconsciousness. (2.) A sudden and temporary development of blue vision. (3.) Spasm of the right side of the face, and stoppage of speech. (4.) Tingling of the index finger and thumb, followed by spasm of the hand and forearm. (5.) A convulsion almost instantly universal with immediate loss of consciousness. (6.) Certain vertiginous attacks. All those six seizures are alike in that they each result from an occasional and excessive discharge of unstable grey matter, which is the one functional alteration of nerve tissue underlying the different phenomena (*West Riding Asylum Reports*, Vol. III., p. 334).

One individual afflicted with epilepsy frequently presents every variety of these attacks, while any one form may exist alone; but the essential features of a fully expressed epileptic attack cannot be mistaken. They consist of—(1.) The simultaneous occurrence of the following symptoms:—Complete loss of consciousness, general quasi-tonic contraction of the muscles, impeded respiration, darkened face and surface generally, with distended jugular veins, dilated pupil, distorted features, throbbing carotids; (2.) These phenomena are quickly followed by—persistent loss of consciousness, clonic violent muscular contraction, laborious respiration, with tracheal gurgling noises; slight return of colour in the face and body generally; oscillation of the pupil and eyeball; chewing movements of the jaws, and foaming at the mouth; (3.) The gradual cessation of these symptoms, and the production of another stage, marked by the following characters:—return of consciousness for a short time, with an aspect of astonishment, alarm, and suspicion; and then followed by drowsiness or profound coma; occasional semi-voluntary movements, such as change of position, laboured, slow respirations, with stertor and tracheal rattle, paleness of face, coldness of surface, with perspiration; the pupils often contracted, and the conjunctivæ injected. (4.) After sleep the patient becomes more natural in manner, and feels some headache and general soreness.

The distinctions between pure hysteria and true epilepsy are generally well marked; but, as we have seen, the affections may be combined in the hybrid condition which has been described as *hystero-epilepsy*. In hysteria there is usually much greater violence and much longer continuance of the paroxysm, more general distribution of the convulsive movements, with greater and more persistent noisiness; there is seldom loss of consciousness, so that the hysterical patient can generally be roused without much difficulty. The tongue is rarely bitten, nor are the evacuations passed in bed; nor does the patient suffer from injury. The skin is hot, and the pupils respond to light. In the *hystero-epileptic*, while the hysteric phenomena may be transient, permanent accidents or phenomena remain as marks of its presence. There are especially—anæsthesia, hemianæsthesia, hyperæsthesia, and visual disturbances, and permanent contracture of certain groups of muscles. It is also of great medico-legal importance to recognise the varied phenomena of *epileptic vertigo*, in which patients perform automatic acts, some of which may be innocent and harmless, while others may be criminal. In neither case are such patients responsible for what they do. The transition from such *epileptic vertigo* and *epileptic somnambulism* to *epileptic mania*, especially after an epileptic fit, is easy and natural. The condition is suddenly characterised by furious excitement. The face expresses rage. The breath may be ammoniacal. The patient destroys anything which comes in his way, spits in one's face, stamps with his feet, knocks the first comer down; suddenly becomes perfectly quiet, and has no recollection of what he has done. Dr. Hughlings Jackson has directed special attention to the temporary state of epileptic patients *immediately after* the paroxysm (the *first paroxysmal* condition), as deserving of more methodic study as to the after effects of epileptic discharges (*West Riding Asylum Reports*, Vol. VI., p. 266). These are especially—coma, mania, limited convulsive seizures, hemiplegia, and a degree of paralysis *beyond* hemiplegia.

In the diagnosis of epilepsy it must be always borne in mind, especially in dealing with soldiers, seamen, prisoners, mendicants, vagabonds, or others with whom powerful motives may prevail to feign diseases, that epilepsy is perhaps more frequently attempted to be copied than any other affection, and often with wonderful success, because the coarser features of the disease are so striking that few can fail, with a little study, to imitate them. The means of detection consist,—(1.) In cross-examination as to the consistency or inconsistency of the accounts of the fits, and general description of the attacks. This can only be well done when a perfect knowledge of the symptoms and grounds of diagnosis are familiar to the examiner. (2.) By observing whether or not a situation (favourable always to the malingerer) is chosen for the seizure. (3.) True epileptics seek retirement, and are frequently hurt by their falls; feigned epileptics delight to exhibit in public, and rarely sustain any bodily injury. (4.) Let the eyes be closely observed. In true epilepsy they are partly open, with the eyeballs rolling and distorted, the pupils dilated, and not contracting by the stimulus of light. The feigning epileptic prefers to shut his eyes completely; and may occasionally be seen to open them to "take a peep," so as to ascertain the effect of his exhibition. *His iris always acts on exposure to the light.* (5.) The skin of an impostor generally perspires from his exertions; that of a true epileptic in the paroxysm is generally cold. (6.) An impostor will not readily bite his tongue, or void his excrements or urine. (7.) Tests peculiar to beadles and police constables consist in dropping melted wax upon the suspected feigning person, putting some gin into the eyes, pressing the thumb nail with force under that of the supposed impostor—an experiment productive of sudden, excruciating, and harmless pain. (8.) The mere speaking of or proposing some severe remedy in the presence of the patient is sometimes enough to detect imposition. Sir Thomas Watson (from whose *Lectures* these statements have been condensed) specially recommends a very harmless and ingenious device—namely, in the hearing of the would-be patient gravely propose to pour *boiling water* on his legs as a remedy, and then proceed actually to pour *cold water* upon them. Three humorous instances of detection are thus related by him :—

> "Dr. Cheyne mentions an instance in which one table was placed upon another, and a soldier who was supposed to be shamming was laid upon the upper one while his paroxysm was on him. The fear of falling from such a height soon stopped his convulsions. Mr. Hutchison relates the case of a sailor who was suspected to be a cheat, in whom the convulsions were instantly removed by blowing some fine Scotch snuff up his nostrils through a quill. This brought on another kind of fit, namely, a fit of *sneezing*, which lasted nearly half an hour; and there was no return of the epilepsy so long as Mr. Hutchison remained in that ship. He tried the same expedient in cases of *real* epilepsy, but never could produce any similar effects, although the patients were not snuff-takers. There was a beggar in Paris who often fell into epileptic fits in the streets. One day some compassionate spectators, fearing that he might injure himself in his struggles, got a truss of straw and placed him upon it; but when he was in the height of the paroxysms, and performing remarkably well, they set fire to the straw, and he presently took to his heels" (Lect. xxxvi.)

Nearly all malingerers keep the fit up too long, and devote too much attention to certain symptoms which they believe to be pathognomonic, such as turning the thumbs in to the hand, and foaming at the mouth. The sphygmographic characters of the pulse in epilepsy have been proposed as an aid in the detection of true from feigned epilepsy, by Voisin, George Thompson, and others. The pulse becomes quicker shortly before the attack, has less force, and the elevations of the tracings are lower and more approached to each other. In true epilepsy, when the attack is well established, the pulse curves are large, the line of ascension is high, with well marked dicrotism, and these characteristics last from thirty minutes to several hours. Similar pulse traces cannot be got from persons made to gesticulate violently, from walking or rapid running, nor from any other form of severe exertions. In the cases of feigned epilepsy the pulse traces bear no resemblance to those of true epilepsy; and as the sphygmographic characters of true epilepsy remain for some time after the paroxysm, in all cases of suspected malingering the pulse traces should be taken several times during the hour after the end of the attack—real or feigned (VOISIN in *Annals d'Hygiène Publique*, Avril, 1868). Another most reliable distinction between true and feigned epilepsy is that dwelt upon by Trousseau as the *initial facial pallor*, and also *the dilated pupil*, already mentioned, even on exposure to bright light, which cannot be imitated. It is unfortunately, however, of too short duration to be of much practical use in many cases of feigned disease.

In idiopathic epilepsy, during the interval between the paroxysms, the condition of the *fundus oculi* has been investigated by many observers.

Dr. W. R. Gowers has carefully examined more than 1,000 epileptics, and has found that in most cases in the *inter-paroxysmal* state any character of the fundus was such as is presented by persons not epileptic. The only abnormality which seemed frequent, is an unusual equality in size of the retinal arteries and veins—the latter not as a rule larger than normal, and the arteries appear as if large from a lax state of their walls. *During the paroxysms*, the appearance of the fundus has been variously described. For obvious reasons the difficulties as to examination are great, and opportunities are rare. One change seems well established, namely,—that the retinal veins, during the stage of lividity, become much distended. Regarding the state of the arteries there is considerable doubt. In two cases, most carefully examined by Dr. Gowers, he could not observe any change in the calibre of the arteries. Dr. Allbutt, Dr. Hughlings Jackson, and Arlidge have observed pallor of the discs immediately after a fit, in several cases. Dr. Gowers has examined patients immediately after a fit without being able to satisfy himself that there was any difference from the appearance of their discs and vessels at other times. In cases of epilepsy in which fits were frequent, Dr. Gowers' observations have yielded negative results. In one case, however, he met with marked changes in the discs, developed under observation during a series of exceedingly severe convulsive attacks, recurring at short intervals for several days. The patient was a young man, and the convulsions were co-ordinated, so that the case was one of *hystero-epilepsy*, marked by paroxysms of struggling, aching of back, throwing about of hands and limbs, so intense that the united strength of three or four persons was required to keep the man in bed. They were accompanied by loss of consciousness, and continued unabated until ice was applied to the cervical spine, when the attacks at once ceased. The optic discs, after some days of convulsion, became reddened and veiled, so that their edges were quite invisible, and there was distinct swelling. After the cessation of the fits the discs gradually resumed their normal appearance. This patient died about three months later, after a series of true epileptiform convulsions beginning in the left hand; and, *post-mortem*, no trace of disease was visible in the brain to naked eye examination. Many cases of apparently idiopathic epilepsy may present traces of old optic neuritis or choroiditis—indicative (the former certainly, the latter probably) that the convulsions originated in organic brain disease—the choroiditis suggesting former syphilis. Traces of old optic neuritis are especially common in cases of epilepsy due to blows on the head. Chronic convulsions resembling epilepsy may also occur in the subjects of lead-poisoning, and chronic renal disease, in each of which simple optic papillitis may be present (*Medical Ophthalmoscopy*—Dr. W. R. Gowers, p. 159).

Prognosis.—An increase in the mortality from epilepsy appears from the statistics collected by Dr. Althaus during the past thirty years. The percentage of mortality to nervous diseases is 3·86, and the percentage of all diseases is ·47. The mortality is greater among men than among women—as 2·13 for men, as against 1·84 for women. It is very fatal in the first year of life. Epileptic convulsions during teething generally subside about the second or third year; children, likewise, first seized between three and four years old, are often cured, or the disease subside at puberty, except when hereditary. Epileptics attacked after puberty are generally incurable, and especially when the ailment is conjoined with insanity. Pregnant women attacked with epilepsy are in great danger. Patients with *epileptic vertigo* may suddenly become maniacal (*epileptic mania*), and in this state may commit suicide, homicide, rape, or arson, after an attack of epilepsy. For the time being they are absolutely deprived of self-control, while they may be capable of very elaborate actions. As to the positive certainty of any cure for the disease, a proper feeling of scepticism prevails. Niemeyer writes that recovery must be regarded as rare, in spite of the opposite opinion of many observers. Nocturnal epilepsy is of a specially malignant and obstinate character. Generally, the more distinctly the malady is traceable to hereditary tendency, the more plainly it depends upon structural disease of the brain, the longer it has lasted, the more violent the fits, the more frequent their occurrence, and the deeper the impression which they leave behind them, the less is the chance of recovery (NIEMEYER).

Nevertheless, well-selected remedies have a power in repressing the paroxysm, and often of indefinitely postponing it—more especially dietetic and regimenal treatment. The duration of the disease before treatment is commenced has an obvious influence over its curability. "It is seldom," writes Dr. Watson, "that any permanent ill effect can be noticed as having been left behind by any one single fit; but, unhappily, this cannot be said of their repetition." "More, probably, depends upon the *repetition* of the fits than upon their precise *nature* or *severity*." "Every successive attack strengthens the *habit*, and renders the individual more obnoxious to future seizures; every arrest or postponement of a seizure is so much gain in favour of the patient, not only by avoiding the pain and the risk of the isolated paroxysm, but still more by diminishing his future liability to the disease" (SIEVEKING, l. c., p. 212).

> "A single paroxysm often leaves the patient in a worse condition than that in which it found him; but this is not perceptible to an ordinary observer until after the alteration has been rendered apparent by repeated fits, and repeated small additions to the permanent injury. The friends of the patient remark that his memory is enfeebled in proportion to the number of the attacks; that his mental power and intelligence decline. His features even assume, by degrees, a peculiar character, and too often he sinks into hopeless fatuity, utter imbecility, or confirmed insanity. It is this tendency which renders epilepsy so sad and fearful a disease. . . . Cases do occur in which epileptic persons preserve their faculties to a good old age; but those who are early epileptic do not often attain old age; and whenever the disease comes on, if it repeat itself frequently, it is followed much more often than not by impairment of the mind, or by some apoplectic or paralytic affection," (WATSON, Lecture xxxv.)

Aretæus, in describing the symptoms of epilepsy, has not neglected to speak of the baneful influence of this disease on the intellect, of the memory being lost, of the imagination being impaired, and of the functions of the brain being, in many patients, so subverted that they fall into incurable insanity. Esquirol gives the cases of 385 epileptics under his care, in the Hospital Salpêtrière, and he states that four-fifths were more or less insane. The remaining fifth had preserved their reason, but, he adds, "a reason so broken!" Acuteness of judgment is lost, memory and power of imagination diminish, the gentler and nobler impulses recede more and more; while the excited and unbridled propensities, lasciviousness, and gluttony, too often impel the patient to commit violent or criminal actions. They often avoid society; are odd, capricious, very troublesome to those around them, and apt to burst into violent fits of anger. The personal appearance of the patient also undergoes a change in cases of long standing epilepsy. Esquirol also notices the coarseness of the features of a confirmed epileptic, his swollen eyelids, his thick lips, faltering look, and general clumsiness of body.

A popular belief prevails that if an epileptic contracts an ague the influence of the malarious poison counteracts the epileptic state, so that the ague takes the place of the epilepsy, and the fits cease. Professor W. C. Maclean relates such a case which came under his notice in Madras (*System of Medicine*, by RUSSELL REYNOLDS, under "Malaria.")

Such are the phenomena associated with the paroxysms of epilepsy—a disease not only frightful from the violence of the symptoms, but also from the serious effects it may produce on the moral character, as well as on the physical frame of the unhappy patient. While some may fall into the fire, and may be burnt to death, others falling into the water may be drowned, although the pool may be but a few inches deep (CHEYNE). They may fall from horseback, from a scaffolding, or over a precipice. Bruises and fractured limbs are thus not unfrequent. They may be choked while eating, or they may be suffocated in bed. Many epileptics have a convulsive action or *tic* of the muscles of the face, or their legs waste, and are unable to support the weight of the body. In some instances the leg has been fixed under the thigh—a contraction which has lasted more than a year; while in others the patient has become paralytic.

Cases of hystero-epilepsy usually do not tend towards recovery, though recovery occasionally occurs. It may continue for many years without any perceptible influence upon the health or mental condition of the patient, which is not the case in true epilepsy; and when death occurs it appears to result from intercurrent disease which, in relation to the primary disease, may be termed accidental.

Treatment.—The treatment divides itself into what is to be done during the paroxysm, and subsequently during the intervals.

When adults are labouring under the paroxysm, little, in general, can or ought to be done except bringing the patient into the fresh air, taking off what may be around the neck, and baring the chest, together with the more imperative duty of preventing the patient doing himself any injury. Bleeding, so often had recourse to, is rarely found beneficial; except, however, as regards females, in cases of suppressed menstruation. If, however, the paroxysm be greatly prolonged, the application of cold to the head, and opening the temporal artery, where symptoms of excessive cerebral congestion are obvious, may be of some service, as in cases recognised as plethoric. It is of great importance to shorten the paroxysm as much as possible. Dr. Sieveking recommends that a tria be made of the galvanic current during the fit. The conductors should be moistened sponges, so as to insure the passage of the current to the deeper-seated tissues. The inhalation of *ammonia* or of *chloroform*, or of *nitrite of amyl;* or a dose of *sal-volatile*, or of *ether*, has been found useful. The *nitrite of amyl* not only postpones, but altogether prevents epileptic seizures. "A vinaigrette, or small stoppered bottle, containing a sponge soaked in *nitrite of amyl*, and carried in the pocket, so as to be at hand, will be found a safeguard to many sufferers from epilepsy. Wherever there is time after the initiation of the coma, and before the development of the proper phenomena of the fit, to breathe the nitrite of amyl freely, the fit, with its terrible accompaniments and disastrous sequelæ, may in many instances be not merely postponed, but abolished" (Dr. CRICHTON BROWNE, *West Riding Asylum Reports*, Vol. III., p. 154).

The paroxysm over, the probable exciting cause of the paroxysm should be investigated, and, if possible, removed; the state of the bowels should be particularly inquired into and regulated, and leeches should be applied to the temples if the headache be severe. In women, also, the catamenia, if defective or excessive, should be remedied. These few simple rules are of the first importance, not only as removing the immediate inconveniences incident to the attack, but also as a means of prolonging the interval, and perhaps preventing its future occurrence. We must take into account, and search out for all causes which may have contributed to the malady. In assuming the charge of an epileptic, it is necessary to commence first by regulating the external relations of the patient, his habits, and his bodily health, so that every suspicious condition of his life to which the origin of the disease can in any way be ascribed may be corrected (NIEMEYER). In a few instances the patient, by the adoption of certain rules, is cured, and the prevailing principles of treatment in epilepsy mainly consist in local derivation, or counter-irritation directed to subdue cerebral congestion, and in the use of such tonic remedies as may be ndicated by a careful inquiry into the condition of the individual organs, and how their several functions are performed. The intensity of the headache suggests more or less active counter-irritation by *blisters, dry or wet cupping, ointment of tartrate of antimony, setons,* or even *the actual cautery applied under chloroform to the nape of the neck.*

The medicinal remedies that have been employed are *valerian, iron, zinc, uinine, musk, opium, asafœtida, the iodide and bromide of potassium, camphor, nd the preparations of turpentine.* The *nitrate of silver*, once esteemed a pecific in this complaint, has not only failed, but, by occasionally staining he *rete mucosum* of a dingy hue, has often permanently disfigured the atient. Of the long catalogue of remedies which has been mentioned, ach medicine is perhaps useful for a few weeks; but after that period its ood effects are, for the most part, lost, so that it would appear to act ather mentally than physically in removing the cause and altering the orbid tendency.

"Whatever remedies or course of treatment you pursue, do not appear to espond, or use any other language to the patient than that of hope. Avoid xtravagant promises, as inconsistent with that love of truth which ought characterise every professional man; but unless you have the strongest evidence against it, do not yourself, nor allow your patient, to abandon hope" (Dr. TODD, "Clinical Lectures," *Medical Times*, August 12, 1854).

The employment of purgatives is indicated for the removal of waste matter, to act as a derivative from the head, to expel foreign matters or worms from the intestines, and generally to regulate the bowels; and the laxatives most suited to epileptics are *rhubarb, compound colocynth pill, aloes, castor-oil, taraxicum, sulphur in combination with magnesia or rhubarb;* and the Pullna, Hunyadi János, Friedrischall, or Carlsbad bitter waters. Half a tumblerful or less, taken in the morning, generally produces a full pultaceous evacuation in two to three hours. The judicious use of an anthelmintic sometimes frees the patient from the disease as well as from a tapeworm or other parasite, which not unfrequently may be the *eccentric* source of the fits. Independently of its anthelmintic properties, however, both Drs. Watson and Sieveking strongly recommend the use of turpentine. It may be given in half-drachm doses every six hours.

If a scar, foreign body, tumor, or *neuroma*, be found pressing on a peripheral nerve, its removal by operation may cure the epilepsy. Dr. Echeverria has recently given a table of 145 epileptics who were trephined, with the results, which furnishes irrefragable proofs of the utility of trephining in epilepsy produced by injury to the cranium (*London Med. Record*, May 15, 1879, p. 169).

The preparations of *iron* and *zinc* are the most useful tonics; and for the general principles on which iron may be administered, the reader is referred to the pages on *Anæmia*.

Dr. Trousseau believed that *belladonna* is the least inefficacious of all the remedies for epilepsy, and from its use he not only obtained alleviation and improvement in many cases, but was able to count upon a certain number of cures (*Clinique Médicale*, t. ii., p. 95). His formula is,—

Atropiæ Sulphatis, gr. j.; *Sp. Vini Gallici*, ♏c. M. One drop of this solution is to be given every day in the morning, if the attacks happen in the daytime; or in the evening, if during the night; the dose to be increased by one drop for each succeeding month, and to be always taken at the same period of the day. It should not be pushed beyond the first toxical indications. If the dose is not well borne, it should be increased only every second or third or fourth month. When improvement is apparent, the quantity given in the last dose should be continued for some time, and then gradually lessened, and finally stopped, to be, after an interval, resumed.

A year is scarcely sufficient to test the influence of *belladonna*, and if in the succeeding year some improvement follows, the treatment is to be maintained for two, three, and four years (l. c., p. 95). Dr. Trousseau, however, in practice seems to have adopted a mixed treatment, giving *belladonna* in the morning, with the *nitrate of silver*, the *sulphate of copper*, or the *lactate of zinc*, in the evening, alternating these last every ten days. The following are his modes of administration:—

℞. *Argenti Nitratis*, gr. ij.; *Pulv. Acaciæ, Aquæ distil.*, āā q. s. Div. in pil. x. One every night. Or,

℞. *Cupri Sulphatis*, gr. xx.; *Pulv. Sacchari*, ʒj. Div. in pil. xx. Two every night. Or,

℞. *Zinci Lactatis*, ʒj.; *Pulv. Sacchari*, ʒij. Div. in pil. xx. One every night. Or, the zinc may be made into pills with the conserve of roses.

Within the past ten years the *bromide of potassium* has been largely used in epilepsy, and the testimony to its utility in the disorder is uniform and strong. Dr. C. B. Radcliffe writes:—"In the summer of 1858 I began to give this medicine almost promiscuously in cases of epilepsy and epileptiform disorder, and from that time to this (1864) I have been continually finding fresh reasons for persevering in the practice. . . . The conclusion at which I have arrived is, that *bromide of potassium* is the only remedy in epilepsy upon which most dependence can be placed." The late Dr. Bazire, in a note to his translation of Trousseau's *Clinical Lectures*, speaks thus of the experience of the physicians of the Hospital for the Epileptic and Paralysed in London, and of his own, with this remedy:—"The results obtained are such as to warrant the conclusion that it is infinitely superior to all other remedies that have been recommended against epilepsy. It is certainly far superior to *belladonna* in its power of diminishing the frequency and severity of epileptic attacks and epileptiform seizures in general; nay more, of warding off the attacks, lengthening the intervals between them, and, in some cases, of bringing on a cure" (Vol. I., p. 99). The most recent, and a very high authority, Dr. J. Russell Reynolds, in his excellent article on Epilepsy (*System of Medicine*, Vol. II., 1868), says:—"*Bromide of potassium* is the one medicine which has, so far as I know, proved of real service in the treatment of epilepsy. In large doses it has scarcely ever failed to give much relief. Given in doses ranging from ten to thirty grains, three times daily, it has had these effects:—In some cases it has completely cured the patient, and the cure has been permanent for years, and is so now. In others it has arrested the attacks, so that none have occurred for periods varying from a few months to two or three years; but on the omission of the medicine the seizures have returned. In such cases the attacks have again ceased on the readministration of the medicine. In a third series of cases it has diminished the frequency and severity of the seizures, but has not removed them altogether; the patients, while taking the bromide, have had one-half or one-third the number of fits to which they were habituated. Such patients have gone back to the old frequency of recurrence when the drug has been omitted, and have again improved when it has been readministered. In a fourth, but very much smaller number, it has been good for a time, and then has appeared to cease; and in a fifth, and yet smaller proportion, it has been apparently without any appreciable effect. . . . It is to be demonstrated that there is yet something 'specific' in the action of *bromide of potassium*" (pp. 280, 281). Dr. Voisin, of the Salpêtrière Hospital says, that it does not cure absolutely, but diminishes the disorder in a marked degree, lessening and even suppressing the nervous erethism (*Bul. de Thérapeutique*, 1867). Dr. Edward Fox reports fifty-two cases in which *bromide of potassium*, in not less than twenty-grain doses, was found "satisfactory," and ten cases in which it wholly failed, in one of which *belladonna* was more successful (*St. George's Hospital Reports*, Vol. II., 1867). Dr. Brown-Séquard, than whom few have had as large experience in this disease, began to use the *bromide of potassium* in large doses towards the end of 1860. He writes:—"I was soon led to associate the *iodide of potassium* with the *bromide:* and it became almost at once evident that, in most cases of epilepsy (whether idiopathic, symptomatic, or sympathetic, but especially in that form, which is much more common than it is admitted to be, in which that convulsive affection is allied with, or due to, a congestion of the base of the brain or its meninges), these two remedies did more good than either of them alone. By the end of 1861, after I had ascertained that the *bromide of ammonium* has a special therapeutic influence in cases of congestion of the *medulla oblongata*, and of the upper parts of the spinal cord, I began to associate that salt with the *bromide* and *iodide of potassium* in the treatment of epilepsy. . . . That mode of treatment has been submitted to an extensive trial, which leaves no doubt as regards its superiority. Although certainly it does not often cure permanently, it usually diminishes considerably, the violence and the frequency of the attacks, and is much more successful than the various modes of treatment by the best remedies against epilepsy, such as *atropine* or *belladonna*, the *sulphate of copper*, the *nitrate of silver*, *strychnine*, *valerian*, *zinc*, *digitalis*, or the means of counter-irritation by applications of *ice*, *moxas*, *croton oil*, &c., on the spine or the head" (*Lectures on the Diagnosis and Treatment of Functional Nervous Diseases*, 1868, pp. 81, 82). His usual prescription is:—

℞. *Potassii Iodidi*, ʒj.; *Potassii Bromidi*, ℥j.; *Ammonii Bromidi*, ʒijss.; *Potassæ Bicarbonatis*, ℈ii.; *Infus. Calumbæ*, *f* ℥vj. M.

A teaspoonful before each of the three meals, and three teaspoonfuls at bedtime, in a little water.

Dr. Ayer and others have preferred the sodium salt instead of the potassium salt, as it does not seem to have the same depressing effect on the cardiac muscle in large doses as the iodide of potassium has. It may be given in from 10 to 30 grains three times a day. Dr. James Ayer's mode of prescription is as follows:—

℞. *Sodii Bromidi*, *Iodidi Bromidi*, *Ammonii Bromidi*, āā ʒiii.; *Potassii iodidi*, *Ammonii iodidi*, āā ʒiss.; *Ammoniæ sesqui-carbonatis*, ʒi; *Tincturæ Calumbæ*, *f* ℥iss; *Aquæ distillatæ*, ad. ℥viii.; *misce.*

The full dose of this compound is a drachm and a half before each meal, and three drachms at bed-time. At the outset the patients are informed that regular treatment must go on for two years, after which the dose must be left to the patient's discretion, when, except for averting threatened attacks, full doses are no longer advisable. As a tonic, drachm doses of the following mixture may at the same time be taken after each meal:—

℞. *Strychniæ sulphatis*, gr. i.; *Acidi sulphurici* diluti ♏ x.; *A*
distillatæ, *f* ℥iv.; *misce.*

The strychnia may be given in smaller doses to begin with. In all cases the diet must be carefully regulated (*London Med. Record,* March 15, 1878, p. 128).

In syphilitic epilepsy, the *iodide of potassium* is increased to five or six drachms. Where the attacks begin with violent laryngismus, the *bromide of ammonium* is raised to three or four drachms, and the *bromide of potassium* diminished by two drachms. The following rules have been laid down as to the use of these remedies :—The quantity of these medicines to be taken daily should be large enough to produce an evident, though not complete, anæsthesia of the fauces and upper parts of the pharynx and larynx, this quantity varying, with the patient's idiosyncrasy, from forty-five to eighty grains of the *bromide of potassium,* and from twenty-eight to forty-five grains of the *bromide of ammonium,* when only one of these salts is given, and a lesser quantity of each, but especially of the *ammonium,* when given together (CLYMER).

These remedies very rarely produce a good effect in epilepsy without causing an acne-like eruption in the face, arms, neck, and shoulders; and there seems to be a positive relation between the intensity of the eruption and their efficacy. It is important, therefore, when there is no eruption, and also when it begins to disappear, to increase the dose, unless the dose given in the twenty-four hours is already so large that any increase brings on great sleepiness in the daytime, a decided lack of will and of mental activity, dulness of the senses, drooping of the head, considerable weakness of body, and a somewhat tottering gait. It is never safe for a patient taking these drugs to be even one day without them, so long as he has not been quite free from the attacks for at least fifteen months.

Iron and *quinine* should not be given in epilepsy, unless the ailment is complicated with *anæmia* or malarial poisoning, except, perhaps, in the form of the double salt of the *citrate of iron* and *strychnine.*

A gentle purge every five or six weeks maintains the power of the bromides (BROWN-SÉQUARD, l. c., pp. 84-86).

In night-seizures the *bromide of potassium* would seem to have less influence than in day-fits (DUCKWORTH, WILLIAMS).

The debilitating effect of the bromides ought to be lessened by the use of *strychnine, arsenic,* the *oxide of silver, ammonia, cod-liver oil, cold douches* or *shower-baths,* and a nourishing diet. There is an antagonism between *strychnine* and the *bromides,* and, when prescribed together, the dose of the *bromides* must be increased.

Dr. E. C. Seguin has also called attention, in the New York Medical Association, to the evil effects which may follow the abuse or over use of the various bromides. The most important of these results are—conditions of impaired nutrition, and of nervous atony, which continued for months or even years; general debility, with weak slow pulse and coldness of the extremities; tendency to stupor, slight difficulty in speaking, the bromic breath, and acne. Regarding the method of using the bromide salts in the treatment of epilepsy and other neuroses, he lays down the following rules :—(1.) The prolonged use of bromides is contraindicated by congenital feebleness, but they are well borne by persons of fairly full habit, and good nervous power. (2.) The bromides are indicated in cases of abnormally great irritability of the nervous system in its motor (muscular and vaso-motor) and ideational tracts; (3.) These contraindications are to be less regarded in the management of that formidable neurosis, epilepsy; (4.) Epilepsy is to be regarded as the only disease which justifies the deliberate production of a degree of *bromism* for its cure.

Dr. Seguin's method of prescribing the bromides in the treatment of a case of "idiopathic" epilepsy is the following. Two solutions are employed:—

R. *Potassii bromidi*, ℥i.; *Ammonii bromidi*, ℥ss.; *Aquæ fontanæ*, ℥vij.; *misce:* to be given by the teaspoonful; and, ℞. *Sodii bromidi*, ℥i.; *Ammonii bromidi*, ℥ss.; *Aquæ font*, ℥vij.; *misce:* to be given by the teaspoonful.

The quantity administered is to be so divided as to give by far the largest dose in the evening. The bromide is to be cautiously increased, still keeping the nocturnal dose the largest, until slight *bromism* is produced; which is usually necessary to be maintained for months, but just as little is to be given as may prevent the attack. The precise quantity required must be studied in each case. Children tolerate the bromides, as well as the iodides, in relatively large doses. It is of importance to thoroughly dilute the bromides in order to facilitate their absorption—the dose to be taken in a wineglassful or half a tumblerful of water. Under no circumstances should the bromides be discontinued; they may be *diminished*, but not *stopped* entirely; and they should be continued at least three years after the last attack. The adjunct treatment consists in the use of measures to prevent the acne to a certain extent, such as the occasional use of arsenic, sulphur-ointment, mercurial plaster, and alkaline lotions; to correct the general debility or slight paresis, by the use of strychnia, nux vomica, oxide of zinc, and quinia; to relieve the dizziness by the inhalation of nitrite of amyl, by stimulants, and by quinia; regulating the patient's diet and hygiene, and the use of cream, cod-liver oil, iron, quinine, phosphorus, strychnia, with nitro-muriatic acid. In certain cases such medicines as acted more directly upon the morbid state of the nervous centres were associated with the bromides, and the favourite among these was belladonna (*London Medical Record*, June 15, 1877, p. 221).

Picrotoxine has been found to possess anti-convulsive properties. It was originally recommended by Dr. Crichton Browne in the treatment of epilepsy, and his experiments have been recently confirmed by Planat.

Clinical familiarity with epilepsy, and a history of its therapeutics, g to show that there are no grounds for belief in any specific against thi affection, and the physician who promises a cure, or even relief, by mean of any one remedy in this disorder, runs the risk of damaging not onl himself, but his art. It cannot, however, be doubted, that, in a certai number of cases of epilepsy not apparently caused by any coarse cerebr lesion, the severity and frequency of the seizures may be greatly abridge so that the sufferers may be brought to enjoy often a great degree immunity and comfort. The proportion of such cases to the whole numb afflicted is, perhaps, few, but it is still sufficiently numerous to give ho and encouragement to further trial. Such results, however, can o be surely gained by means which will increase and develop vital po generally. It should be borne in mind that a tonic treatment does not alone consist in the administration of a tonic drug. Whilst many cases of epilepsy demand the use of tonic medicines to fulfil certain present indications, they should in no sense be looked on as curative, or even remedial, but only as adjuvants, and when they have done their work should be laid aside. Of this class of remedies none, perhaps, is more valuable than *arsenic*, given in minute doses. Where there is *anæmia*, *iron* or *manganese* is required. But a general restorative system must be adopted and persevered in.

The diet of the epileptic should not consist of much meat (nor much nitrogenised food), nor much of anything. It ought to be spare diet—simple and unstimulative, and to the absolute exclusion of alcohol. After a prolonged investigation on the influence of diet in epilepsy, carried on at the West Riding Asylum, by Dr. John Merson, he came to the conclusion that a farinaceous diet is likely to be more useful in the treatment of epilepsy than a nitrogenous one (*West Riding Lunatic Asylum Reports*, Vol. V., p. 1). The food should contain, as soon as the state of the digestive organs will permit, a certain proportion of fatty and oily constituents, and in most cases *cod-liver oil* may be given with advantage. The *hypophosphites*, as a vehicle for the introduction of *phosphorus* into the system, would seem to be of service in improving the general nutrition.

The maintenance of the activity of the cutaneous function is of the first importance. In the beginning an occasional vapour or hot-air bath may be taken; afterwards tepid salt or fresh-water baths, followed by a general grooming of the skin. Exercise in the open air, and gymnastics, both measured by the strength of the patient, are to be insisted upon, as well as such means as will promote the expansion of the lung tissue. With physical training, mental and moral training should be combined.

Such are the general principles of the rational or restorative treatment of epilepsy, which, it is believed, will give a larger measure of success than a reliance upon any one of the innumerable specifics that have had questionable and temporary repute. If a successful issue is to be had, perseverance and confidence, both on the part of the physician and the patient, are chief conditions of success, and this should be fairly stated at the outset to the sufferer and his friends.

Ice applied to the cervical spine has caused the severe paroxysms of *hystero-epilepsy* to cease (Gowers).

CATALEPSY.

Latin Eq., *Catalepsis;* French Eq., *Catalepsie;* German Eq., *Catalepsie;* Italian Eq., *Catalepsia.*

Definition.—*A sudden suppression of consciousness; but instead of falling down convulsed, as in hysteria, the patients maintain the position in which they were when the attack commenced. The limbs and trunk persist in a state of balanced muscular contractions; the same expression of countenance is preserved which may chance to be at the moment of seizure. If sitting, the patient continues to sit; if standing, he continues to stand; if occupied in any* *mechanical employment, he continues fixed in one attitude; and if he is under the influence of any passion, the countenance retains its expression.*

Pathology.—This is an extremely rare form of nervous disease, apparently intermediate between hysteria and epilepsy, but probably more allied to hysteria. It affects the two sexes nearly with equal frequency. Dr. Reynolds has observed the cataleptic state ensue in cases of chronic ramollissement of the brain, and in tubercular meningitis; and Dr. Laycock compares the condition to the state presented by the so-called "*brown study.*" The combination of fixed attitude and of unvarying expression gives to the patient the air of a statue rather than that of a living being, and he appears as if suddenly changed to stone. The most remarkable circumstance, however, in this disease is, that the attitude of the body and position of the limbs admit of being changed almost into as many new forms as a painter's lay figure, and the new position, however inconvenient, and almost volitionally impossible, is preserved till again changed, or until the paroxysm has subsided.

Besides this singular state, consciousness is suspended, and the patient neither receives any impression from external objects, nor retains any recollection of what has happened during the fit. In this respect the disease approaches in character to epilepsy. The organic functions of life, however, continue to be performed, though feebly. The pulse and respiration are regular, only the former is smaller and the latter less frequent than in health. The colour of the countenance is either pale or undergoes no change. The fit may last a few minutes or a few hours, and is said to have continued in some cases three or four days. The patient at length awakes as from sleep, and generally with a deep sigh, when all the functions of the body are suddenly restored. The attack is generally sudden, and without any previous symptoms; but it is sometimes preceded by headache, stiffness of the neck, or some obvious torpor of the mind or body. The return of the paroxysm is very uncertain, but the disease seldom subsides with the first attack. Most cases occur shortly after the development of puberty, in consequence of violent emotion, and, more particularly, shock to the affections. It has also been known to occur after severe fits of ague, and it is occasionally followed by religious mania, mysticism, somnambulism, and ecstasy (ALTHAUS). In this latter condition the patient is absorbed in some all-engrossing fancy or delusion in the widest and wildest range of variety.

In connection with these particular states, two most interesting cases are referred to here in illustration of some very remarkable phenomena allied to hysteria, epilepsy, catalepsy, mania, and double consciousness. They are described by Dr. Bristowe in the *British Medical Journal* of Feb. 1 and 8, 1879.

The following case, given by Dr. Gooch, will exemplify the cataleptic state:—

A lady who laboured habitually under melancholy, a few days after parturition was seized with catalepsy, and presented the following appearances:—She was lying in bed motionless, and apparently senseless. It was thought the pupils of her eyes were dilated, and some apprehensions were entertained of effusion on the brain; but on examining them closely it was found they readily contracted when the light fell upon them. Her eyes were open, but there was no rising of the chest, no movement of the nostril, no appearance of respiration. The only signs of life were warmth and a pulse which was 120, and weak. Her fæces and urine had been voided in bed. In attempting to rouse her from this senseless state the trunk of the body was lifted up and placed so far back as to form an obtuse angle with the lower extremities, and in this posture, with nothing to support her, she continued sitting for many minutes. One arm was now raised, and then the other, and in the posture they were placed they remained. It was a curious sight to see her sitting up staring lifelessly, her arms outstretched, yet without any visible signs of animation. She was very thin and pallid, and looked like a corpse that had been propped up and stiffened in that attitude. She was now taken out of bed and placed upright, and attempts were made to rouse her by calling loudly in her ears, but in vain; she stood up, indeed, but as inanimate as a statue. The slightest push put her off her balance, and she made no exertion to regain it, and would have fallen had she not been caught. She went into this state three times; the first lasted fourteen hours, the second twelve hours, and the third nine hours, with waking intervals of three days after the first fit, and of one day after the second; after this time the disease assumed the ordinary form of *melancholia*. It might be supposed that symptoms such as these were feigned; but there are cases beyond suspicion of this kind; and another very interesting case is related by Dr. George Buchanan in *The Glasgow Medical Journal* for July, 1857. It is an instance of this singular affection occurring in the male sex.

Prognosis.—The affection is generally innocent; but as it is apt to be associated with cerebral disease (of which it may be a symptom) which may end in *cerebritis, apoplexy*, or *disorders of the intellect*, and also with serious organic disease of the viscera (Reynolds, Wood), it behoves the physician to be guarded in predicting results, especially in our ignorance of its nature. In children it may occur as a sequela of tubercular meningitis.

Treatment.—No constant line of treatment can be stated. The individual case must be judged of upon its own merits, and prescribed for according to the principles which have guided the dictates of treatment in the allied nervous affections.

TETANUS.

Latin Eq., *Tetanus;* French Eq., *Tétanos;* German Eq., *Tetanus*—Syn., *Starrkrampf;* Italian Eq., *Tetano.*

Definition.—*Involuntary, persistent, intense, and painful contractions, cramps, or spasms of more or less extensive groups of voluntary muscles; so that the whole of the body may seem to be affected, the spasm yielding and becoming intermittent in some groups of muscles, while at the same time it exists in others, or the continuous tonic spasms are characterised by paroxysmal aggravation of extreme intensity.*

Pathology.—This is one of the most formidable diseases of the nervous system. It was well known to the ancients, and is described by Aretæus with all his usual terseness and precision.

(*a.*) *Causation.*—Sometimes idiopathic in its origin (but more often traumatic), it has been classed as of functional origin; and its treatment has been assigned to the physician or the surgeon according to the latent or apparent source of the disease. But such distinctions are purely conventional, since it matters not whether an external wound be the cause of the disease, so far as the nature of the disease is concerned. Some antecedent disturbing cause exists, usually, if not invariably, peripheral in its nature, and affecting some distant part of the nervous system. *Idiopathic*, therefore, is not to be understood as synonymous with "*causeless*" or "*spontaneous*." It merely or more often means that the cause is unseen, or has not been identified or demonstrated, and is therefore probably unknown, or a mere matter of conjecture.

Tetanus is capable of being induced by a variety of noxious or poisonous agents; but it is most frequently met with as the result of wounds, especially of contused or lacerated wounds, as gunshot; or wounds made by large projectiles, as cannon-balls, bombs; or of the amputations rendered necessary by those wounds; or from punctured wounds, or wounds in which foreign bodies remain lodged in the part. Also after burns by fire, quicklime, or boiling water, or by scalds from steam. Injuries of this kind are more dangerous upon the extremities than upon other parts of the body. Hence tetanus is always the frightful accompaniment of wars and battles; while it also occurs from various accidents and injuries, as well as idiopathically or spontaneously in civil life. The wound need not be extensive. The most insignificant injuries have been followed by tetanus; whilst most formidable lacerations, injuries, and operations have not been followed by the disease. As examples of insignificant injuries being followed by tetanus, the following may be quoted:—Extraction of a tooth, the operation of cupping, the bites of leeches, cutting the nail to the quick, the application of a blister, or of a seton or issue, the sting of a bee, the prick of fish-bone in swallowing. The proclivities of certain parts of the body, rather than others, has also been noticed as regards the development of this disease. While a wound is generally the remote cause of true tetanus, its nature and site appear to predispose to the development of the affection. Thus it is most common after injuries of the ginglymoid joints, as that of the elbow or knee, or when the bone is extensively fractured or comminuted, and especially when spiculæ of bone press upon nerve trunks. Its occurrence is also more probable if a foreign body remains in the wound, and especially if, after amputation, a nerve has been included in the ligature round the artery, thus causing laceration, contusion, or partial division of the nerve by the ligature. In other respects, the state of the wound does not appear to influence the attack, for it appears to take place equally whether it be open or cicatrised, granulating or suppurating, incised or contused; but if there be any difference, Larrey thinks the time of detaching of the eschar, especially if the stump be exposed to cold, is the most critical period. Time tends, however, to destroy the predisposition given by the wound. Excessive irritation of gunshot wounds, when they are in an inflamed condition before suppuration has commenced, may give rise to tetanus (Dr. W. C MACLEAN).

The relative proportion which the occurrence of tetanus bears t various classes of surgical lesions are given by Mr. Poland as follows, from the Records of Guy's Hospital for seven years (see *Holme's System of Surgery*, Vol. I.):—

Major and minor operations,	1364	cases.	Tetanus occurred	in	1
Wounds of all varieties,	593	,,	,,	,,	9
Injuries and contusions,	856	,,	,,	,,	1
Burns and scalds, .	458	,,	,,	,,	3
Compound fractures, .	396	,,	,,	,,	9
	3667				23

It thus appears that tetanus is most frequently met with in the more severe varieties of injury and accident, such as compound fractures, burns, and injuries to the fingers and toes. It is still a matter of doubt whether the seat of injury has any influence in establishing the disease. One of our greatest authorities on Military Surgery observed it oftener after wounds of the elbow and knee (HENNEN); others, again, from injuries of the thumb and great toe. The ends of the nerves in the fingers and toes seem to be particularly susceptible to the influences which excite tetanus; and so also are injuries to the tendons. A popular belief prevails that wounds of the ball of the thumb are prone to induce tetanus. In 128 cases recorded by Mr. Curling, 110 had received some injury to the extremities. On the other hand, it must be remembered that wounds (generally) of the extremities are also far more frequent than wounds of other parts, which may also account for their being considered more especially liable to tetanus (POLAND).

The conditions most favourable to the induction of tetanus are sudden changes of temperature, especially hot days followed by cold nights; so that the wounds are the exciting, and exposure to cold, damp, and chill the predisposing causes, as occurred after the battles of Ferozepore and Chillianwallah, where the wounded were exposed during the cold nights after exposure to a burning sun by day. It also occurred in Egypt under the same circumstances, as mentioned by Larrey; and after the battle of Ticonderoga, where the wounded were exposed the whole night after the action in open boats upon Lake George. It seems also more apt to occur in debilitated subjects when the nervous system is lowered, and is said to be more common in hot than in cold climates; and while it occurs in all states of the atmosphere, it is more apt to occur during sudden atmospheric changes, and especially a rapid change from hot to cold and damp weather. It is more frequent in spring and autumn than in summer or winter (PORTER, HAMILTON). When tetanus arises from the first of these causes, without any other being evident, the tetanus is said to be *traumatic;* when it arises from cold, damp, and chills, it is said to be *idiopathic.* The latter, however, is believed to be extremely rare in this country. Negroes seem more liable to the disease than Europeans. It follows also strains, contusions, and lacerations, and it is principally from these latter causes that it is met with in civil life. Most authors consider it to be most common in hot variable climates, as that of Egypt. After the battle of the Pyramids, says Baron Larrey, five of the wounded were attacked with tetanus. The same authority adds, that the tetanus of Egypt was much more intense than that he had observed in Germany. He states, also, that this disease is much more common in all countries at those times of the year when the temperature passes rapidly from one extreme to the other, than in seasons when the temperature is more equal. Thus, after the battle of Eylau, fought during the depth of winter, not one of the Guard, and very few of the line, were seized with this affection.

Besides wounds, strains, and contusions, some morbid poisons appear to produce tetanus. *Sulphuretted hydrogen, strychnine, brucine,* or their salts, or vegetable matter containing either or both those alkaloids, as *nux vomica, St. Ignatius's beans,* or the juice of the *upas tree,* also *thebaine* and *picrotoxine,* are well-known poisons, each of them capable of producing this affection; and the poison of *cholera,* in severe cases, has also had the same result. These poisons, administered by the stomach or inoculated into the system, induce all the symptoms of intense tetanus, and which has therefore been termed *artificial tetanus,* or *tetanus toxicus.* There is no test by which the *artificial* can be distinguished from the *real tetanus,* by which the results of poisoning and of disease can be distinguished, except that the disease never proves so quickly fatal as the rapid cases of poisoning with *strychnia* (CHRISTISON; see also *Reports of Palmer's Trial.*)

Rheumatic tetanus may come from the influence of cold alone without injury, especially after exposure to damp, as by sleeping on the damp ground, or by getting wet through when perspiring or living in a damp house or atmosphere. The symptoms are usually slight stiffness in neck and jaw, but rarely proceeding to complete lock-jaw. Nevertheless, it is sometimes so severe as to be dangerous to life. *Tetanus,* apparently idiopathic, has been said to come on after great fright and from irritation of peripheral nerves in the internal organs, such as worms, ulcer of the rectum, pleurisy, and pericarditis. Such cases are excessively rare (ALTHAUS).

All ages are liable to this disease, and even new-born children suffer from it—the "*trismus nascentium*"—ascribed to the tying of the navel-string. It occurs between the first and fifth day after separation of the cord. Although not so frequent in Europe, it is a form of tetanus of frequent occurrence in the tropics, especially amongst the coloured population. It has been very prevalent in the West Indies to newly born children, and also in the Western Islands of Scotland. It is most common however, in adult age; and if less frequent in old age, this circumstance is probably owing to persons in advanced life being little exposed to those accidents which usually produce it. Both sexes suffer from it; but men far more commonly than women. It occasionally happens to women afte parturition.

(*b.*) *Morbid Anatomy.*—Admitting thus the influence of wounds, cold, an such exciting or predisposing causes, the question comes to be considere What is the condition of the peripheral nerves at the seat of injury so to induce or promote the development of tetanus? A very careful ar exhaustive analysis of a case of tetanus has been recorded by Profess Nothnagel of Jena. The patient was a strong man, 19 years of ag in whom tetanus appeared eight days after an injury to the dista phalanx of the left thumb by an axe, which injured fibres of the rad and median nerves. A group of symptoms afterwards led to the conc sion that those nerves in the upper arm were in a state of abnorm increased irritability. But the *post-mortem* results did not establish the existence of a *neuritis.* Lepelletier, Curling, Froriep, Rokitansky have convinced themselves, by anatomical investigation, of the existence of an inflammatory process in the nerves in many cases of tetanus; and also that they have seen *neuritis* proceeding from the wound and ascending by leaps, and finally inducing medullary and meningeal disease. Nothnagel was thus led to conclude that the mode of development of tetanus *may possibly* be—(1.) Traumatic injury of nerve twigs—inflammatory processes (*neuritis*) in those extending in the cerebral direction to their respective nerve stems (*neuritis ascendens*), and finally passing to the spinal cord. The peripheral neuritis does not remain a local disease, but as an irritation becomes the source of a "discharging" lesion, not only in tetanus, but also in some cases of epilepsy, as after gunshot and other injuries of upper arm and hand (VIRCHOW, DIEFFENBACH, SCHNEE, ECHEVERRIA, HITZIG, WEIR MITCHELL, and others). This ascending inflammatory process does not always pass continuously, but sometimes by jumps, localising itself at certain points in the course of the nerve stem (*neuritis disseminata*). In traumatic tetanus Nothnagel's case showed that we may have a functional increase of the irritability in nerves encountered by the wound, without finding any evidence of neuritic or perineuritic processes. We must, therefore, recognise the influence of ascending neuritic processes in the development or excitation of many forms of neurosis (*German Clinical Lectures*, 2nd Series, p. 235; *New Sydenham Soc.*, 1877.) The nerve in the wound in traumatic tetanus has been found diseased. Mr. Erichsen says that the twig in the wound is generally diseased. In thirteen cases recorded by Mr. Poland, the nerve twig in the wound was found inflamed in five, and bulbous in one. Trousseau has also given some instances of morbid conditions of the nerve in the wound.

Rokitansky and Demme were the first to describe an excessive germination of connective tissue between the white matter of the cord, with destruction of nerve cells, the formation of oil globules and of amylaceous bodies. These alterations, however, were shown not to be constant; and in some instances to have resulted from the methods of investigation which were employed. There can be no doubt, however, that Dr. Lockhart Clarke contributed the first and most important observations made in England microscopically, which he discovered in the structural lesions on the spinal cord in six cases which he examined (*Med. Ch. Trans.*, 1865, p. 255). In one of these the medulla and upper end of the cord were free from disease; but at the part corresponding to the second cervical nerves, there were streaks and irregular areas of disintegration in different parts of the grey substance, particularly round the central canal, on the right side of which was a space of considerable size, containing a fine granular fluid, with the debris of blood-vessels and nerves. The posterior and lateral white columns, especially along the edges of fissures which transmit blood-vessels, were similarly damaged. These structural lesions continued to increase downwards through the cervical enlargement, chiefly behind and at the sides of the canal. The posterior commissure was wholly, and the anterior partially, destroyed by a fluid, transparent, and granular area. Similar lesions were discovered throughout the cervical enlargement, which varied from a state of softening to one of complete solution, diminishing at intervals, or almost disappearing, to return shortly in the same form. In the dorsal and lumbar portions of the cord similar changes were discovered. In the dorsal portion large extravasations of blood were formed along the whole lateral part of the grey substance, which now and again alternated with a few small areas of disintegration. He believes these changes to result from a morbid state of the blood-vessels, which are commonly found dilated and disintegrated. He does not regard the other structural nerve lesions as resulting from the functional excitement of the cord, because they more frequently occur in the central parts of the grey matter round the canal, where the nerve-cells are few; and because the nerve cells of the anterior grey substance, which give origin to the anterior (motor) nerve roots, remain unimpaired. Similar lesions also occur in cases of paralysis which are not accompanied by spasms or convulsions. Hence Dr. Clarke's conclusions as to the development of tetanus rather coincide with those of Nothnagel. He considers tetanus to depend upon a morbid condition of the peripheral nerves by which the excitability of the cord is increased, either reflexly by the action of the blood-vessels excited by these nerves, or by extension of the state of irritation along the nerves to the substance of the cord itself. The grey matter of the cord then becomes unduly excitable from the hyperæmic state of the blood-vessels, with the exudations and disintegrations which follow. Destruction of the texture of the cord by means of effusion has also been noticed by Dr. Dickinson, attacking the central grey matter especially, with great injection of the spinal dura mater and pia mater. The blood-vessels appeared to be an early seat of change. The tension of their coats seemed to have been lessened, and in some places blood-corpuscles were extruded; or a translucent structureless material had been exuded, which constantly lay in contact with the vessels, and often surrounded them, and held the most changeable relations to the nervous matter. It lay in the grey substance, in the white, and abundantly in the fissures, and occasionally outside the cord altogether.

Dr. Clifford Allbutt has recorded in four cases a diminution of consistence of various degrees and situations in the cord, with hæmorrhage in two of the cases visible to the naked eye. Microscopically there was great distension of the blood-vessels in both white and grey matter, with occasional exudation and disintegration of tissue around them;—isolated patches of disintegration in both white and grey matter, with numerous vacuities in the latter. Dr. E. L. Fox has also described varying changes of a similar kind (*Path. Anat. of Nervous Centres*, p. 355). Hyperæmia of the cells of the anterior horns, and granular degeneration, are recorded by Benedikt. The muscles are generally pale, sometimes torn across, with blood extravasation, or, if the case is prolonged, they are in a state of fatty degeneration. Dr. Coats of Glasgow has described vascular and perivascular changes throughout the pons, in the grey substance surrounding the aqueduct of Sylvius, and in the corpora quadrigemina, while the longitudinal vessel which lies in the deepest part of the floor of the fourth ventricle is usually surrounded by red blood-corpuscles.

From the effects of some poisons producing tetanus, it has been inferred by some that in traumatic tetanus some poison (unknown is generated in the wound; and that spasms result from certain change in the muscles due to this poisoning of the blood (Sir THOMAS WATSON and Dr. RICHARDSON).

At the Pathological Society of London, on the 29th April, 1879, Dr. Ross showed microscopical sections illustrative of the pathology of the nervous system in *hydrophobia* and *tetanus.* The lesions were,—(1.) Vascular engorgement, along with evidences of effusion of a granular material and migration of leucocytes similar to those described previously by Allbutt, Lockhart Clarke, Dickinson, Benedikt, Coats, and Gowers; (2.) the yellow degeneration of the ganglion-cells described by Allbutt; (3.) in two cases of tetanus and two of hydrophobia, a large number of colloid bodies, and what appeared to be the condition described by Dr. Batty Tuke, and by Mr. Kesteven, as *miliary sclerosis.* As, however, these cords had been partially hardened in spirit, it was doubtful how far they might be regarded as evidences of disease. Especial reserve is necessary as regards the importance to be attached to the presence of colloid bodies, as Mr. C. Creighton has especially shown (see p. 598, Vol. I.). They seem to result from the coagulation of albuminoid fluids under particular circumstances; and in regard to the cord they appear to be readily produced by *post-mortem* changes in it, possibly during a process of *hystolesis* (see p. 120, Vol. I.) The most marked evidences of disease in the cord were found on each side of the central canal; but the posterior and anterior horns were also affected. In *tetanus,* the vesicular column of Clarke was always injured, often completely destroyed; and it was also injured, but to a less extent, in *hydrophobia.* A distended vessel was generally observed to pass between the median and antero-lateral groups of ganglion-cells of the anterior horns; and the ganglion-cells on each side were destroyed, the destruction of cells being usually greater in *tetanus* than in *hydrophobia,* but never wanting. A similarly distended vessel was frequently observed passing through the postero-lateral group, the destruction of ganglion-cells here being usually more marked in *hydrophobia* than in *tetanus.* In the inferior portion of the hypoglossal and spinal accessory nuclei, the cells were found nearly destroyed in *tetanus;* those of the hypoglossal were less so in *hydrophobia;* but the damage to the spinal accessory and vagus nuclei was much greater in *hydrophobia* than in *tetanus.* The condition of the medulla in *hydrophobia* corresponded closely to that described by Dr. Gowers. In two cases of *tetanus,* vascular changes and colloid bodies were found in the cortex and adjoining white substance of the cerebellum. In *hydrophobia,* colloid bodies, vascular changes, and at times yellow degeneration of the large cells of the fourth layer of the cortex, were observed. The condition described as *miliary sclerosis* was observed in two cases of *hydrophobia.* In the cord, the patches occupied the posterior and antero-lateral columns. Beyond the cord, the patches were observed in the anterior pyramids of the medulla, and in their continuations through the pons Varolii, middle third of the crusta, and middle third of the internal capsule. Similar patches were observed in the formatio reticularis, and in the portion of the pons which corresponds to the anterior root-zones of the cord, and also in the parts of the corpus striatum adjoining the internal capsule. The state of the cord and medulla appeared, on the whole, to be very much the same in the two diseases, and the differences in the symptoms might perhaps be due to the fact that in *tetanus* the morbid changes vere directed more to the cerebellum, while in *hydrophobia* the cerebrum was more affected. Minor differences with respect to the alterations of the nerve-cells in each affection might be described, but the broad fact remains that the groups of cells which are shrunken and altered in hydrophobia, are also affected in tetanus (*Path. Soc. Trans.*, Vol. XXX., p. 219).

Nevertheless, notwithstanding those positive observations, there can be no doubt that the body has often been most minutely examined by competent observers, after the patient has died from tetanus, without any lesion having been discovered. In a few cases the membranes of the brain have been found congested; but not in a greater degree than might have been predicated from the violent and long-continued muscular action incident to the disease. It seems proved, therefore, that tetanus commences as a disease of function; and, as Majendie has shown, if the spinal cord of a living animal be divided into as many segments as there are vertebræ—that the animal, if poisoned with strychnine, still becomes tetanic, although all direct connection of the muscles with the brain is destroyed—it seems probable that the cord, as high as the fifth pair, and not the brain, must be the great seat of this affection. The morbid irritation of the motor nerves proceeds from the spinal marrow; yet real tetanic spasms are seldom seen in cases of grave organic disease of the spinal cord, and, as a rule, lesions which give rise to great excitement or exaltation of function, are not susceptible of anatomical demonstration. When the disease commences, spasms are induced by trifling but very appreciable irritants, which, acting on the peripheral nerves, induce the spasms in a reflex manner. After several repetitions by such reflex irritation, the spinal marrow seems eventually to assume a condition of permanent excitement. Some organic change is established in the nerve-centre, which no longer needs the primary irritation in the circumference or periphery to maintain it. Hence the questions require consideration, whether the lesions described are the effects of the phenomena of tetanus or the cause of them, and how have they arisen?

My own observations on the specific gravity of portions of the cord in tetanus lead me to the belief that the blood-vessels, and thus vascularity, rather than nerve-tissue lesion, have much to do with the phenomena. In numerous cases I examined in Glasgow Hospital, where I chopped up the cord into pieces, those pieces connected with the nerves going to and proceeding from the injured part, showed a marked increase in specific weight compared with other portions of the cord. (See articles on "Tetanus," by Dr. LAWRIE, in *Glasgow Medical Journal*, No. IV., Jan., 1854) This morbid state of the cord, which I determined to exist in thes four cases of tetanus, exhibited thus one character in common, pointing t the spinal cord as the seat of a lesion which was not manifest to th naked eye, but which was determined to exist with certainty by th abnormal specific gravity of the cord substance. Each of the four cas showed that the general specific gravity of the cord throughout increased in cases of tetanus, the average specific gravity of the healt cord having been determined to be 1·036. They showed also, in ea case, that a change was *suddenly* indicated about the region of the cord immediate communication with the wounded part; and in one case of idiopathic tetanus the change was uniform throughout. In the first case which I examined, where the wound was in the occipital region, the uppermost three inches of the cord were of the highest specific gravity; and the difference became *suddenly*, and not gradually, manifest at the fourth inch. In the third case, a very marked difference was apparent when the cervical region was compared with the rest of the cord; and the difference became *suddenly* marked where the roots of the cervical and first dorsal nerves left the cord to form the brachial plexus. The wound in this case was in the fingers. In the fourth case the difference was *suddenly* manifested in the lowermost part of the cord, corresponding to the region where the nerves were in communication with the lower limb, which was the seat of the injury.

But there is no one lesion which has been here described, which may not also be found in cases of ordinary paralysis, progressive muscular atrophy, locomotor ataxy, and the like, where there has been no phenomena analogous to tetanic spasm.

Symptoms.—The interval between the reception of the injury and the first tetanic symptoms varies from a few hours to the fourth and the fourteenth day, and rarely exceeds twenty-two days. Sir James Macgrigor gave, as the result of his great experience, that no person is attacked with tetanus after the twenty-second day from receiving the wound, a period which Sir Gilbert Blane extends to the fourth week. Some time in the second week is the most common period. The more rapidly the disease comes on, the more likely to be fatal will be the result. There is usually a certain order in which the different sets of muscles are affected. The invasion of tetanus can scarcely be mistaken; but the general premonitory symptoms may be so undefined as to appear unimportant, and so escape notice; not so, however, the uneasiness about the neck, the sense of constriction about the throat, and the stiffness of the jaws. The muscles of the neck, jaws, and throat, are almost always the first to indicate the disease, as well as a peculiar expression of the face given by the contraction of the muscles about the eyebrows.

There are five varieties of tetanus—*trismus* or *lockjaw*, *tetanos*, *emprosthotonos*, *opisthotonos* and *pleurosthotonos;* and when either of these forms of the disease terminates within eight days, the case is said to be *acute;* but if prolonged beyond that time it is considered *chronic.* (1.) *Trismus* is that state in which the disease is limited to the muscles of the lower jaw and throat, thus preventing the opening of the mouth, and hence the term "*lockjaw.*" (2.) *Tetanos* or *Orthotonos* is marked by the flexor and extensor muscles of the body generally being equally and strongly contracted, keeping the whole frame in such a state of tension that if you attempt to raise the leg, you raise the whole body, it being as inflexible as in *rigor mortis* (Baron Larrey). The whole body appears straight. (3.) *Emprosthotonos* is when the flexor muscles bend the body forwards, so that the spine is arched in the back. (4.) *Opisthotonos* when there is such spasm in the long extensors of the spine that the head is drawn backwards and the spine is curved in an arch with the convexity forwards. (5.) *Pleurosthotonos* is when they bend it laterally, or on one side only.

The frequency of occurrence of these different forms of tetanus is not accurately determined; but *trismus* is the most common, and though it may exist *per se*, it is generally the first and concomitant symptom of all the other forms. After *trismus*, *opisthotonos* is the most common, both in this country and throughout Europe; but Baron Larrey says that *emprosthotonos* was most common in Egypt. Of *pleurosthotonos* only a very small number of cases are to be found recorded.

The attack of either form of tetanus may be sudden; but more frequently it is preceded by an uneasy sensation and tension of the præcordia, followed by stiffness of the neck, shoulders, and lower jaw. There is a difficulty and uneasiness in bending or turning the head—a sensation like that commonly known as "*stiff-neck*." The mouth opens with difficulty, and at last the jaws close completely, sometimes gradually, but always eventually with great firmness, so that the disease has popularly got the name of "*lockjaw*." At length the patient feels a sudden and painful traction of the ensiform cartilage; and this latter symptom is considered a pathognomonic sign of the disease. The muscles concerned in swallowing become affected. At this point the disease may stop, and the phenomena be limited to *trismus;* but commonly the patient takes to his bed, and the disease assumes one of its more severe forms, as of *opisthotonos*, *emprosthotonos*, *pleurosthotonos*, or of *tetanus*. The pain in the sternum is one of the most distressing parts of the disease. It is an acute pain at the lower part of the sternum, piercing through to the interscapular region of the back, and is believed to depend upon cramp of the diaphragm.

In *opisthotonos*, in addition to the *trismus*, the muscles of the face are generally spasmodically affected. The forehead is wrinkled, the brow is knit, the corners of the mouth are drawn, giving to the patient a most wretched grin—the *risus sardonicus*. The muscles of expression, by their tonic contractions, thus impress upon the features of the patient a fixed and painful look, which becomes intensified during each recurring spasm. The angles of the mouth are drawn outwards, and the lips are drawn apart, so as to expose the firmly clenched teeth. The eyes also are almost motionless and sunk in their sockets; and, during the attack, the tongue is projected against the teeth, so that, except for the trismus, it might be caught by a convulsive snapping of the jaws, and severely injured. The characteristic of this form of the disease, however, is, that the flexors of the back are thrown into such powerful action that the spine becomes arched, and sometimes to such a degree that the body rests on the occiput and heels, as on the extreme points of the segment of a circle. A mos graphic painting of such a patient, by Sir Charles Bell, exists in th Museum of the College of Surgeons of Edinburgh. The flexors of th back are not the only muscles affected, for the shoulders are thrus forward by a strong action of the pectoral muscles, while the extremiti are elongated and tightly braced by strong contractions, both of the flexors and extensors. Indeed, the whole of these different sets of muscl are thrown into action at the same moment, as if by the discharge of powerful galvanic battery. The contracted muscles seem as hard as wo and are the seat of such frightful pain as can only be appreciated by a one who has suffered from severe cramp in the muscles of the leg. T shock is transitory; and having passed off, an interval succeeds which varies from a few minutes to half an hour, an hour, two hours, or longer, according to the severity of the disease. Exacerbations of the spasms commonly occur every ten or fifteen minutes, usually beginning by an increase of pain at the sternum, and lasting for two or three minutes. These paroxysms become more frequent—a shorter interval between—as the disease advances. But during this interval, or interparoxysmal period, the patient lies stiff and motionless on his back, with his arms close to his sides, and his legs stretched out and touching each other, fearing lest the slightest motion should produce a recurrence of the attack. His nights, or such few as he lives through, are sleepless, or only marked by a few minutes' broken slumber. The pain in the epigastrium, the impossibility of finding a comfortable position, and the interference with respiration, cause a feeling of suffocation and anxiety. Such is an attack of opisthotonos. The other forms of the disease differ only by the different sets of muscles affected.

It is difficult to give an idea of the distressing violence of the spasms. They may be imagined when it is stated that Desportes gives a case in which both thighs were broken. But notwithstanding this strongly powerful action of the muscles, the patient's mind is seldom affected. The intellect is not involved in tetanus; and in this respect it is more allied to chorea than to any other nervous disease. The intercostal muscles partaking in the general spasm, the respiration is carried on principally by the diaphragm; and when the attacks are frequent the breathing is short and laborious. The respiratory muscles are *specially* involved in tetanus; and the *diaphragm* is no doubt affected in company with other and voluntary muscles of inspiration and expiration. Respiration is not generally accelerated, but is difficult and forcible, and may become arrested during a convulsive attack.

The excitement of the spinal cord, as a centre and source of reflex motor power, overrides the controlling influence of the brain. The inhibitory authority of volition is thus suspended, leaving the voluntary muscular system generally under the undisputed sway and dominion of excited reflex action (Le Gros Clark).

Neither the nature of the wound nor its condition bear any relation to the intensity of the symptoms; and the tetanic spasms may not be developed till the causative lesion is healed, and may then prove speedily fatal. There is much thirst and hunger, which cannot be *appeased*, on account of the difficulty of swallowing. The tongue is dry and coated with a thick fur. The saliva, scanty and viscid, clogs the mouth. The heart's action may not at first be altered, and the pulse may be regular; but it soon becomes rapid and feeble (90 to 120), and its feebleness nd rapidity increase as the disease advances, especially during the aroxysms, so that towards the end it cannot be counted. During a fit, the heart may stand still during diastole (probably from irritation of the root of the pneumogastric nerve in the medulla), so that the patient may thus die in a fit.

The skin is, after a short time, covered with a profuse sweat, as during reat exertion. The bowels are generally constipated, and the *sphincter ni* so contracted, that it is difficult to introduce a glyster-pipe. In cases in which tetanus supervenes on a suppurating wound, the sore dries up and is painful, while the muscles of the part are highly irritable.

There is no increase in the excretion of urinary solids (Senator). The body temperature remains normal so long as there are no convulsive seizures, and no complication from surgical fever connected with the original injury. It soon, however, becomes subject to irregular diurnal variations, so that in the fits there is at first a fall of heat, and afterwards a rise above the normal, which continues after the attack is over. It does not usually exceed 100° or 101° Fahr., but may rise from time to time, even in cases which ultimately do well, to 102° or 103° Fahr. Each fresh attack sends the mercury higher up, and it is in cases of tetanus, also, that the maximum of body temperature has been observed—112·55° Fahr. by Wunderlich, and 111·9° Fahr. by Lehmann; and fifty-seven minutes *after* death, in the first of these cases, Wunderlich observed the temperature rise to 113·675° Fahr. These rises are accomplished in very brief spaces of time. The Professor of Veterinary Surgery at Dorpat (Hofrath) also informs Wunderlich that he has observed temperatures above 107·6° Fahr. in fatal cases of tetanus in horses. The temperature has also been seen to go below the normal in the course of some cases. The irritation of the contractions increase the irritability of the muscles, so that the muscles become more irritable after each paroxysm; and when an irritation follows sooner than usual on a previous one, the tetanus is greater than if the repose had been longer. The irritability seems to increase more rapidly when the intervals of repose are the shortest, and great exhaustion attends such periods of increasing irritability (C. S. Minot in *Journal of Anatomy and Physiology*, Vol. XII., p. 312). Hence also the increasing temperature in the muscles, after the paroxysm is over, compared with the rectum, may be accounted for.

In mild cases the paroxysm returns only three or four times in the twenty-four hours; while in severe cases it returns not only every hour, or every quarter of an hour, but every motion of the body or attempt to open the mouth is followed by an attack. In the last stage the situation of the patient is most pitiable, the spasm returning every few minutes, till he is at last cut off during one of unusual violence Death usually results from a mixture of causes, but chiefly from *apnœa*, or "want of breath," due to spasm of the larynx, or to th fixed condition of the respiratory muscles lasting longer than th necessities of respiration can withstand; or to *asthenia* from flagging the heart's action; or by a combination of those conditions. It n unfrequently occurs in one of the spasmodic attacks from spasm the respiratory muscles. "Unrelaxing cramp, exacerbations recurri with increasing frequency and violence, the broken rest, the imperfe nourishment of the frame, the exhausting effects on the nerve-centr resulting from the continuous demands on them by the unremitti spasms—all aid in bringing to a speedy close the acute form of t disease; and the unhappy victim sinks into a state of prostration, b physical and mental, which brings with it some relief to his sufferi before he dies" (Le Gros Clark).

Diagnosis.—Independent of *tetanus*, the jaw is sometimes locked enlargement of the cervical glands, by difficult dentition of the wisd teeth, and also in some forms of hysteria. The tumor, however, in the one case, and the hysteric symptoms in the other, are circumstances which readily enable us to distinguish them from *trismus.* The formidable phenomena of tetanus are seen in no other disorder, except cholera; but the other differences between the two diseases are so extremely marked, that it is impossible not to distinguish them.

Prognosis.—The prognosis in this disease is always most grave and gloomy. Almost all traumatic, and a great majority of idiopathic, cases die. Taking all forms together the mortality is at the rate of 88 per cent. (POLAND). In 128 fatal cases reported by Friedrich 83 died within the first four days, and the remaining 45 at variable intervals after the fourth day. In the war between Austria and France in 1859, Demme records 80 deaths out of 86 cases. The most rapidly fatal cases in the experience of Mr. Poland are fatal in four or five hours; but death has been delayed as long as to the thirty-ninth day. More than half the total number of fatal cases die during the first five days. Generally it may be stated, that notwithstanding the relative unfrequency of the tetanic spasms, and of the existence of but slight increase of reflex irritability, if the tetanus has appeared only a few days after an injury, if from the outset the muscles of deglutition are involved in a high degree, if there be a high pulse rate (120), even if temperature be but slightly elevated (101·8° Fahr.), and if finally, but most important, if the muscles of respiration, especially the *pectorals* and *scaleni,* are in a state of tonic contraction, so that the patient can only breathe with his diaphragm, and the mobility of the upper part of the thorax is in abeyance, each of these must be regarded as rendering the prognosis more or less serious. The prognosis also is influenced by the age of the patient. It is most fatal in proportion to cases during the first decade of life. Professor Yandell gives the following results of an analysis of 415 cases:—(1.) It usually supervened between four and nine days after the injury. (2.) The largest number of recoveries are found in cases in which the disease occurred after a lapse of nine days from the injury. (3.) When tetanus continues fourteen days, recovery is the rule and death the exception, apparently independently of the treatment. (4.) Tetanus arising during the puerperal state is the most fatal form of the disease. In the Peninsular War, although hundreds of cases were treated by many different methods, yet few, very few survived. In civil life the chances are something more favourable; and if the accident be of little moment, and the patient very young, he sometimes recovers. The prognosis in traumatic tetanus may be looked upon as always unfavourable. The chances of effecting good "when the malady is fairly established are very slender in any case, however mild" (GROSS'S *System of Surgery,* Vol. I., p. 644). In Holmes's *Surgery* this sentence occurs: "In acute traumatic cases the prognosis is most unfavourable, and there is scarcely a well-authenticated instance of recovery on record" (Vol. I., p. 327). Dr. Parry writes, if the pulse be not more than a 100 or 110, up to the fourth or fifth day, the patient almost always recovers. If the temperature exceeds 103° Fahr. the symptom is of serious import. The danger decreases in general also in proportion to the duration of the disease: few patients die after the fourteenth day. The danger in the early stages is to be estimated by the frequency and violence of the paroxysms.

As is the case with certain fevers, so is it with tetanus: it seems to have a definite course to run; and the duration of the disease is various. In some instances death occurs in twenty-four hours; more commonly on the second, third, and fourth day, and is seldom protracted beyond the eighth. Some few persons survive till the seventeenth or twentieth day, and in such cases the disease generally terminates in recovery.

Even when recovery has fairly set in, convalescence is always gradual and protracted over weeks and even months. Certain forms of paralysis are also apt to remain—*e.g.*, paralysis of the portio dura, perhaps the results of *neuritis ascendens*, and paralysis of some of the muscles of the extremities, which may be due to the persistence of the lesions in the cord which have been mentioned (ALTHAUS).

Treatment.—So long as the phenomena are peripheral and reflex, the removal of the offending part may arrest the disease; but when it has become central, it is probably always fatal. There are thus two forms of tetanus which demand recognition for treatment as early as possible. Baron Larrey affirms that this disease, if left to nature, is quickly fatal. Sir James Macgrigor says that all the most powerful remedies were fully tried in the Peninsular War, and that little or no dependence could be placed on any of them. There is great tolerance of all kinds of sedatives and narcotics.

Much good, however, is gained by attempting to restore the secretions to a healthy state; also by supporting the patient with food and drink, and by endeavouring to tranquillise the high irritation under which he is labouring. The medicines to be employed are moderate doses of purgative medicines, with *tinct. opii* ♏ xxv., or its equivalent, 10 grains of *Dover's powder*, given every three or four hours; and these conjoined with moderate quantities of wine, sago, mixed with strong beef-tea or soup, or other nutritious diet. *Musk*, also, in ten grain doses, has been given with some advantage. Some authors lay much stress on a local treatment in traumatic tetanus. Baron Larrey, as the result of his great experience, says,—"When it is caused by the wound, we should not hesitate to operate on the first symptom of tetanus, and thus, as far as possible, remove the causes of irritation." If tetanus follows amputation, he recommends the stump to be sprinkled with powdered cantharides; and in cases where a nerve has been included in the ligature, that the ligature be removed either by section or by actual cautery. In the British army, however, all these proceedings have been adopted, and with very little success. Amputation has been frequently performed without any mitigation of the symptoms. The wound has also been excised, submitted to actual cautery, been blistered, and dressed with every kind of ointment; but the disease has run its course, either uninfluenced, or its fatal termination has been accelerated. Hennen has even seen the wound heal and the patient die on the same day. Nothing, in fact, is so unsatisfactory as the results yet obtained from either the general or local treatment of this fatal affection. Larrey has often attempted, from the difficulty of swallowing fluids that sometimes attends *tetanus*, to pass an elastic tube; but in all cases he says he met with a contracted state of the œsophagus impossible to overcome, while the attempt was constantly followed by the immediate occurrence of the severest spasmodic attacks.

"If, however," as Mr. Poland observes, "we can help our patient on one day after another, we gain much. Constant watching and constant attention are required by night as well as by day, and unflinching perseverance on the part of the sufferer to aid all efforts made in his behalf, besides the avoidance of all causes of excitement, and more especially drafts of cold air or winds, taking care to preserve a uniform temperature as much as possible." No hope of recovery may be indulged in unless the spasmodic seizures continue to diminish in length and frequence, distinct relaxation of the contracted muscles occurring during those intervals of diminished spasm; and unless the patient becomes able to take food, recovery in the fortunate cases takes place with extreme slowness. It is not till after the lapse of weeks that the muscles entirely lose their spasmodic tension. Russian vapour baths are well spoken of by Hasse and Niemeyer.

Too free use, alike of *narcotics* and of *anæsthetics*, must be guarded against. They are valuable aids in maintaining the existence of the patient from paroxysm to paroxysm. *Aconite* has been used in large and repeated doses by De Morgan, Smith, Sedgwick, Woakes, and Wunderlich. It ought to be used hypodermically, and there appears to be a great tolerance of the remedy. *Cannabis sativa* or *Indian hemp*, is a remedy of real value, if it can be got genuine and fresh. Three grains of the *extract*, or thirty drops of the *tincture*, may be given every half hour or hour till narcotism is attained. Cold should at the same time be applied to the spine (MULLER, O'SHAUGHNESSY, CHUCKERBUTTY).

The more or less obstinate constipation of the bowels which characterises this disease, and which is accompanied by a fœtid state of the dejections, ought to be overcome by (1.) turpentine enemata, and (2.) calomel and jalap purgatives. This is most important.

A case of traumatic tetanus under the care of my friend, Dr. John Liston Paul, Surgeon-Major of H. M. Indian service, is reported in the *Madras Monthly Journal of Medical Science* for July, 1872, which recovered under the persistent use of *belladonna tincture*, carefully observing the effects of the drug upon the system. The bowels were first freely acted upon by *calomel*, gr. v., and *compound jalap powder*, gr. xx.; and immediately on the action of the bowels commencing, *ten minims* of the *tincture of belladonna* were given in half an ounce of water. In an hour *five* minims more were given; and every hour afterwards an increase of *three minims* was added to the previous dose, till twenty minims were given at one dose. The administration of *belladonna* was commenced at 10.30 A.M., and by 4.30 P.M. a dose of twenty minims was thus gradually reached. This dose was continued in half an ounce of water every hour till 10 P.M., after which it was given every second hour till the following day, when no relief to the rigidity or paroxysms being obtained, this dose was increased to *twenty-five* minims every hour. This treatment was persevered in, with the aid also of *chloral hydrate* occasionally, as well as ice applied to head and spine, and movement of the bowels by active purgatives of *jalap* and *croton-oil*. At three intervals the belladonna had to be suspended, on account of its poisonous physiological action being obvious in the delirium, thirst, and other symptoms. The patient was under active treatment with *belladonna* for fifteen days—the amount of tincture taken in that time being ten ounces four drachms and thirty minims.

Yandell states that *chloroform* up to this time has yielded the largest percentage of cures in acute tetanus (*Brain*, Oct. 1878). He is of opinion that the true test of a remedy for tetanus is its influence on the history of the disease in the following respects :—(*a.*) Does it cure cases in which the disease occurred prior to the ninth day after the injury? (*b.*) Does it fail in cases whose duration exceeds fourteen days? Tried by these tests he asserts that no agent has yet established its claims as a true remedy for tetanus. But among the numerous medicines used in the treatment of tetanus *chloral* is one of the latest, and appears to possess many advantages. Bauer, in his article on tetanus in Von Ziemssen's *Cyclopœdia*, states that other remedies are practically and properly being more and more supplanted by *chloral-hydrate.* This is superior to every other means, even the inhalation of *chloroform*; and the control of the spasm will be attained most satisfactorily by the combination of *chloral* with *bromide of potassium.* Billroth also records success in cases of tetanus from the use of *chloral.*

Dr. J. R. Beck has collected (*St. Louis Medical and Surgical Journal*, June, 1872) thirty-six cases of traumatic tetanus treated essentially by *chloral* in which a recovery took place in twenty-one instances. Dr. H. C. Wood has tabulated (*Treatise on Therapeutics*, pp. 292, 293) eighteen additional cases, resulting in nine recoveries and nine deaths. In the *Practitioner* for November, 1872, Dr. Macnamara, of Calcutta, gives his experience in tetanus among the natives, and says that he treats them by giving forty grains of *chloral* at bedtime, and by providing proper diet of a nourishing kind. In severe cases, an additional thirty-grain dose is given at midday. Out of twenty successive cases seventeen recovered. Though these cases were probably idiopathic in many instances, the testimony is nevertheless valuable. Within the past few years the medical journals have been constantly reporting cases of recovery from tetanus under the use of *chloral*, but isolated cases have their value diminished, because the fatal cases are not so likely to appear in print. Nevertheless the fact remains that many cases of traumatic tetanus have recovered while such treatment has been employed. The drug has been introduced into the system by various channels; M. Oré has treated cases of tetanus, though unsuccessfully, by intravenous injections of *hydrate of chloral* (*Practitioner*, August, 1877); while success has followed its administration hypodermically in acute traumatic tetanus (*American Journ. of Med. Sciences*, April, 1877, p. 534), and it is stated that a case recovered where *chloral*, in conjunction with *bromide of potassium*, was given by enema (*Hospital Gazette*, New York, April, 1877, p. 15, from London *Lancet*). Dr. Agelastos, of Bucharest, believes that he prevents the occurrence of locked-jaw in traumatic cases by the timely employ of *chloral* (*New York Med. Journ.*, April, 1877, p. 436). Verneuil says that *chloral* allays the muscular contractions, and especially those of the respiratory apparatus, which in the last stages of the disease cause asphyxia; and that it changes the acute into the chronic form of the disease. He gives instances of cure where the patients took 100 grains and 245 daily without any bad symptoms. The first of these tetanic patients recovered after thirty days, having been given ℥vj. of *chloral* during that space of time (*New York Med. Journ.*, 1876 p. 97). While in charge, as Resident Surgeon, of Dr. R. J. Levis's war in the Pennsylvania Hospital, Dr. Roberts had the care of four cases of traumatic tetanus, all of which were treated with *hydrate of chloral;* of this number three recovered, and one died out of nineteen cases.

The evidence appears, indeed, to be very strong in support of the efficacy of *chloral hydrate* in the treatment of *tetanus;* and if one thing stands more prominently out in the history of medicine during recent years, it is the influence for good which is being exercised and becoming more easily controlled by the judicious use of those very powerful remedies. Dr. Van Someren of Madras also records successful cases from the use of *chloral hydrate*, and recommends a more heroic administration of the drug (*Madras Monthly Journal*, May 1872).

Woorara or *Curare* is reported to have cured thirteen out of thirty-three cases (Demme, Busch). Appropriately given it effectually controls the spasms, and thereby spares exhaustion from them. The hypodermic injection of a solution of *curare* deserves extensive trial, commencing with such small doses as ⅛ to ¼ of a grain, gradually increasing from ½ to 1½ grains. A solution of *one* to *two* grains in 100 drops of water should be made fresh, and 10 drops of this solution injected as a dose. The action lasts about four or five hours, and upon this fact its repetition must be regulated (Demme).

The *Calabar bean* seems next or equal in importance as a remedy in tetanus. In 1874 an analysis of published cases showed that out of 63 cases of traumatic tetanus treated by this medicine, 33 recovered. Since then this unusual proportion of cures of a disease which was before nearly always fatal, has been considerably increased. When the treatment has failed, the failure has been mainly attributable to an inefficient use of the remedy. The minimum dose of the *extract of physostigma* which should first be administered, is one-third of a grain. It should be repeated every quarter of an hour, until its specific operation is developed, and the spasms are completely overcome. This effect should be steadily maintained, and then reproduced as often as the spasms recur. It is usually necessary to increase the dose in order to maintain its original impression (Stilbe and Maisch, *The National Dispensatory*, p. 1080). In the *New York Medical Record* of July 6, 1878, Dr. Burke reports two cases which occurred under his care in New York. One was seen on the tenth day after injury to the foot from treading on a rusty nail. Drop doses of *fluid extract of physostigma*, of a strength of 60 grains to the drachm, were first ordered, together with castor-oil, as the breath was foul. On the following day the dose of *Calabar bean* was increased to a *drop every half hour* combined with ʒi. of whisky to counteract the depressing effects of the *Calabar bean.* Finally, the dose was gradually raised till ♏xiii. of the fluid extract of *Calabar bean* were administered every half hour; whilst as much as two pints of whisky were given daily. The other ingesta were always fluid, consisting of milk, eggs, and beef-tea. As good effects were produced the quantity of the extract was gradually reduced, and finally, in about six weeks, entirely discontinued, good care and nourishment completing the cure. In a second case the dose was gradually raised till ♏xv. of the fluid extract were given every hour, whisky, at the same time, being always administered. The full physiological action of the drug was also obtainable by injecting hypodermically

♏xxii. to ♏xxxiii. of the alcoholic extract of the *Calabar bean* every hour, ʒiv. of whisky were also given by hypodermic injection, and about an ounce by the mouth. The patient steadily improved under a treatment in which the alcoholic extract was injected hypodermically in doses ranging from ♏x. to ♏xxv. every hour. He was ultimately discharged cured. One grain of the *extract* of *Calabar bean* may be given by the stomach; or one-third of a grain, mixed with x. or xv. minims of water, and neutralised by *Carbonate of soda*, by subcutaneous injection, repeated in two hours, and increasing the dose gradually.

Sir Joseph Fayrer records two cases of traumatic tetanus treated and cured by *opium smoking* and internal administration of *chloroform and hemp*. Both patients were Hindoos. The average quantity of *opium-goolic* smoked daily by one patient was 84 grains. The *chloroform* and *hemp* were given as in the following prescription:—

℞ *Chloroform*; ♏x.; *Extract of Indian hemp*, gs i.; *Mucilage*, ʒi; *Camphor Mixt.* ℥i. *Misce.* To be repeated every six hours.

Surgeon-General Gordon, during the siege of Paris, reports that M. Demarquay saved several patients' lives who had manifested early symptoms in the form of *trismus* by means of *very hot air-baths* followed up by the hypodermic injection of *morphine*. In the fully developed cases these remedies were not successful. The *inhalation* of the *nitrite of amyl* has been recommended by Dr. W. S. Forbes of Philadelphia, in quantities of three to five drops.

A case of tetanus having occurred, the bowels should first be relieved by a large enema with turpentine, after which perfect quietness in a darkened room, free from noise or currents of air, must be insisted upon, as the best means of preventing the fits of spasm. The use of the ice-bag along the spine, as recommended by Todd, has afforded relief by allaying spinal irritation; but its action is very depressing. By far the largest mass of testimony has accumulated in favour of supporting the strength of the patient by the frequent administration of nutritious foods, tonics, stimulants, and opiates, the latter of which must be given in the most liberal and persevering manner. These remedies (nutrient and medicinal) must be administered both by the mouth, by the rectum, and by the hypodermic method (HAMILTON). Stimulants and abundant nourishment appear to give more prospects of success in the treatment of tetanus than any other means; and with the use of *chloroform, chloral*, and *Calabar bean* to lessen the spasms, perfect rest, quietness, and freedom from currents of air, the sufferer may be able to withstand the exhaustion, and live through the acute period of the disease.

Systematic feeding of patients with liquid and strengthening food, at short intervals, has been employed with very good results in the hospital of the University of Pennsylvania. The food must be given at intervals of every two or three hours; and should consist mainly of milk, with a small quantity of alcohol. Solid food is to be avoided. Medicinally, the patient must be brought well under the influence of the *Bromide of Potassium*, by an initial dose of two drachms to half an ounce, to be followed by half a drachm to a drachm every three or four hours. Thirty grains of *chloral* are to be given with some *opium* at bedtime. *Chloral* also is to be used when necessary in the daytime. *Nitrite of amyl* and *chloroform* should not be used continuously, but should only be resorted to from time to time to stop violent spasms. If bromism should be produced, *chloral* and *opium* should be relied on; or, *cannabis indica* may be substituted for the bromides. Where there is much cerebral congestion a blister may be put on the nape of the neck (*Philadelphia Medical Times*, Oct. 13, 1877, and *London Medical Record*, Dec. 15, 1877, p. 500).

Nerve-stretching has been recently advocated as a remedy in tetanus. It is an operation first introduced and recommended by Nussbaum for the cure of neuralgia and other painful and spasmodic nervous affections. Within the last few years it has been tried in this country in the treatment of tetanus, by Milner, Verneuil, Callender, Eben Watson, and by Mr. Henry Morris in the Middlesex Hospital, London, and by Surgeon-Major J. J. L. Ratton, in Madras Medical College, with varying success. M. Verneuil's case occurred after a crush of the hand. Traction was made on the median nerve at the elbow, and on the ulnar near the wrist. The man completely recovered, but no particulars are given as to the duration of the case, or as to its being acute or chronic. Two cases reported by Dr. Eben Watson both died. The patient operated upon and reported by Mr. Henry Morris also died. The case differed from the others in its very great severity and acuteness; and he did not think that the progress of the case had been in the least retarded or mitigated by the nerve-stretching. On the contrary, the course of the symptoms seemed to become more severe and more rapid; and he could not help expressing his conviction that nerve-stretching in tetanus would not prove a favourable mode of treatment. It seemed to be unscientific in principle; and so far as experience went, most unsatisfactory in practice. The idea was to destroy the conductivity of the nerve-trunk, so as to cut the spinal cord off from its communication with the peripheral nerves at the seat of injury. In doing this a very severe shock or irritation was likely to be excited by the nerve-stretching itself; so that the condition of the cord, be it what it may, whether a material change, a state of excitement from irritation, or a "bad habit" induced by a "depraved current," seemed more likely to be aggravated than mitigated by a new and great disturbance of the same nerve channel at a spot much nearer to the spinal centre. But the operation is still *sub judice* (*Brit. Med. Journ.*, June 21, 1879).

On the other hand Surgeon-Major Ratton writes hopefully of the operation, aided by the administration of *chloral*. The case he relates is that of a strong Hindoo woman thirty years of age, in whom symptoms of lockjaw appeared after having taken a cold bath on the third and last day of a menstrual period; and in whom there were also deep syphilitic ulcers over the left ankle, some with sinuses and tense cicatricial bands. He regarded the case as one of traumatic tetanus, the symptoms of which rapidly developed into an explosion of acute tetanic violence, which he believes would have killed the patient but for the nerve-stretching; and which, at the same time, would not have saved the patient but for the *chloral*. The sciatic nerve was stretched with "the force required to lift a three-pound weight" for the space of two minutes, and afterwards jerked once or twice with about double that force before being replaced. Not a single spasm recurred for 18 hours after the operation, when slight spasms reappeared; and if at this time *chloral* had not been pressed, the spasms would have gathered strength. This is the fifth time Surgeon-Major Ratton has stretched the sciatic nerve for traumatic tetanus, and each time the operation has suppressed spasm for many hours; and if during these valuable hours of rest, *chloral* is properly plied, he believes the chances are that spasm will not return. He has given *chloral* very freely, so as to maintain narcotism for ten days. During the three critical days of the case of the Hindoo woman, on and after the operation day, she had 300 grains of *chloral* daily. In such cases there is a great tolerance of *chloral.* In this particular case he purposely refrained from operating until the symptoms of tetanus were alarming, for he desired to test the value of nerve-stretching in acute cases only, for subacute cases often get well of themselves, while really acute cases never do. He doubts if any treatment will avail to cure acute tetanus unless age (say about thirty years, or prime of life) and stamina of constitution are with the physician. In this case the duration of the disease was twenty-three days. It is only the strong who can hope to battle through the exhausting effects of tonic and clonic tetanic spasms prolonged for days, generally with insufficient nourishment, often for days at a time able to take nourishment by rectal alimentation only; yet considering the well-merited reputation of tetanus as a fatal disease, surely it is a solid gain that even a few cases may, under certain circumstances, be saved by the resources of modern medicine (*Brain*, Part VII., Oct., 1879, p. 483).

HYDROPHOBIA.

LATIN EQ., *Hydrophobia;* FRENCH EQ., *Hydrophobie;* GERMAN EQ., *Hundswuth*—Syn., *Wasserschen;* ITALIAN EQ., *Idrofobia.*

Definition.—*A disease peculiar to animals of the canine or feline race, the specific poison of which being implanted by them in man, or in other animals, produces a similar malady. The saliva or secretion issuing from the mouth of the diseased or rabid animal contains and so conveys the poison which inoculates rabies, either through a wound or through a thin epidermis with abrasion. The period of incubation of the poison after inoculation varies from four to sixteen weeks, or even longer, before the malady becomes developed. The disease is characterised by severe constriction about the throat, spasmodic action of the diaphragm, and distress at the epigastrium: all of which are aggravated or brought about by attempts to take fluid, or by the least breath or current of air on the surface of the body, which produces, in the first instance, an effect resembling that produced upon stepping into a cold bath. Saliva, tenacious and clammy, issues from the mouth. Paroxysms of phrensy, or of uncontrollable impulsive violence (rabidity), supervene. The duration of the disease varies from* three *to* six *or* seven *days, the greater number of cases terminating in death on the second and fourth days from the accession of symptoms. Death is generally sudden, and unexpected at the moment.*

Pathology.—(*a.*) *Causation.*—Hydrophobia originates in animals of the canine and feline races, as the dog, the fox, the wolf, the jackal, and the cat, as a specific inoculable disease, but how it originated or first became developed in them, is altogether undetermined. There is no evidence that it ever arises in these animals spontaneously. It is probably at all times to a certain extent endemic, and occasionally epidemic among these animals. It has been supposed that it is excited in them by the great heat of the dog-days, or by the *œstus veneris;* but Troilliet has shown that canine madness occurs with nearly equal frequency in winter, spring, summer, and autumn. The poison is not peculiar to any country. Rabies is found equally in Europe, Asia, and America. Neither is it limited to climate. It prevails in the frozen regions of Canada, as well as in the East and West Indies. The difficulties attending any explanation of the origin of this poison are at present not to be surmounted; but hydrophobia once originated in the animals that have been mentioned, they have the power of reproducing it through inoculation only, or by the introduction in any way of the saliva of such diseased animals into the tissues of those which are healthy. This inoculation is generally by the bite of some of the canine or feline species, not only in each other, but probably in all warm-blooded animals, certainly in all domesticated animals, as the horse, the elephant, the sheep, the ox, even in the common fowl, and in man.

It will be necessary to the proper understanding of hydrophobia, to give a short outline of the disease as it occurs in the dog, so constantly associated with us in domestic life, and the principal source of the disease in the human subject. The symptoms of this formidable affection, as witnessed in the dog, are:—Some singular departure from his ordinary habits, such as picking straws or small bits of paper off the floor, and swallowing them, licking the *noses* of other dogs, or other cold surfaces, such as stones or iron. He is observed, besides, to be more lonely, shy, and irritable; his voice is so changed that his bark would not be recognised by those who have known his voice before; and he is less eager for his food, or refuses it altogether. His ears and his tail droop; his look is suspicious and haggard; and sometimes, from the very commencement, there is a redness and watering of the eyes. In a short time saliva begins to flow from his mouth, he "slavers," his fauces may be seen to be inflamed, and he is feverish. The animal, though highly irritable and easily provoked, still obeys the voice of his master, and it is remarkable "that the dread of fluids, and even the sight of them—so striking a feature in man—is often wanting in dogs and other animals, for dogs lap water during the disease" (YOUATT). In many dogs the symptoms never rise higher than these; but in others there is a repugnance to control, and a readiness to be aroused to extreme rage on the appearance of a stick, whip, or other instrument of punishment, or on any attempt at intimidation, which strikingly characterises the disease. In this state, however, he seldom fights a determined battle, but bites and runs away; still even this mitigated irascibility usually ends in indiscriminate aggression, till at length he dies, apparently of convulsions or asthenia, or from mere nervous excitement and functional derangement. Majendie has inspected the hydrophobic dog, and found no characteristic morbid change. In all cases, however, in which the poison has had time to set up its specific actions, the principal lesions of structure are found to be in those parts supplied partially or entirely by the eighth pair of nerves. The tongue is swollen; the fauces, the salivary glands, and the mucous membrane at the back of the larynx behind the epiglottis, are more or less inflamed. The bronchial membrane is also occasionally inflamed, and so is the mucous membrane of the stomach, which generally contains a strange mixture of straw, hair, paper, hay, horse-dung, and earth, showing the peculiar morbid propensity of the animal; or, being void of those substances, it contains a fluid resembling the deepest-coloured chocolate. Such are the symptoms and phenomena of hydrophobia in the dog, the chief source of this fatal malady in the human race.

The saliva or other viscid secretions which are furnished by the mucous glands of the mouth and fauces, and by the salivary glands of the dog or other animal labouring under rabies, is either the virus, or contains (as any *menstruum* would) the poisonous principle—the *animal poison* which by inoculation produces *hydrophobia* in the human body. The disease is so named, not because there is any dread of water, but because in man the most prominent symptom is an inability to swallow, or to attempt to swallow, any fluid, on account of the extreme spasms which the attempt produces. The experiments of Hartwig have proved that the poison is of a definite character, that it may impregnate various substances, and that it retains its activity for a long period. Inoculations of the saliva of rabid animals succeeded in 23 per cent. of the animals operated on by him; and, according to Faber, out of *one hundred and forty-five* persons bitten by rabid animals in Würtemberg, only *twenty-eight* had hydrophobia.

Two points in the pathology of *rabies* are peculiar—namely, *first*, that a long period of latency exists in the human subject; and, *second*, that inoculation is not always followed by the development of the specific disease. With regard to the first of these peculiarities, it is to be noticed that, although in some cases pain has been felt in the cicatrix a considerable time after the accident, and in a few a slight fever or a rapid pulse has been remarked to continue from the receipt of the injury to the outbreak of the malady, still the symptoms of the disease in man seldom show themselves sooner than the *fortieth* day after inoculation, and rarely after two years. A matured zymosis seems essential to the production of the full influence of the poison; and it may be that a double zymosis takes place, as in the case of the venereal virus, first in the part and afterwards in the system (Miller), the result of which is either to multiply the poison or to increase its virulence. Undoubted instances are, however, on record in which the characteristic symptoms appeared as early as the *twelfth* day (Sidey), and on the *eighth* day (Troilliet), who even quotes instances of their occurrence as early as the day following the injury. The duration of the period of incubation, however, is sometimes of extreme duration. It has been satisfactorily proved to extend over five and a half, six, or even nine months (Bergeron, Brandreth); and there is on record a large body of evidence in favour of the opinion that the incubation stage of hydrophobia may be prolonged not only over a series of months, but also over one year at least. An analysis of sixty authentic observations by Romberg has shown that the shortest interval between the introduction of the poison and the appearance of the disease is fifteen days the longes from seven to nine months, and that the average period is from four to seven weeks. The inquiries of Drs. Hamilton and Hunter give to the majority of cases a period of incubation from *thirty* to *fifty-nine* days. In the *Transactions of the Vienna Medical Association*, a case is recorded of a period of incubation extending over two years (HASSINGER); but this is discredited both by the elder and younger Gurlt of Berlin, whose experience in veterinary pathology has been very extensive. In all such extremely long periods of incubation the question may be asked, whether the disease has been actually inoculated at a period so far back, or has there occurred a re-inoculation at some intervening period? It is known that the dog in the early stage of the disease has a disposition to lick the hands, face, or other exposed parts of persons, and especially of those with whom it is familiar; and there are cases on record where the disease has been implanted in this way. Mr. Lawrence mentions the following:—"A lady had a French poodle, of which she was very fond, and which she was in the habit of allowing to lick her face. She had a small pimple on her chin, of which she had rubbed off the top; and, allowing the dog to indulge in his usual caresses, he licked this pimple, of which the surface was exposed. Thus she acquired hydrophobia, of which she died." While this example teaches us that hydrophobia may be implanted without a bite being inflicted, in this almost unconscious manner, it ought to deter us from permitting such indulgences to a dog. The greatest anxiety and misery have frequently been experienced for many months by those who have been thus imprudent, owing to the circumstances of rabies having subsequently appeared in the animal so indulged (COPLAND). There are instances, however, recorded of very long periods of incubation after a bite, where subsequent inoculation, independent of a bite, could not have taken place. For example, there is a case published by Mr. Hale Thomson, in Vol. I. of the *Lancet*. The subject of it, a lad aged eighteen, had been twenty-five months in close confinement in prison, and during that time had never been exposed to the bite of any animal. He had been bitten severely by a dog seven years before in the right hip, and the scar still remained. During the whole period he was under observation he was sullen, gloomy, and reserved, and was never known to look the person in the face to whom he spoke. Death occurred after a three days' illness, during which "the most decided symptoms of hydrophobia were manifested." On the 15th of May, 1854, a case was admitted into Guy's Hospital, under the care of Dr. Hughes, in which hydrophobia appeared to have been developed five years after the bite (*Med. Times*, 1854). Such observations render it extremely probable that the period of incubation of the specific poison of hydrophobia is not yet defined; and the circumstances which, in man especially, seem to shorten the duration of this period, or prolong it, are in a great measure quite unknown. The shortest period, according to Niemeyer, is about *eight* to *ten days;* the longest *twelve* or *thirteen months;* in the majority of instances, about *forty days* after the reception of the bite.

The reasons for the great inequality in the periods of incubation are obscure; but there are some circumstances which seem to show that during the long interval of apparent latency the quantity or the virulence of the implanted poison seems to increase, locally at least, if not also more extensively in the system. (1.) In some instances there are evidences of a slow and silent change going on in the constitution, indicated by sallow looks, sunken eyes, a pulse somewhat accelerated, more easily excited and weaker, combined with symptoms of general debility (COPLAND). (2.) The observation of Dr. Marochetti, a Russian physician, who visited the Ukraine in 1828, and who maintained that in that country characteristic pustules were observed to form beneath the tongue, near the orifices of the submaxillary glands, between the third and ninth day after the infliction of the bite. A similar observation was made by Dr. Xanthos of Siphnos, and the presence of these vesicles had long been known traditionally in Russia and in Greece. These observations were confirmed by M. Magistel, at Boulay, in France, in 1822, who noticed that the pustules formed from the *sixth* till the *thirty-second* day. He observed two forms of pustules, a crystalline and an opaque, the latter of which, when opened, left a small ulcerated cavity. They were situated on the sides of the *frœnum linguœ*, and on the lateral parts of the inferior surface of the tongue. They have not been found, however, in all cases. (3.) Changes which take place in the cicatrix before the development of characteristic symptoms, indicate that the implanted poison there undergoes some process, the nature of which is as yet not known.

Neither age nor sex is exempted from hydrophobia; but no instance is known of any person being affected with hydrophobia unless antecedently bitten by an animal suffering from *rabies*. It is a question of much moment whether the saliva of a patient labouring under hydrophobia will or will not communicate the disease. It may be stated as an undeniable fact that, during the many years hydrophobia has been studied, no instance is known of its having been communicated from one human being to another, although many instances have occurred of the attendants having been bitten, or otherwise accidentally inoculated with the saliva of the hydrophobic patient. The only circumstance which makes this statement at all questionable is, that Majendie and Breschet inoculated two dogs with saliva taken from a diseased patient, shortly before his death from rabies, and that one dog shortly afterwards died of hydrophobia. Persons have also been seized with rabies in consequence of having wiped their lips with napkins or cloths, or other articles, which were soiled with the saliva (ENAUX, CHAUSSIER, and AURELIANUS). The dog's tooth generally implants the poison, or at least some abrasion appears to be necessary, either of the cutaneous or mucous surfaces. The ancients were aware of this, for Celsus observes that the integrity of the lining membrane of the mouth is necessary to the operation of the Psylli, whose office it was to suck out the poison after the bite of a rabid dog; and Dioscorides expressly orders them first to wash their mouths with astringent wine, and afterwards to lubricate the cavity with oil. With regard to dogs, Meynill observes that "such of them as have been thought to become affected merely by the contagion of the same kennel will generally be found, upon minute examination, to exhibit the marks of bites, though concealed by the hair." When a scratch or other abrasion exists, a rabid dog merely licking the part is sufficient to implant the poison of rabies.

The number of deaths from hydrophobia during each of the ten years from 1866 to 1875 inclusive, in each county in England and Wales, is given in a return presented to Parliament in 1877, from which it appears that the total number of victims to this disease during the ten years in question was 334, of which 110 are recorded against Lancashire and 62 against the West Riding of Yorkshire. These districts stand highest on the list, many of the counties having been free from the disease. From these data and the frequency of reported cases the disease seems to be increasing generally in this country. Thirty-five deaths are credited to the parts of Middlesex, Surrey, and Kent, which are included in London. The 334 deaths are apportioned to each of the ten years as follows:—thirty-six to 1866; ten to 1867; seven to 1868; eighteen to 1869; thirty-two to 1870; fifty-six to 1871; thirty-nine to 1872; twenty-eight to 1873; sixty-one to 1874; and forty-seven to 1875. One death only from hydrophobia is returned as having occurred in Scotland during the ten years referred to, and that took place in the county of Forfar in the year 1870.

(*b.*) *Morbid Anatomy.*—The effects of the hydrophobic poison are often so rapid and violent in the first instance as to cause the early death of the patient, leaving not a trace of inflammation or other morbid phenomena. More commonly, however, some structural alterations have been found, limited to slight appearances of inflammation of the brain, the spinal cord, or of their membranes, and of the lungs, stomach, or structures supplied by the *eighth pair* of nerves. The brain, the lungs, or the stomach may be either separately or conjointly affected—presenting phenomena in no degree dissimilar to what have been observed in hooping-cough, where the poison seems to act chiefly on the vagus nerve.

The appearances in the membranes of the brain have been generally great congestion, especially of the *plexus choroides*, also effusion of serum, sometimes muddy, into the arachnoid cavity, and into the ventricles. In an interesting case recorded by Dr. R. W. Cunningham, of Her Majesty's 4th Bengal Europeans, the layers of the arachnoid were found adherent in many places, especially along both sides of the longitudinal sinus. The adhesions were quite soft and recent, and flakes of coagulated fibrine floated in the fluid. The brain has, in some very few cases, been supposed to be harder or softer than usual, and to have more bloody points than in health. There has been no lesion noticed in the brain, however, that could be directly connected with the malady; but rather with the mode of death. The pathological changes are mainly those dependent on the asphyxia which occurs in the last stage (TROUSSEAU). Changes in the *medulla oblongata* and the *spinal cord* have not yet received sufficient attention. In the case just referred to, related by Dr. Cunningham, there was a reddish spot in the substance of the *pons Varolii*, having the appearance of inflammatory softening. On the lower surface of the *medulla oblongata*, at the origin of the *seventh, eighth,* and *ninth pair of nerves*, the membranes were highly vascular, thickened, softened, and matted together; but the substance of the nerves at their exit, and of the *medulla*, seemed normal. There are strong reasons for believing that changes actually exist in these parts which escape the detection of our unaided senses, but which the specific gravity test, combined with microscopic examination, may yet demonstrate. The mucous membrane of the pharynx and œsophagus have been seen either greatly congested or diffusely inflamed, as also that of the stomach, and of the trachea and bronchia. The latter have been found covered with a considerable quantity of frothy mucus, while the pulmonary tissue has shown marks of inflammation, though more commonly only of great congestion. The salivary glands have likewise occasionally been observed increased in size, and vascular. In a case of hydrophobia which I had an opportunity of dissecting at Renfrew, near Glasgow (in 1850 or 1851), the most prominent morbid change was visible in the greatly increased vascularity of the lungs, and of the mucous membrane of the back part of the mouth, pharynx, and larynx, as far as the vocal cords. The whole of these parts were covered by a tenacious frothy mucus, tinged with blood. The glands surrounding the papillæ over the back part of the tongue were very much enlarged, not unlike what I have observed in severe cases of *cholera*. So also were the submucous glands of the pharynx, the epiglottis, and the larynx, even in its cavity, and of those beneath the tongue. Inflammatory appearances in these parts have been observed by Morgagni, Babington, Watt, Portal, Troilliet, Copland, and others. In 1875, Wasilieff, of St. Petersburg, examined the brains and heart of a woman aged thirty-two, who died in the wards of Dr. Bokin, from hydrophobia, with marked symptoms of paralysis of the heart. The following parts of the brain were examined microscopically:—Corpora striata, thalami optici, pons Varolii, medulla, and cerebellum. (1.) Some of the nerve-cells of the medulla oblongata appeared turbid, and their outline and nucleus indistinct. Similar, though more pronounced changes, were observed in Purkinje's cells. (2.) In the interstitial tissue of the brain there were large masses of indifferent round corpuscles, of the size of white blood-corpuscles, which were darkly tinged by the staining agent. These elements (very probably white blood-corpuscles) were specially to be found in the perivascular spaces or in their neighbourhood; although some of them, collected into groups of six or ten, lay far from the vessels, amidst the neuroglia. Others lay in the pericellular spaces, and even projected into the protoplasm of the nerve-cells. (3.) The blood-vessels were distended and overfilled with blood-corpuscles, and their epithelium partly swollen up; here and there were vessels whose walls consisted of a finely granular, highly refractive yellow substance, soluble neither in absolute alcohol nor in turpentine. The most pronounced appearance, however, was the presence (specially in the cortical layer of the cerebrum) of a special dimly hyaline and highly refractive substance *in the perivascular spaces.* Sometimes this substance was so arranged round the vessel, that on transverse section it appeared to be surrounded by an irregular ring, exercising so strong a pressure as to narrow its lumen considerably; in other cases this hyaline substance lay in small heaps, which were sometimes arranged around the vessel so symmetrically as to put one in mind of epithelium. This substance was not coloured by any of the staining re-agents, nor did it dissolve in boiling caustic potash or strong acids (acetic and hydrochloric); and a negative result was obtained with absolute alcohol, with turpentine, and on testing for amyloid substance. In other parts of the brain, the perivascular spaces were more or less dilated.

The changes in the cardiac ganglia were these:—(1.) The epithelioi lining of the sheath of the nerve-cells was partly swollen up; in the interior of the sheath, and in the interstitial tissue of the ganglia, round corpuscles of the size of white blood-corpuscles were found. The blood-vessels surrounding the ganglia, with the exception of the large veins, appeared for the most part to be bloodless. (2.) In the nerve-cells themselves, the protoplasm appeared more or less dim, consequently their nuclei were either indistinct or invisible; in some cells there was a collection of finely granular pigment. The most pronounced *never failing* changes consisted in this, that the nerve-cells never completely filled their sheath; but a free space always existed between, across which only processes from the nerve-cells stretched. (A similar appearance was described by Lubimoff in the œdema of the sympathetic ganglia in cardiac disease.) Micrometric observations were made to ascertain whether this space was due to shrinking of the protoplasm of the cell, or to œdema of its capsule. It was proved that it was due to an accumulation of an œdematous fluid between the protoplasm and its sheath. This œdemic condition of the cardiac ganglia must be of not unfrequent occurrence, for the ganglia lie in cellular tissue rich in fat, which affords little resistance to the dilatation of the capsule, and the number of blood-vessels surrounding the ganglia predispose to it (WM. STIRLING, D.Sc., M.D., in the *London Medical Record*, Jan. 15, 1877).

At the Pathological Society of London, June, 1877, Dr. Gowers showed microscopical sections illustrating changes in the medulla oblongata and spinal cord in four cases of *hydrophobia*. The changes found resembled those found by Benedikt in the convolutions in the dog, and by Coats in the lower centres in man. In all four cases, the vessels of the grey matter were greatly distended. In three of the cases, the larger veins presented aggregations of small cells within their perivascular lymphatic sheath. In some they formed a single layer; in others, they were so densely packed as to compress the vessel they surrounded. In most parts, imilar cells were scattered through the tissue among the nerve-elements, and in some places near the hypoglossal nuclei they were so dense as to orm "miliary abscesses." Adjacent to many vessels were areas of granular degeneration. In two of the cases, many of the larger vessels, hiefly veins, contained clots, parts of which were evidently of *ante-mortem* ormation. By some of these the appearance of embolism was closely imulated. Attention was drawn to the indications of formation *in situ* fforded by the gradation between the normal and the altered clot, and y the curved lines of pressure to which the clot had been subjected mmediately after its formation. In one specimen, the inner coat of vein was thickened opposite the older part of a clot, which was reduced size correspondingly, as if formed secondarily to the change in the coat of e vein. In the vessel there were several cells in the perivascular sheath, d leucocytes within the old clot, and also in the inner coat of vein, fording strong evidence that the cells outside were migrated corpuscles. he nerve-cells presented comparatively slight changes, being merely wollen and granular in some cases, and surrounded with granular egeneration here and there. These were most marked in and about the ypoglossal, pneumogastric, and glossopharyngeal nuclei; slighter in e nuclei of the auditory, facial, and fifth nerves; slight also in the cord, and even less in the upper part of the pons. Dr. Clifford Allbutt had also found less definite changes to be most distinct in the medulla. In Dr. Gowers' cases, the changes were most marked in the region of the respiratory centre in the medulla. Similar microscopic preparations were more recently shown at the Glasgow Medico-Chirurgical Society (Feb. 20, 1880), by Dr. David Foulis and Dr. Joseph Coats, illustrative of the pathology of hydrophobia. The sections of the chief nerve-centres showed the presence of numerous leucocytes in the perivascular spaces, as had been previously pointed out by Dr. Coats and other observers; but, in addition, an examination of other organs of the body, as the liver and salivary glands, revealed the presence of these leucocytes to the same large extent in their connective tissue. Although these changes have been demonstrated to exist in dogs suffering from this disease, this was one of the first occasions in which they have been shown in these parts of the human body.

There seems every reason to believe that the virus of hydrophobia mainly exerts its influence on the sensory and emotional regions of the central parts of the nervous system, and in many respects the lesions are very similar to those seen in tetanus. The order of severity of the lesions seems to be—the medulla, the cord, the cerebral convolutions, and the central ganglia of the encephalon. But there seems to be no constant and definite lesion. There is also a profound disturbance of all the larger organs of the body, as occurs in other acute specific febrile diseases, of which hydrophobia must be regarded as one, and with which it perhaps ought to have been classed rather than here.

Diagnosis.—When hydrophobia is fully formed there is no disease with which it can be confounded; but there are many reported cases in which the imagination of a patient bitten by a dog has been so powerful as to induce symptoms resembling the disease. In hysteria the difficulty of swallowing exists, but no other symptom. Tetanus is the disease with which rabies is most apt to be confounded; yet the differences are sufficiently marked. The spasm of the muscles is more continued in tetanus, less remitting, and never intermitting. The jaw is usually much in motion in hydrophobia, in frequent attempts to clear the mouth and throat from the peculiar tenacious mucus; in tetanus it is fixed. Tetanus is rarely attended with aversion to liquids; on the contrary, the bath is grateful; nor are the tetanic paroxysms increased by the sight, hearing, or touch of fluids. Also tetanus makes its accession usually at a much earlier period after infliction of the injury. Physiologically, while tetanus is a disease of the true spinal system, hydrophobi involves the brain also, as evinced by the disorder of intellectual functio and special sense, even early in the disease. Further, the two disease differ greatly in their mode of induction. Tetanus, in the traumatic case is caused by irritation of a nerve, and by disease of the spinal marrow i those which are idiopathic. Hydrophobia is the result of a specif poison introduced into the circulation, and thence affecting the nervo system as a poison would (Miller). While in tetanus the stimul which excites the paroxysms operates through the true spinal cord; hydrophobia it is often conducted from the ganglia of special sense, even from the brain, so that the sight or sound of fluids, or even the id of them, occasions equally with their contact, or with that of a current of air, the most distressing convulsions" (Carpenter).

Prognosis.—The disease is almost invariably fatal. There are few instances of any patient or animal suffering from this disease having recovered. But it is important to know that the virus never inoculates when it is applied to a sound surface: and also, that only a small proportion of those who are bitten by rabid dogs or cats suffer from this disease. The proportion has been variously estimated at from 5 to 50 per cent. It has also been estimated that only ninety-four persons are known to have died out of one hundred and fifty-three bitten, making the chances of escape as three to two nearly. It has been thought that this occasional immunity does not arise out of any want of susceptibility to the action of the poison, but that from the person being bitten through his clothes, the dog's tooth has been wiped clean from all saliva and venom before it had penetrated the skin. Menières, however, says he met with seven cases in which the dog must have bitten through several folds, and yet they all proved fatal, showing, as he imagines, the little importance of dress as a protection from this malady.

Symptoms.—The wound inflicted by the bite, whether neglected or dressed, generally heals up kindly, leaving a cicatrix, and for a time the patient usually suffers no other derangement of health than the depression of spirits which his apprehensions are calculated to excite. A few weeks or a few months having elapsed, the latency of the poison terminates, and the disease becomes fully developed. After this local incubation of the poison is complete, its specific action appears to be exercised upon the *medulla oblongata* and the *eighth pair of nerves*, and subsequently lesions of the structures supplied by the branches of the *eighth pair*. The action of the poison appears in the first instance to be made distinctly manifest by the œsophageal branch of the *eighth pair*, producing that derangement of function which gives rise to the characteristic symptom of the disease, or to the extreme difficulty of swallowing, especially of fluids; while the spasmodic catching of the breath, consequent even on touching the lips with any liquid, proves that the recurrent nerve is equally affected. Subsequently the eye and ear become distressed by every ray of light or impulse of sound, and likewise the sense of touch is most painfully excited on the slightest breath of air passing over the surface of the body, all of which distinctly show that the central and spinal nerves must be functionally affected. In a still more advanced stage the suspicion, the irritability, the violence, and generally the outrageous and uncontrollable derangement of mind which often seizes the patient, bring on convulsions, which show that the brain itself is likewise a principal seat of the action of this terrible poison, especially the region of the *medulla oblongata*. Its course is usually divided into three stages—the first stage comprising the symptoms which precede the difficulty of swallowing; the second commences with the difficulty of swallowing, and terminates with the overthrow of the mind; the last stage embraces all the concluding phenomena, which are mainly those of asphyxia.

The first stage commences, in a few instances, by the patient's attention being aroused to a numbness extending towards the sensorium from the injured part (which, if an extremity, may become tremulous); or pain is felt in the cicatrix, sometimes severe and sometimes trifling, which shoots up the bitten limb, following in general the course of the nerve towards the trunk. It shoots as if towards the heart, but there is no evidence of lymphatic absorption. Pain, however, is by no means constant, and is for the most part absent. In the latter case the first symptom is chilliness, with headache, or a slight attack of fever, and the patient is more excited or depressed than usual. It is sometimes described as the *melancholic stage.* The patient may then be feverish with shivering, suffering from a dry mouth and thirst. He is unable to sleep, and experiences epigastric uneasiness, with some sighing and anxiety. He is generally pale, his eyes restless and pupils dilated. He becomes fidgety, and cannot remain at rest; and he constantly speaks in short sentences, delivered in a jerky, abrupt manner. The heart's action is increased in frequency. There is loss of appetite, and perhaps nausea and vomiting. A feeling of constriction about the fauces, with disinclination to swallow fluids, may appear early. The respiration becomes quickened and sighing; there is general hyperæsthesia, and a tendency to priapism and seminal discharges (Bristowe). These premonitory warnings last but a few hours, or at most a few days, when the fatal but characteristic symptom, "the difficulty and dread of swallowing"—a symptom which distinguishes this malady from all others—appears, and the hydrophobic stage commences.

The second, or hydrophobic stage, is ushered in with a great difficulty, if not an utter impossibility, of swallowing any liquid—a symptom which generally comes on suddenly; and such horrible sensations accompany the effort, that whatever afterwards even recalls the idea of a fluid excites violent agitation and aversion. The muscles of *deglutition* seem to be specially affected, while those of *mastication* are not so. The symptoms point to special implication of the *eighth pair of nerves.* "The distressing thirst, accompanied by the dread of making the effort to satisfy it; the wild and wandering expression of the countenance; the suffused eye; and, beyond all, the helpless, purposeless, unremitting restlessness of this disease, suggestive of the undefined apprehension of something more terrible than death itself, under which the senses reel and the intellect staggers, distinguish *rabies* from *tetanus* and all other convulsive affections, and mark it as the most dreadful of diseases to suffer or to witness" (Le Gros Clark). "To command the hydrophobic patient to swallow is to tell him to strangle himself" (Mead). Some patients who have been able to give some account of themselves describe the hydrophobic sensation as a rising of the stomach which obstructs the passage; others, as a feeling of suffocation or a sense of choking, which renders every attempt to pass liquids over the root of the tongue not only impossible, but which excites convulsive action in the muscles of the larynx, pharynx, an abdomen. In this state, says Dr. John Hunter, "the patient finds som relief from running or walking, which shows that the lungs are not yet th seat of any great oppression." The hydrophobia, or inability to swallo fluids, is shortly accompanied by an increased flow of saliva (the "*hydr* *phobic slaver*"). This secretion, as the disease advances, is not only copio but viscid, so that it adheres to the throat, and causes incessant spittin The quantity expectorated may be taken as the measure of the violen of the disease. The ejection of the saliva from the mouth as it forms, is mainly owing to the dread of attempting to swallow it, causing the spasmodic paroxysms of suffocation. By some this increased flow of saliva is considered as an effort of the system to eliminate the poison through these excretory glands; and therefore mercury, in large doses, to promote salivation, has been recommended to promote elimination in this way, and to reduce the extreme excitability of the nervous system (LIGGET), but with no such result. The aversion to fluids is no sooner established than another series of symptoms of dreadful severity, or a highly exalted state of every corporeal sense, is added. Indeed, it is hardly possible to depict the sufferings of the patient from this cause; for not only does he shrink at the slightest breath that blows over him, but the passage of a fly, the motion of the bed-curtain, or any attempt to touch him, produces indescribable agony, almost amounting to convulsions. Dr. Elliston states that the effect produced by these causes very much resembles that produced upon stepping into a cold bath. Sexual excitement may also continue, and greatly disturbs the patient; and he passes urine frequently. M. Robin has analysed such urine. He finds that in quantity it is considerably diminished. The density is increased, and the acidity remains, whilst there is a decrease of solid materials—namely, of urea and chlorides. The amount of the uric acid is increased, as also its proportion to urea. An increased proportion of the phosphoric acid to urea is also noticeable, due to the acid retaining its normal proportions, whilst the quantity of urea greatly diminishes. The phosphoric acid is present in the urine as sodium and potassium phosphates; earthy phosphates, on the other hand, are markedly diminished in quantity. Albumen is present, sugar is absent; fat is found in large quantities; leucin and margaric acid, as well as those various kinds of bacteria which are seen in urine which has begun to putrefy, are also found. This increase of alkaline phosphates, of potassium phosphate, and the presence of fat and margaric acid, are facts which *seem* to indicate an active *denutrition* of the nervous centres. *Leucin*, *calcium*, *hippurate*, and the *extractives*, have a relative value in the view that the two former are not met with in the normal state (D'ARCY POWER, in *London Medical Record*, March 15, 1879, p. 110.) The sense of sight is no less a source of terror than that of touch; the approach of a candle, the reflection from a mirror or other polished surface, occasions the same distressing effect. The hearing is as strongly affected as the other senses, so that the least noise, and especially that of pouring out fluids, throws the patient into a fearful paroxysm. An attendant who sat up with a hydrophobic boy made water within his hearing, which threw the sufferer into a most violent agitation. The degree to which this painful state of the senses arrives may be understood when it is stated that Majendie records the case of a deaf and dumb child who heard distinctly in this stage of the disease. The patient, thus incessantly harassed and pained by every circumstance around him, becomes peevish and irritable; and at length sees his family, relations, and strangers, with feelings of dislike and aversion, and sometimes apparently with horror.

The third stage commences by the cerebral functions becoming disturbed, the mind being either filled with dreadful apprehensions, or being so completely overthrown that paroxysms of uncontrollable impulsive violence follow. A rabid impulse overtakes the patient to tear in pieces who and whatever opposes him. This rabid impulse greatly distresses him; and it is often strongest against those to whom he is most attached, although he struggles to suppress it. In this stage horror is strongly depicted on the countenance; every symptom is aggravated, the saliva grows thick and ropy, while the poor sufferer, not daring to make the slightest attempt to swallow, spits it out incessantly, oftentimes with frequent retchings and vomiting. In this state he sometimes turns black in the face, falling into convulsions, in which he expires from sudden asphyxia; or, exhausted by his great efforts, a sudden calm ensues, and a quiet death, usually from paralysis of the heart.

Treatment.—As there are but few authenticated cases of recovery from hydrophobia, so there are few instances of any mitigation of the symptoms by the use of medicine. All that remains is to mention the most leading experiments that have been made, with the hope that, as they have not been successful, they may not be wantonly repeated.

Dr. Hamilton gives twenty-one cases, and adds, "many hundreds more are on record," in which *venesection* has been unsuccessful, though frequent and copious. *Opium* has been given by Dr. Babington, to the enormous amount of 180 grains of solid opium in eleven hours, without the slightest narcotic effect, or the slightest mitigation of the symptoms. Nord has given a drachm of *belladonna* in twelve hours, without any benefit. Dr. Atterly gave to a child eight years old two drachms of *calomel* by the mouth, and rubbed in two ounces and a half of strong mercurial ointment in a few hours, with an equal want of success. A case, however, is related by Ligget, which is said to have been successfully treated by half-drachm doses of *calomel*, given to the extent of ptyalism, induced in three days, after four and a half drachms of calomel had been taken. The case really appears to have been one of hydrophobia; and recovery is said to have been complete by the twelfth day (*Amer. Quar. Journal of Med. Science,* Jan., 1860). *Iron, arsenic, nitrate of silver, camphor, musk, cantharides, turpentine, tobacco, acetate of lead, ammoniacal solutions of copper, hydrocyanic acid, galvanism, strychnine, nitrous oxide, chlorine, and guaiacum,* have all been given in equally large doses, but have signally failed. In addition to these, Ploucquet, in his *Literatura Medica Digesta,* has enumerated nearly 150 other medicinal remedies. The failure of every remedy by the mouth, and the inefficacy of *opium,* of *morphine,* and of *laurel water,* even when injected into the veins, so convinced Majendie that in hydrophobia the constitution was armed against the action of any medicinal substance, that on a patient labouring under this disease being brought to the Hôtel Dieu, he determined to rely for all treatment on an *injection of warm water into the veins.* The patient, at the time of the operation, is represented as being absolutely insane, so as to require to be restrained. In this state, and with a pulse of 150, Majendie injected into his veins, in the course of two hours and a quarter, two pints of water, at the temperature of 100°. At the conclusion of this operation the pulse had fallen to 80, and the patient recovered his senses, so that restraint was no longer necessary. The sequel, however, renders it doubtful whether this mitigation was desirable at the price of the intense suffering which followed. The poor man lived eight days afterwards, but the despondency and mental agitation quickly returned, and at the end of three days the poison (or the state of the blood induced by it and the warm water) appeared to set up a new series of actions on the synovial membranes of the wrists, elbows, and knees, attended with excessive pain, so that he was unable to bear the weight of the bed-clothes, and he died in great torture. The articulations thus affected were found, on *post-mortem* examination, to be greatly inflamed, and their cavities filled with pus. This case is remarkable as being the one in which life was prolonged for the greatest period of time recorded of this disease. The experiment has since been repeated by Gaspard and others; but the mitigation, if any, has been so slight and transient as to give no encouragement for repeating it; and, tried on the rabid dog by Youatt and Mayo, t proved eminently unsuccessful.

The property which some animal poisons have of controlling and of nterrupting the actions of other morbid poisons on the constitution has caused even animal poisons to be tried in the cure of this disease. The rapid and powerfully acting poison of the viper led to the hope that the bite of that reptile might prove an antidote to the hydrophobic virus; but the experiment, tried in France, Germany, and Italy, upon animals, has been entirely unsuccessful. M. Grindard conceived that the vaccine virus might influence hydrophobia, and he vaccinated a hydrophobic child in three places, and afterwards injected five charges of vaccine lymph into the veins; but the child died without any marked remission, nd in the usual time. The following draught has been found rather to promote euthanasia than to hold out any prospect of cure:—

℞ Spirit. Æther. Sulph., Tinct. Opii, āā ♏xx.; Spirit. Ammon. Aromat., ʒss.; Chloroform, ♏xx.; Mist. Camph., ℥iss.; *misce.* To be given s often as may be *considered safe* (CUNNINGHAM, CARDEN).

On the same principle chlorodyne may too be given. The vapour bath s sometimes useful in moderating spasm. Having regard to the symptoms generally, and to the morbid appearances as being in many respects similar to tetanus, the remedies recommended in that disease might be successful in some cases of hydrophobia. Sialagogues have recently been advocated upon theoretical considerations, especially the *extractum jaborandi uidum,* of which one drachm represents one drachm of the leaves as repared by Messrs. Gall & Co. *Curare* has also been employed, but not with success, mainly at the suggestion of the late Dr. Francis Gibson of t. Mary's Hospital, London. For this purpose Messrs. Gall & Co. have repared an *injectio curare* (three minims being equal to one-third of a rain), which it is advised should be injected at intervals of a quarter of n hour or more, as soon as indications of the physiological action begin o diminish.

Preventive Treatment.—The probabilities are, that unless the operation f excision, or cauterisation, be performed within a few minutes after the ite of the rabid animal, it is impossible to save the patient from the tal disease, which, according to the susceptibility of his constitution, may threaten him at any moment. In all probability no prophylactic medicine exists in nature, and the administration of any potent substance by way of prevention is worse than useless; for, without protecting the patient, it may injure his constitution. Mild remedies, if they tend to tranquillise his mind and appease his apprehensions, may be innocently employed.

The theory which maintains that a zymotic incubation first takes place in the wound, by which the poison is originally implanted, suggests the most rational prophylactic—namely, to destroy entirely by *potass fusa* the whole cicatrix, where practicable; or, by some other surgical means, entirely to remove it at as early a period as possible, and *previous* to the occurrence of symptoms.

SUNSTROKE.

LATIN EQ., *Solis Ictus;* FRENCH EQ., *Coup de Soleil;* GERMAN EQ., *Sonnenstich;* ITALIAN EQ., *Colpo di Sole.*

Definition.—*An affection of the nervous system associated with vertigo, and sometimes with headache, or the gradual accession of listlessness and torpidity, with a desire to lie down* (LONGMORE, BARCLAY). *These febrile phenomena may culminate in more or less sudden and complete insensibility, without the power of sense or motion, the breathing rapid, and getting more and more noisy as death approaches. Convulsions of the extremities usher in a complete state of coma, in which the patient gradually dies. The approach of death is indicated by the failure of the heart's action, the fluttering of the pulse, the irregularity of the respiration; and the fatal event may supervene within five minutes to a few hours after the disease has become fully expressed. Death is either by syncope, apnœa, or by a combination of the two. In cases which recover, various sequelæ are apt to supervene, such as forms of paralysis, more or less complete, choreic movements, melancholia, and other forms of disorder of the intellect.*

Pathology.—(*a.*) *Causation.*—This singular and fatal affection of the nervous system has been described under a great variety of names—e.g., *Heat apoplexy, Heat asphyxia, Coup de soleil, Insolatio, Ictus solis,* and lastly, *Erythismus tropicus.* Notwithstanding that sun heat and the sun's rays have been considered the main agent in producing this disease, it is not less true that the full expression of the disease not unfrequently occurs at midnight. The name implies a common, and certainly a most powerfully exciting cause of a disease, which has been variously and erroneously described as of the nature of apoplexy, or of some form of continued fever. A very great variety of views have been put forward regarding the pathology of sunstroke; and the following account of its nature is based upon the very interesting reports which have been given by many Indian medical officers, and especially by Surgeon-General Longmore, C.B., Professor of Surgery in the Army Medical School, when he was medical officer of the 19th Regiment at Barrackpore; by Surgeon Butler at Mean Meer; by Dr. Barclay, of the 43rd Light Infantry, while on the march from Jubbulpore to Calpee; and by Sir Joseph Fayrer K.C.S.I.

Instances of the occurrence of *sunstroke*, and the circumstances unde which it has been observed to occur, will best convey an idea of its nature. One of the earliest accounts of *sunstroke*, in which its pathology was distinctly recognised, is that given by Mr. Russell, of the 73rd Regiment, while in medical charge of the 68th, in May, 1834. The regiment had then recently arrived in Madras—a fine corps of men in robust health. The funeral of a general officer being about to take place, the men were marched out *at an early hour in the afternoon, buttoned up in red coats and military stocks, at a season, too, when the hot land winds had just set in*, rendering the atmosphere dry and suffocating even under the shelter of a roof, and when the sun's rays were excessively powerful. The funeral procession forthwith advanced, and after having proceeded two or three miles, several men fell down senseless. As many as eight or nine were brought into hospital that evening, and many more on the following day. Three men died—one on the spot, and two within a few hours (GRAVES' *Clinical Lectures*, Vol. I., p. 181). A nearly parallel example is related by Sir Ranald Martin as having occurred in the experience of Dr. Miligan, of the 63rd Foot, from the exposure of his corps to the sun during a military funeral at Madras. The greater number of the men were in the prime of life; but there were some old soldiers who had served twenty years and upwards in the West Indies, and who were much broken down by service and intemperate habits. The entire corps had just arrived from the Australian colonies, where spirituous liquors can be had on easy terms. The regiment landed at Madras in the month of May; and from the date of the "untoward circumstance" of the funeral, the hospital became filled with cases of fever. Two men dropped down and died on the day of the funeral, and for several days afterwards the fever cases augmented considerably (*The Influence of Tropical Climates*, p. 205).

The dreadful march of the 43rd Light Infantry from Jubbulpore to Calpee, a march extending from the 24th December, 1857, to the 17th of April, 1858, when the regiment arrived at Jubbulpore, furnishes the most instructive and melancholy example of the causation of *sunstroke.* It must be remembered that the regiment had previously marched throughout the greater part of the length of the Madras Presidency. During this first march the regiment enjoyed great immunity from sickness, owing to the sanitary precautions which had been taken. The march, however, told seriously upon the strength and endurance of the men in general. They lost condition, and in a great measure their robust appearance; and they were in urgent need of rest. But after a rest of five days only they had to commence the march again from Jubbulpore to Nagode, a distance of 163 miles, arriving there on the 8th of May. The heat on the march was excessive, and it told very much on the health of the men, already exhausted as they were by a previous march of almost unexampled length. After having been *four months and thirteen days in the field, and after they had marched* 969 *miles*, a fatal case of *sunstroke* occurred; and from that date cases gradually increased in frequency. At Nagode the regiment remained eight days; and although the indications of exhaustion in the altered looks of the men, their loss of flesh, and their evidently failing strength, were so obvious that they forced themselves on the observation of every one, *yet the men were ordered still to continue their march to Banda.* The periodical hot winds were blowing at the time day and night, with scarcely any intermission, and the heat of the weather was almost unbearable. After leaving Nagode they were obliged to encamp for four days in the bottom of a deep and narrow ravine, with nearly precipitous sides upwards of a mile in height. The heat in this valley was insufferable, and exceeded anything the regiment had ever been exposed to before or since. The thermometer varied during the day from 115° to 118° Fahr. in the largest tents, and in the smaller ones it reached 127° Fahr. Night brought but little relief. On one occasion Dr. Barclay observed the thermometer standing at 105° Fahr. at midnight. Such was the overpowering effect of the heat in this "valley of the shadow of death," that even some of the natives were struck down, and died with all the symptoms of *sunstroke* in less than an hour. The number of cases among the men of the regiment, especially during the first day, was very great. They were carried into the hospital tents at every hour of the day and night; and although a large proportion of them recovered, two officers and eleven men were buried under one tree in the neighbourhood of the camp. Marching was resumed on the 24th of May. On the 27th the regiment arrived at Banda, a distance of about 100 miles from Nagode, having lost during the march two officers and nineteen men. The health of the regiment improved daily during a short stay at Banda; but the men were still in a miserable state of exhaustion when the regiment *again began to march for Kirwee on the 3rd of June.* At Kirwee five men died from *sunstroke*, and on the return to Banda two more died. The regiment *now marched for Humeerpore on the 28th of June, en route for Calpee.* The weather was again excessively hot, and the men suffered severely. They were by this time completely worn out and prostrated. There was scarcely a man in the regiment whose strength was not reduced to a level with that of a child; and the officers were not in a better plight. Many men broke down altogether, and had to be carried as could best be managed—in *doolies*, in sick carts, or on baggage and commissariat carts after they were filled. It was painful to see many others who, a few months before, had been in robust health and full of vigour, staggering from weakness as they endeavoured to keep up with the column, throwing themselves down completely exhausted at every halt, and scarcely able to rise from the ground when the "*assembly*" sounded. Calpee was reached on the 5th of July, and between the 28th of June and the 7th of July one officer and five men had died.

These details are given for the purpose of showing *the effects of protracted exposure to intense heat in a body of men in the field debilitated by fatigue and want of rest.*

It may now be shown how the effects of protracted exposure to intense heat in a body of men may operate upon them when in *quarters*, as described by Mr. Longmore. He records sixteen cases of *sunstroke* as having occurred in the 19th Regiment between the 23rd of May and the 14th of June, 1858, when they were quartered at Barrackpore. The period was marked by an unusually elevated degree of temperature, and generally by great dryness of the atmosphere. The quarters were of a temporary and imperfect nature, some of them being merely hired *bungalows.* Of the sixteen cases, five occurred in non-commissioned officers, and eleven in privates of the regiment, the proportion attacked being greatly more among the former than among the latter; their occupations, especially when on orderly duty, caused them to be more exposed than the men with whom they lived. Simple exposure to the external atmosphere and the solar rays were not of themselves sufficient to induce the disease. Of the five non-commissioned officers attacked, two were on duty at the time as orderly sergeants; one had had fever for several days, but had not been exposed to the sun on the day of attack; one had been slightly exposed; the fifth not at all. Of the privates, eight were attacked by the disease within doors, and three while on sentry. No cases occurred of sudden sunstroke —*i.e.*, of insensibility instantly induced by the direct rays of the sun in a man previously healthy. The characteristic feature with regard to atmospheric temperature, when Mr. Longmore's cases were observed, was the little variation of it night or day. There was no rain; and the ground and buildings became so heated that, long after sunset, the radiation of heat maintained a high temperature within doors. Prolonged high atmospheric temperature was recognised as the essential cause of the attack; but nervous depression from solar exposure, fatigue, and previous illness, were associated with that prime or essential cause.

But a most important element of causation is still to be mentioned—namely, *the influence of vitiated air when men are congregated without sufficient ventilation.* One-third of the cases, and nearly half the deaths, occurred under such circumstances; so that, *in quarters,* the predisposing causes of *sunstroke* may be comprised in the following conditions:—(1.) Prolonged atmospheric heat, with a dry and rarefied state of the air; (2.) Nervous exhaustion; (3.) A contaminated atmosphere (LONGMORE, TAYLOR); while (4.) An increase of the average prevailing temperature sufficed to act as the more immediate exciting cause for the development of *sunstroke.*

The experience of Surgeon Butler at Mean Meer has led him to write, that "when the thermometer ranges beyond 98° Fahr. in crowded barracks, cases of (heat) apoplexy almost invariably occur." Dr. Crawford also, in writing on *sunstroke,* notices an electric condition of the atmosphere as influencing the respirability of the air, and refers especially to that peculiar state "in which the hairs of a horse's tail repel each other, in which the hairs of the head stand on end, in which a man exposed to its influence becomes irritable, headachy, and restless, without knowing exactly what is the matter with him. Such a state of atmosphere will generally be found to exist in localities where cases of *sunstroke* occur, whether such localities be the crowded barrack, in the still more crowded cantonment, the tented field, or the march in column through the still valley, the deep gorge, or the thick forest." Dr. Barclay notices that cases of *sunstroke* occur with increased frequency immediately before a thunderstorm, and that they cease as soon as the electrical discharge has taken place.

Generally, it may be stated that heat, and atmospheric conditions of the nature already indicated, concur with the following predisposing circumstances to induce cases of *sunstroke:*—(1.) Plethora and unacclimation; (2.) Debilitating causes of every kind, particularly such as lower the tone of the nervous system or increase its irritability—*e.g.*, excessive fatigue and prolonged exposure in extreme temperature, prolonged marches, bad ventilation in tropical temperatures; (3.) A febrile state, from whatever cause. As a general rule, Dr. Barclay found that plethoric men incur greater danger from exposure than others. (4.) Intemperate habits; (5.) Exposure to an atmosphere highly charged with electricity (CRAWFORD, BARCLAY). (6.) During the season of prevalence of sunstroke the temperature would seem to have ranged from 96° to 120° Fahr. in the shade—extremes of atmospheric heat chiefly observed on the Coromandel Coast, Central India, the North-West Provinces, Scinde, and the Punjaub (MOREHEAD, l. c., p. 615).

The effects of the constant stimulation of excessive heat in producing nervous irritability and pervigilium cannot be doubted (MARCH, BARCLAY); and Indian medical officers can bear witness, from personal experience, to the extreme misery resulting from such excessive stimulation, in combination with the conditions already mentioned.

Various opinions are entertained with regard to the mode of action of the various agents which combine to produce this singular affection of the nervous system. The phenomena during life, in one set of cases—namely, those of simple syncope from exhaustion caused by heat—may occur during great fatigue or over-exertion, or when there is depression of vital power from any cause during exposure to a high temperature, as in the case of stokers and engine-room men of steamers in the Red Sea or the tropics, when the temperature rises to 120° Fahr. and upwards in the vicinity of the furnaces where they are employed; or in the case of men (especially Europeans) in the tropics who are exposed to the intense heat and light of the sun's rays which, taking effect on the head, neck, and body, produce a condition like shock acting through the vagus and vaso-motor system. There is depression of nerve force and of muscular power, the skin is pale, cold, and moist; the pulse feeble. Death may occur in this state from failure of the heart, but complete recovery more frequently occurs (Sir JOSEPH FAYRER, in *Brain*, Oct., 1879, p. 298). The *post-mortem* appearances are in accordance with death from coma, slowly induced, or from syncope.

The manner in which heat acts in the production of such asphyxia as is seen in a second set of cases of *sunstroke*, has been variously interpreted by writers on the subject; and observations are very much to be desired as to the exact range of temperature of the body-heat in such cases. It is known, however, to be excessive, and the increased temperature of the blood, which results from prolonged exposure to great heat, must have a deleterious influence upon the constitution during the metamorphosis of tissue going on under such circumstances. Intense heat applied to the whole body tends to produce death by *syncope*, as in concussion of the brain (ALISON). Heat acting on the peripheral distribution of the nerves, and accumulating in the system, as it seems to do in *sunstroke*, produces such an effect on the heart, the lungs, and the brain, as to produce the phenomena of *syncope* and *coma*. Asphyxia and apnœa may come on after premonitory symptoms of depression and weakness, during exposure of the head and spine to the direct rays of a powerful sun, whe the atmosphere is much heated and the nervous energy is depressed b over-fatigue, illness, or dissipation. The brain and respiratory nerv centres are overwhelmed by the sudden rise of their temperature, respiration and circulation fail, the latter probably owing to inhibitory action the vagus. The heart's action is really brought to a close by heat, the same manner as it has been shown by Claude Bernard and Lauder Brunton that the effect of a very high temperature on animals is first to accelerate, and finally to stop the heart (and especially the ventricles) in a state of tetanic contraction.

The pre-existing cutaneous derangement in all the cases, the total inaction of the skin, its dryness and intense heat, betoken an accumulation of heat in the blood which cannot fail to influence the delicate textures of the brain and lungs. The "embarrassed, hurried, and heavy breathing;" the "sense of weight over the sternum" (Longmore, Barclay); the "catching at the chest;" the "constricted feeling, as if of approaching suffocation, caused by wind at a temperature of 112° Fahr." (M'Grigor), indicate that physiological state of "*anxietas*" which prompts to such acts of inspiration as are seen on the approach of *syncope*, or of *apnœa*, from depression of the nervous influence of the *medulla oblongata*. Pollution of the blood, from the prolonged continuance of function under such circumstances as impair the normal action of the skin, the lungs, and the kidney, produced by the atmospheric conditions already described, and the phenomena of the disease in its severe form, denote the culmination of functional efforts to get rid of the rapidly accumulating elements of disintegration which must have resulted—as indicated by progressive emaciation, augmentation of animal heat, and total suppression of the cutaneous function.

Most observers are agreed that intemperance cannot be charged with being the immediate exciting cause of the disease, although it is a universally acknowledged predisposing cause. The men of Dr. Barclay's regiment were extremely temperate, robust, and well-formed men; and Mr. Longmore gives a no less favourable account of his men as to temperance. The drunkard, indeed, is rarely to be found in the ranks under circumstances favourable to the development of *sunstroke*. He is either in prison, or skulking at some depôt, or in some hospital, where he may be left for want of transport; or, if with a regiment, "the pains" come on opportunely, to save him from fatigue and danger. The vascular system of a habitual drinker soon shows unmistakable indications of over-stimulation in the suffused eye, the bloated countenance, the profound sleep which follows the slightest indulgence, and the subacute meningitis which sooner or later supervenes in India (Crawford). Such are not the kind of cases which furnish the deaths from *sunstroke*. When exposed to the stimulus of a tropical sun, such cases may sink under it, as they would under any other great excitement; and a debauch or an ephemeral fever will alike predispose a man to an attack of *sunstroke*, inasmuch as both may bring about that state of nervous depression which seems essential to he occurrence of this disease.

"The most serious cases of sunstroke are those that come on under cover, by night as well as by day, and apart from the direct solar rays. The ardent fever of India is of the character supervening on the ordinary phenomena of ephemeral fever. Heat alone, especially when the atmosphere is loaded with moisture, so as to prevent evaporation from the person, is the real cause of the disease. Malarious and hygrometric conditions have no special influence beyond that which they may exert on the general vigour of the constitution, thus predisposing him to suffer. The dry atmosphere of Upper India, with its hot winds, is much better tolerated than the damp atmosphere of Lower Bengal, or Southern India, though the temperature is lower. Hot dry air favours evaporation, and thus keeps the body cool; whilst in the damp air, as evaporation is diminished the natural cooling power is greatly diminished. Vigorous healthy persons of moderately spare frame, with sound viscera, and who are of temperate habits, if the atmosphere be pure and moderately dry, can sustain a great amount of heat. Acclimatisation has also some influence in conferring toleration. Fresh arrivals in the tropics are more prone to suffer than those who have become accustomed to the climate, and have learned how to protect themselves. . . . The extent and duration of the toleration of heat depend much on the vigour of constitution and actual state of health. The refrigerating powers of the body when in health, enable it to support a very high temperature, considerably above that of the blood. Thus, in hot winds little inconvenience is felt so long as perspiration is free; but when that fails, suffering soon ensues, and the danger is great" (Sir Joseph Fayrer, l. c.).

(*b.*) *Morbid Anatomy.*—In Mr. Longmore's cases, in which a vitiated atmosphere, from the want of ventilation, was associated with the extreme heat, the appearances after death were those usually found in death by asphyxia—namely, excessive engorgement of the lungs, amounting to complete obstruction of the pulmonary circulation. Some parts of the lungs had all the appearance of true interstitial apoplexy. Cerebral congestion was less marked in character and less constant in amount, and it seemed to be secondary to the failure of the due performance of pulmonary functions, resulting, perhaps (as Mr. Longmore suggests), from loss of tone in the vessels, and from the enfeebled action of the heart, consequent upon the imperfectly oxygenated blood it was receiving. The congestion of the head was generally expressed by engorgement of the vessels of the *pia mater* and *choroid plexus*, and by numerous *blood-puncta* in the substance of the cerebrum, as shown on section. The appearance of the brain indicated general sanguineous determination without serous effusion; and when serous effusion had taken place, it was generally into the cavities of the lateral ventricles, and sometimes into the subarachnoid space. Other observers record engorgement of cerebral meninges, but no congestion of brain-substance (H. C. Wood). Others again record marked cerebral congestion (Flint); or the vessels of the dura mater full of dark liquid blood, easily pushed along by the handle of the scalpel.

The investigations and experiments of Obermier show that the symptoms of *sunstroke* or *insolatio,* do not, as was formerly supposed, depend on hyperæmia of the brain. The characteristic symptoms consist in *paralysis* of all the functions of the brain, occurring either suddenly or gradually. In the latter case the paralysis is preceded by excitement, delirium, and other symptoms of cerebral irritation. In our country, latitude, and climate, the action of the sun's rays is not alone sufficient to induce such severe attacks as are seen in the tropics; but if great fatigue is superadded to a very hot day, and if, at the same time, the action of the skin is in abeyance, sweat being very little, then a severe attack may be the result. Radiation of body-heat is reduced to a minimum, while the production of body-heat is increased by active muscular exertion there is overheating of the body and increase of body-temperature to degree incompatible with the continuance of life. Fulness of the veins of the meninges is no indication of a hyperæmic brain: under such a condition of fulness the brain-tissue is generally anæmic. Others record the brain softened and breaking down by its own weight, or by the slightest pressure, with myriads of minute red points (torn vessels?) on its broken surface; the choroid flexus of a dark purple hue; no effusion into the ventricles; uncoagulated blood on the under surface of the brain; the liver full of liquid blood, which poured from it on pressure like water from a saturated sponge (LEVICK). Others record the veins of the membranes of the brain greatly congested, and the brain moist, with large blood spots (BAUMLER). In Dr. Barclay's cases, the brain was healthy in all; no congestion or accumulation of blood was observable. A very small quantity of serum was effused under the base of one; but in all three the lungs were congested, even to blackness, through their entire extent; and so densely loaded were they, that complete obstruction must have taken place. There was also an accumulation of blood in the right side of the heart, and in the great vessels. The blood is always fluid (MOREHEAD). Its re-action has been recorded as faintly alkaline (LEVICK), and also as acid (H. C. WOOD). Levick, Gerhard, and H. C. Wood hold, that the most obvious, constant, and essential lesion in sunstroke is an *altered* state of the blood. That alteration indicates a loss of its life, as shown by the change in its physical properties, especially in the inability of the fibrine to coagulate, the shrunken condition of the corpuscles, their escape from the blood-vessels, forming myriads of petechial spots, and rapid putrefaction, beginning before actual death has taken place.

Cases of sunstroke occurring "in quarters" seem to die from *coma*, nducing *apnœa;* and the most common complication is undoubtedly pulmonary congestion from oppression of the *medulla oblongata*, evidences of which are found on *post-mortem* examination in the majority of fatal cases, as originally pointed out by Dr. Marcus Hill (*Indian Annals*, Vol. III., Oct., 1855), and afterwards by Mr. Longmore (l. c., Vol. VI., July, 1859, p. 396). In those cases of death by *coma*, the most striking point in the *post-mortem* appearances is the enormous congestion of the lungs; on which Dr. Parkes remarks, that although he has dissected men in a very arge number of diseases, both in India and in England, he has never een anything like the enormous congestion observed in two or three ases of this kind (*Practical Hygiene*, p. 345). Pulmonary engorgement, owever, is not always present. On the march and in the field, the unctional phenomena are chiefly of the cerebral, spinal, and sympathetic ystems, as indicated by a painful state of nervous irritability from overtimulation of intense heat. The long exposure of the eyes to the glare f the sun in camp may account for the more constant occurrence of their ongested state in cases of *sunstroke* in the field, compared with such cases ccurring in quarters.

Symptoms.—From the accounts given by these several observers it eems clear, as Dr. Barclay observes, that the symptoms of the disease are able to be greatly modified by accidental causes, and that those phenomena which are most prominent under one set of circumstances are either bsent altogether under another, or so very much less urgent as scarcely o attract observation; that the disease, in fact, varies in several important points according to the nature of the circumstances under which it occurs. The phenomena which at any time have presented themselves under the various circumstances detailed by Mr. Longmore, Dr. Barclay, and others, may be summed up as follows:—When careful observations are made, the affection seems gradually induced by protracted exposure to extreme heat in a dry and rarefied air, combined with a vitiated atmosphere from defective ventilation, or with physical exertions of an arduous character, implying excessive fatigue of extreme duration, so as to bring about great debility and weariness of the body. Sleep at last cannot be obtained, or it is greatly interrupted, and of short duration. Deterioration of the general health is thus progressive, while altered looks and loss of flesh indicate extreme exhaustion. The heat of the surface increases to an intense degree; but accurate records of the temperature of the body in cases of *sunstroke* are not yet in existence. The bowels tend to become obstinately constipated. The urine becomes copious, and the calls to pass it are frequent, or even incontinence may prevail (LONGMORE, BARCLAY). Under such circumstances those morbid phenomena intervene which are noticed in the definition. In Dr. Barclay's experience the deterioration of the health of his men in the field was progressive. For a long time before the occurrence of the first case of *sunstroke* every one had suffered more or less from "prickly heat," the severity of the affection being, as a rule, in proportion to the amount of perspiration from each individual; and when the heat of the weather became still more intense, one of the first symptoms of its producing an injurious effect was the gradual disappearance of this cutaneous eruption, the skin becoming rough and scaly, and the perspiration ceasing. In many cases the interruption of perspiration appeared to be complete—not the slightest feeling of dampness being perceptible in any part of the dress at any period of the day. On this point Dr. Simpson observed to Dr. Morehead,—"Every man seized with sunstroke, and who could answer questions, informed me that he had not perspired for a greater or less extent of time—sometimes not for days—previous to being attacked, and that he had enjoyed good health as long as he perspired; but that on the perspiration being checked, he felt dull and listless, and unable to take much exertion without making a great effort" (*Researches on Disease in India*, p. 617). The heat of the surface became at the same time much increased. The bowels became obstinately constipated. The appetite gradually failed; and a feeling of nausea was generally complained of, the sight of food often exciting loathing. In other instances there was nearly complete anorexia.

The urine became copious and limpid, and the calls to pass it frequent; so much so, that Mr. Longmore especially notices a frequent desire to micturate as constant in all the cases in which there was an opportunity of ascertaining the point; and he justly remarks—"If this symptom should prove to be a general precursor of the attack, it might be rendered valuable as an indication of the approaching danger, which, by early and proper care, might then probably be averted; and its presence at a time when heat-apoplexy was prevalent would make the surgeon alert to obviate the more serious symptoms which might be expected to follow" (*Indian Annals*, Vol. VI., 1865, p. 399). Sleep from the first was much interrupted, and the periods during which it could be obtained became gradually shorter, until at length no sleep could be got during the night. The pulse was frequent, sharp, and small. The tongue white posteriorly, but seldom foul or dry. Thirst was seldom very urgent. Vertigo was frequently complained of, but headache rarely; and, as a general rule, there was no complaint of pain. The general complaint was extreme debility, weariness, and prostration on any exertion; vertigo, nausea, and in many cases incontinence of urine, more particularly after exposure to the sun. "I cannot hold my water" being a very general answer to the first inquiries of the medical officer. These premonitory symptoms were attended with rapid and progressive emaciation. No one during the march (in the ravines of Northern Bundelcund, so well described by Dr Barclay) became the subject of *sunstroke* without having previously suffered from some or all of these premonitory symptoms. Such phenomena, however, prevailed in a much larger proportion of cases in which symptoms of *sunstroke* did not supervene at all; and they seem to stand somewhat in the same relation to *sunstroke* that the premonitory diarrhœa does to *cholera*.

The attacks of *sunstroke* came on generally when the men were in their tents, during the day sometimes, but in several instances during the night, and also on the line of march. During the night the patient had generally laid himself down, often seemingly asleep, or trying to induce sleep, when the attention of his comrades would be directed to him by his hurried and heavy breathing, and on attempting to rouse him he was found to be insensible. In other instances he started up suddenly, and attempted to escape from the tent, staggering about, and struggling violently when laid hold of, evidently much alarmed, and anxious to escape from some imaginary object of terror; but in a very few minutes he became insensible. In one or two instances the first symptom of the disease was reputed to have been an uncontrollable burst of laughter, without any apparent cause, and in sad enough circumstances,—insensibility and death speedily following. In a few instances the patient would come to hospital himself, or with the assistance of his comrades, exhibiting some of the symptoms (premonitory) already detailed, when insensibility or a tendency to sleep would gradually supervene. The time of the day at which most of the cases occurred is important in connection with the elevation of temperature. Ten out of sixteen cases occurred between the hours of 2 and 5 o'clock P.M., and five cases between 5 and 9-30 P.M. (LONGMORE). In Mr. Cotton's experience at Meerut the seizure usually happened towards evening. The thermometer generally indicated the maximum temperature of the twenty-four hours to be about 4 P.M.; and the variation in the thermometer-range was very slight from 2 P.M to sunset; and even for some hours after sunset the temperature scarcely at all declined (LONGMORE).

Dr. H. S. Swift records, in the *New York Journal of Medicine* for 1854, some interesting particulars regarding sixty cases of sunstroke observed at the New York Hospital during that year. The seizure in all was sudden, in the midst of work, and was characterised by pain in the head, a sense of fulness and oppression in the epigastrium, occasionally nausea and vomiting, general feeling of weakness, especially of the lower extremities, vertigo, dimness of vision, and insensibility. Surrounding objects appeared of uniform colour, generally blue or purple. In one case everything was red, in another green, in another white. In three cases the attacks occurred between 8 and 11 A.M., in forty cases between 11 A.M. and 4 P.M., and in seventeen cases between 4 and 9 P.M. The general result being that the majority of cases occurred during the *maximum* of the day's temperature. When the disease was fully expressed, the symptoms were constant and regular. The patient lay on his back, without sense or motion, breathing rapidly, and, as death approached, more and more noisily, from the vibration of the *uvula* and the *velum pendulum palati;* and although such *stertor* was present in most of the fatal cases, yet it never approached in degree to that which is common in true apoplexy. The eyes were *fixed, and turned slightly upwards*, becoming gradually more and more glassy, as if from the formation of a film over the cornea; the *pupils greatly contracted* (generally to the size of the head of a pin); the *conjunctivæ pinky, the colour gradually becoming deeper;* the congestion at first deep-seated, and the first symptom of it a pinky zone around the cornea, the superficial vessels afterwards becoming affected.

In Dr. Barclay's cases *the face was invariably pale;* the surface dry, harsh, and burning to the touch, far beyond what he ever felt in any other disease. This pallor of the face and surface seems to be more an attribute of the disease in the field than in quarters. The symptoms observed (and they were alike in the three cases) were—first, excessive thirst and a sense of faintness; then difficulty of breathing, stertor, coma, lividity of the face; and in one whom Dr. Russell examined, contraction of the pupil. The remainder of the cases (in which the attack was slighter, and the powers of reaction perhaps greater, or at all events sufficiently great) rallied; and the attack in them ran on into either an ephemeral or more continued form of fever. In Mr. Longmore's cases the heat of skin greatly exceeded that occurring in pneumonia, and was without parallel in his experience.

Sudden and remarkable elevations of temperature are recorded by Wunderlich. In 1867 Schneider observed a temperature above 104° Fahr. in a fatal case, two and a half hours after admission to hospital. Helbig records three similar cases. Ferber observed a temperature of 104° Fahr. in a case which recovered. Bäumler (in *Med. Times and Gazette*, August 1, 1868) observed a temperature of 109·22° Fahr. in a fatal case, one hour after admission. Levick records a temperature of 109·04° Fahr. in a man fifty-five years of age, who recovered from "heat-fever" in the Pennsylvania Hospital (*Report* for 1868), and a similar record in a man aged forty, in the same place and year. He communicates also a number of similar observations—amongst which, one by Dowler is said to have reached as high as 113° Fahr. Temperatures of 103°, 104°, and 105° Fahr. are not uncommon a few hours after the attack commences (WOODMAN). See also Dr. Levick in *American Journal of Medical Science*, Vol. XXXVII., p. 40, who publishes cases with temperatures as high as 109·04° Fahr., *ending* in *recovery*.

The *heart's* action was very rapid and sharp (BARCLAY), excited and irregular (LONGMORE), the impulse and pulsation in the carotids being perceptible to the eye from a considerable distance. The pulse was frequent and sharp, and at first moderately full, giving the idea of a thinner fluid than blood circulating beneath the finger. Frothy mucus, sometimes clear, at other times of a brown colour, was in most instances ejected from the mouth and nose for some time before death, and often in large quantity. When the disease was about to terminate fatally, the heart's action soon began to fail, the pulse to flutter, and the breathing to be irregular; and in a period varying from a few minutes to a few hours, death closed the scene. In nearly all the fatal cases there was occasional convulsive muscular movements of the extremities up to the time of death. These generally ushered in a state of complete coma, in which the patient gradually sank (Longmore).

In a large proportion of the cases, however, from the commencement of the attack to its termination in death, the patient never moved a limb, or even an eyelid; and a comparatively small number of the cases on the march from Jubbulpore to Calpee were from the first attended with convulsions. These generally began in the upper extremities, or in the muscles of the face; and in some cases they did not extend further, the patient either becoming rapidly insensible or recovering. In other instances they extended to the whole of the voluntary muscles, and were of the most violent description, ceasing frequently for from two or three to fifteen or twenty minutes, and recurring again with increased severity. In a few instances the nervous irritability seemed as much increased as in hydrophobia; and some patients appeared to be in a state analagous to *somnambulism.* Although unconscious, and incapable of understanding or of answering questions, yet the countenance indicated the greatest terror,—the eyes rolled wildly about; and a few drops of water poured on the ground near him were sufficient to throw him into the most violent convulsions, and to elicit from him screams of agony. In most of these cases the convulsions ceased some time before death. The symptoms then became identical with those which characterise the ordinary course of the disease. In a few, however, the convulsions continued to the last; and in one or two death took place when the body was still contorted with them (Barclay).

Prognosis.—The mortality from *insolatio* is equal to 42·734 per cent. (Barclay); or 43·3 per cent. (Butler); one half the cases (Swift); 560 per 1,000, as the result of the following extension of Dr. Morehead's statistics:—

Observer.	Cases treated.	Deaths.
Sir Charles Napier, 1843, June 15, Nussurpoor,	44	43
Dr. Joshua Jones, 1864, Andersonville,	53	53
Mr. Hill (collected cases),	504	259
Dr. Taylor, Gazeepore,	115	16
Mr. Longmore, Barrackpore,	16	7
Mr. Lofthouse, 14th Light Dragoons,	80	10
Dr. Simpson, 71st Regiment,	25	6
Mr. Ewing, 95th Regiment,	60	17
Field Hospital, Hansi,	29	10
	926	421

In 1877 it is stated that 235 cases of heat-apoplexy and sunstroke occurred in the army in India, of which seventy were fatal. The admission rate was 4·1. Of the 235 cases 189 occurred during the four months of May to August. It gives rise to a fatality of 12·2 per cent. in the *first* and *second* years of the European soldier's service in India (*Fourteenth Annual Report of Sanitary Commissioners with the Government of India*).

In the cases which terminate favourably a gradual remission of the urgent symptoms takes place; but the irregularity of the heart's action and oppressed breathing may persist during the next day; and if the patient has been exposed to the influence of malaria, paroxysmal febrile phenomena may supervene.

The patient cannot be considered free from danger till the skin becomes cool and moist (SIMPSON, BARCLAY); indeed, a relapse of all the worst phenomena may occur even after free perspiration and sleep has been procured. Dr. Crawford relates such a case:—An orderly being left in charge of the patient during the night, with instructions to keep a cold lotion to his head, and to call the surgeon in the event of any change occurring, no matter how slight; the surgeon, visiting the hospital at one in the morning, when summoned to see another patient then taken ill, found the orderly asleep, and, to his horror, his patient moribund,—the face swollen, of a dark livid colour, the eyes protruding from their sockets, with stertorous breathing and spasmodic twitchings of the muscles of the chest and arms. The patient died shortly afterwards.

Practically, it has been observed, as Dr. Barclay points out, that there are at least four different ways in which death may occur in cases of *sunstroke*, which are necessary to be borne in mind with regard to the line of treatment necessary to follow. (1.) The affections of the nervous system alone, more particularly those which occur during active exertion "in the sun," when the intense heat acts on the surface with the greatest power, producing at last a condition similar to severe concussion, and more or less instantaneous death by *syncope* (ALISON, CRAWFORD, BARCLAY, MOREHEAD). (2.) Death may be prolonged, when pulmonary complication may occur from destruction, more or less complete, of the pulmonary circulation, and death by *asphyxia* ensues. Or, (3.) There may occur cerebral congestion, and death by *coma*. These states may, and generally do, co-exist together, and symptoms of either may predominate. (4.) Recovering from the immediate effects of these conditions, the patient may die two or three days afterwards, a febrile attack succeeding, with serous effusion within the cranium.

Sir Joseph Fayrer has especially called attention to the sequelæ of sunstroke. Recovery may only partially occur, to be followed by relapse and death; or secondary consequences, the result of tissue change, may destroy life or impair health and intellect at a later period. Recovery may take place after being in great danger, but with damaged nerve-centres, so that such cases are quite unfit for further service, or even residence in a hot climate. Of those who do not die many are permanently injured, and remain invalids for the rest of their lives, which are frequently shortened by the changes produced. These may be due to obscure cerebral or meningeal changes which affect the sufferer in various degrees of intensity. Irritability, impaired memory, epilepsy, or epileptiform attacks, headache, mania, partial or complete paraplegia, partial or complete blindness, extreme intolerance of heat, especially of the sun's rays, rendering a person otherwise fairly healthy quite incapable of serving either in hot climates, or of enduring any exposure to the sun; or it may be gradually ending in complete fatuity, dementia, or epilepsy, perchance both; chronic meningitis with thickening of the culvarium, accounting for the intense cephalalgia; or in a lesser degree in disordered innervation and general functional derangement, which seriously compromise health. Such are the sequelæ to which Sir Joseph Fayrer calls attention in severe cases of sunstroke (*Brain*, Part VII., Oct., 1879, p. 303). When recovery is mentioned in such cases, it is merely the recovery of reaction. Complete recovery never occurs. Thus, the sequelæ of sunstroke are often very distressing, and render the patient a source of great anxiety and suffering to himself and to his friends. The less severe symptoms—those probably of the slighter forms of meningitis, or of cerebral change—occasionally pass away after protracted residence in a cold climate; they are, however, not only unfrequently the cause of suffering, but of danger to, and shortening of life, pointing to permanently disturbed, if not structurally altered cerebro-spinal centres. Seventeen cases of insanity following sunstroke, uncomplicated with insobriety, are related from the histories of Dr. Thomas B. Christie. The attack in most cases was of a violent character, the symptoms at the onset being of an inflammatory character (as shown *post-mortem* by adherent and thickened membrane), gradually settling down into imbecility. The worst form of insanity after sunstroke is of an acute inflammatory character, attacking the membranes of the brain and the grey substance; so that the mental symptoms are more clearly defined than the physical. Only in one case were the physical characters clearly marked where paralysis was permanent from the first onset of the disease. In the absence of actual mania, there may still be inability to use the brain as formerly, associated with such grave symptoms as headache, squinting and incapability of fixing attention, coupled with sleeplessness (*Brain*, l. c.).

Diagnosis.—Sir Joseph Fayrer states that under the designations of sunstroke, coup-de-soleil, heat-apoplexy, heat-asphyxia, thermic fever, ardent fever, insolation and others, are included certain pathological states, which, though differing from each other materially, are not unfrequently confounded:—(1.) There is simple syncope from exhaustion caused by heat. (2.) A condition analagous to shock, due to the action of the direct rays of a powerful sun on the brain and cord; the nerve-centres, especially the respiratory, are affected; respiration and circulation rapidly fail, and death may result; recovery is frequent (from the primary shock), though not always perfect. (3.) Overheating of the whole body, blood, and nerve-centres, either from direct exposure to the sun's rays, or more frequently, to a high temperature out of them, causing vaso-motor paralysis and intense pyrexia; respiration and circulation fail, and asphyxia follows. Recovery frequently occurs, but is often incomplete, owing to structural changes in the centres giving origin to a variety of symptoms indicative of lesions of a grave character.

Macnamara describes bright injection of the discs as occurring during the headache produced by exposure to the sun—a condition which may actually increase to papillitis when actual meningitis is developed (Gowers, l. c., p. 114).

Treatment.—Keeping in view the nature of this disease, and the various modes in which death may approach, the line of treatment may be indicated as follows:—

With regard to the class of cases in which death tends to occur suddenly from *syncope*, there is little opportunity afforded for treatment; but the measures indicated are,—removal to a cooler place, if possible, as from the engine-room or stoker's place on board steamships to the open deck; the *cold douche*, but not too prolonged; keep the surface wet and exposed to a current of air, or assiduously fanned; exclude light as far as possible; remove or loosen tight and oppressive clothing; the immediate employment of stimulants, external and internal, by the rectum as well as by the mouth, and *ammonia* to the nostrils. *Depletory measures of any kind are not to be thought of.* In the less rapidly decisive cases prompt treatment is of the greatest use, while delay is fraught with the greatest danger. The patient must be immediately stripped of his outer clothing; and, being placed in a semi-recumbent position, the cold douche is to be applied, from a height of three or four feet, over his head and along his spine and chest, his extremities being at the same time sponged over with cold water. Relaxation of the pupil is the first symptom that shows the good effect of the treatment, which may require to be repeated several times, on account of returning insensibility; but if there is any evidence of failure of the pulse, this treatment must be discontinued, and the application of cold to the head is then all that can be borne. Stimulants administered by the rectum are recommended by Dr. Levick, to counteract the tendency to prostration. In some cases in India rousing by flagellation with the sweeper's broom was had recourse to with good effect by Sir Joseph Fayrer. The hair is to be cut short as soon as possible, and a blister applied to the nape of the neck, the surface having previously been well sponged over with the *acetum lyttæ.* When the first violence of the attack is subdued, increasing confidence in the ultimate result may be indulged in so soon as vesication takes place; and in cases where insensibility recurs, after an interval of ten or twelve hours, it may be removed by the application of a second blister to the vertex; which may even be again repeated, there being no doubt as to the good effect it produces. Sir Joseph Fayrer, on the other hand, expresses a want of faith in their efficacy. A blister may also sometimes be applied along the spine in the worst cases. Stimulation by the use of the electro-galvanic current, with the moist sponges applied along the sides of the neck, chest, and epigastrium, ought also to be employed. *Sinapisms* ought generally to be applied to the extremities, and to the chest or sides.

As soon as possible after the employment of the douche, a strong purgative enema ought to be given, those of a stimulant nature being preferred. But as the enema may have to be repeated several times before any effect is produced on the bowels, it may be advisable to let the first enema be of a simple purgative character, and afterwards let it be followed up by turpentine enemata.

If cerebral congestion is indicated by the state of the eyes already described, a few leeches to the temples may relieve the congestion (LONGMORE, BARCLAY); but the prevailing opinion among medical officers of experience in the treatment of this disease is against the employment of blood-letting by venesection, even in severe cases. In all the cases which have been recorded in which it has been employed, it seems to have been generally hurtful, and to have hastened the fatal termination. In cases when it has seemed at first to give relief and to mitigate the symptoms, the improvement has been only transient, and followed by relapse into a more dangerous and fatal condition. But no absolute rule can be laid down in regard to abstraction of blood. There are cases in which it may be necessary, in order to avert suffocation; but they are the exception, and it is possible there may be more danger to life in the labouring and distended heart and the embarrassed lungs, than in the loss of a few ounces of blood; which, if it would have tided the patient over the danger, as it sometimes might do, would be the lesser evil, and as such should be chosen. Each case must be treated on its own merits (Sir JOSEPH FAYRER, l. c., p. 305).

In cases where the breathing is much oppressed, and the bronchial tubes loaded with mucus, the patient should be turned occasionally over on his face and side. In the convulsive form of the disease, where the greatest irritability of the nervous system prevails, the douche is found to be inadmissible, from the agony which it occasions. In such cases Dr. Barclay has seen great benefit from the inhalation of chloroform. After a few inspirations the convulsions for the most part ceased, and sleep was very easily induced; but in one or two instances, after a considerable interval of consciousness, febrile symptoms increased in severity, coma supervened (probably effusion having occurred within the cranium), which was followed by death. But the cases in which chloroform can be used are comparatively few; and very great care is necessary in its employment, so that the inhalation may be suspended at once, as soon as any effect is produced upon the pulse.

In the most severe forms of the disease the principle of management is to reduce as quickly as possible the blood-heat, and that before tissue changes have begun. As the hyperpyrexia is due not only to the direct operation of heat on the nerve centres, blood, and tissues, but to the fever set up by vaso-motor disturbance, those remedies are indicated which have been already mentioned as useful for the reduction of temperature (see Vol. I., p. 302). The unduly heated blood is a poison for the cardiac and vaso-motor centres tending to cause death, either by *syncope* or *asphyxia*. This is best effected in sunstroke by rubbing the body over with ice, as large as can be handled, as recommended by Dr. Levick, and as has been proved to be successful by Baümler and Dr. Woodman at the London Hospital. Tepid body baths, with cold affusion to the head and back of the neck, or general cold affusion, may be employed; also, injections of iced water. Keeping pieces of ice in the axilla until there is returning consciousness, which may not be for several hours, and, therefore, great caution is required in such use of ice, so as not to induce gangrene of the skin, or continued depression, as indicated by a thermometer in the mouth, axilla, or rectum.

The sequelæ of sunstroke are, generally, persistent headache, fixed or shifting pain in the back, choreic movements of the forearm and hand, convulsive disorders, mental weakness. These symptoms are suggestive of hæmatoma of the dura mater, as a probable lesion in such cases, and indicate rest, attention to the functions of the skin, and change of air, and removal from a hot to a cold climate. All stimulants and all excesses of diet, or errors in diet, are to be specially guarded against. When the pain is fixed and severe, long-continued counter-irritation at the nape of the neck, and a course of *iodide of potassium*, may benefit the patient; but many cases are quite unrelieved by treatment, as the records of the invaliding hospital at Netley show every year (Dr. MACLEAN).

Measures for the Prevention of Sunstroke.—(1.) If a march is to be undertaken during hot weather in India, all weak and sickly men should be weeded out and left behind. (2.) The costume should be suitable for the early morning hours before sunrise, as well as for the scorching period which follows, when the men are for the most part in their tents. It should consist of materials of slow conducting power, of a colour by which heat is not readily absorbed, and its fitting should be as loose and light as possible. A flannel shirt should be worn, to prevent exposure to sudden chills, and a flannel belt round the loins may be worn with advantage, except in the hottest weather. The shirt-collar should either be open, or made so wide as to prevent all risk of its pressing injuriously on the veins of the neck. Above all, some form of knapsack should be devised other than that hitherto in use. The authorities, strongly urged by the late Dr. Parkes, have adopted a valise equipment, which is a great improvement on the older knapsack, and Surgeon-Major W. S. Oliver has devised another, which has also been favourably reported upon. Both of them do away with the use of crossbelts, so injurious to the functions of the organs within the chest. The march ought not to be commenced too early in the morning. The troops should be on the *new* ground about an hour after sunrise, and the pace should never exceed three and a half miles an hour. There should be a halt for seven or eight minutes every hour, or oftener, if the men are exhausted; and a longer halt half way, when each man should have a cup of coffee and a biscuit. They ought also to have their ration before starting in the morning. An ample supply of water should be provided for the men by "*bheesties*" attached to each company, and always compelled to march with it. No man should be allowed to fall out without being accompanied by a non-commissioned officer, who should bring him to a medical officer at once if sick, and if not, he should bring him up to the column at the halt. All men so falling out should be brought up to hospital tents for examination immediately on the arrival of the regiment in camp. The men should carry nothing on the march but their rifles and ammunition—the quantity of the latter being kept as small as is consistent with safety. They should be allowed to march "easy" and loosely clad, more particularly in passing through jungles or ravines. No halt should ever take place on such ground when better may be had within a moderate distance. When the sun is up, halts should be so timed that shelter may be obtained by open *topes.* Camps should be formed on as high and open ground as possible. The sites for camps in India marked out by the "official" pillars, are generally the most objectionable that could have been selected (BARCLAY, INDIAN SANITARY COMMISSIONERS, and others). As much space should be allowed between the tents as the ground will admit of. Tents should be pitched as speedily as possible, camels and elephants being provided for their transport—carts never. "*Kus-kus*" mats should be kept constantly wet.

Troops in the field during the continuance of the hot winds in India should have the best description of tents that can be got for hospital purposes, and be provided with the best known means for keeping them cool. An abundant supply of water in camp is of the utmost importance, care being taken that the bags containing it are in good repair, hung up within easy reach of the men, and kept always filled with water. Sentries should be under cover during the heat of the day; and the men should be instructed, whenever they go out in the extreme heat of the sun, to put a wetted towel or thick handkerchief over the head, under the cap, and around the back of the neck and face. The men should be encouraged to take exercise during the cool of the evening; or, at all events, to leave their tents, so as to permit of their thorough ventilation; and wherever it is practicable, bathing should not be omitted. Rations of spirits ought to be discontinued in India. It may be a question as to what should be substituted instead; but all authorities are agreed that the system of "spirit rations" tends to convert young soldiers into drunkards. The sleeplessness, which sometimes is a premonitory symptom of the disease, may be best relieved by *chloral hydrate* combined with *bromide of potassium,* followed by a purge.

ATHETOSIS.

Definition.—*A form of spasm hitherto observed chiefly in the muscles of the hand and of the forearm, characterised by the impossibility which the patients find in keeping the fingers and toes in any desired position; and the inability to prevent a continuous action of them, which is slow, regular, and never ceasing, except when the limb is supported, or during sleep. The movements are not disorderly, like hysteria and chorea, nor tremulous, like those of paralysis agitans and various forms of sclerosis.*

Pathology.—(*a.*) *Causation.*—This disease was first named and described by Dr. Hammond, of New York, in 1871, as an independent affection; but some years before—namely, in 1853, Charcot, and in 1860, Heisse, had described phenomena analogous to those defined by Hammond. Since that time several cases have been put on record. The term *athetosis* is derived from ἀθέτος, without fixed position. Dr. Gowers looks upon it as a form of slow mobile spasms following hemiplegia. Unilateral numbness of the body preceded this affection in one of Dr. Hammond's cases. In a case recorded by Berger of Breslau, occurring in a man nineteen years of age, who had convulsions and hemiplegia when a year old, the paralysis was only partially recovered from; a few months later epileptic fits, *affecting the left side only,* set in, and the patient has been subject to them ever since. The involuntary rhythmical movements of athetosis were first observed about two years after the original attack of hemiplegia, and they also affected only the left side. Hemianæsthesia of the left side co-existed with the motor hemiparesis; the whole lower limb and upper arm of the left side were somewhat atrophied, but the left forearm was rather larger than the right. The temperature of the left forearm was higher than that of the right. After the muscular movements of the left arm had been controlled for a long time, the temperatures were, right, 84·9° Fahr., left, 86·9° Fahr.; but after the movements had been allowed to continue for some time, the temperature on the left side rose to 89·9° Fahr. Dr. Berger considers this conclusive proof that the muscular contractions are the cause of the increased formation of heat. The temperature in the axilla was only 0·3°, and that of the calf from 0·2° to 0·6° greater on the left than on the right side. The slight difference in temperature of the lower extremities is due to the movements being there very slight as compared with the upper limb.

Dr. W. T. Gairdner, of Glasgow, records a case, in a girl seven years of age, in whom the athetosis began when she was six, and was obscurely connected with a story of a fright received at the time. There was no previous history of convulsions or of paralysis. Syphilis in another case had been an antecedent; and four years before athetosis appeared the right internal muscle of the right eye became paralysed; a year later violent cephalalgia and nystagmus set in; still later on hemianæsthesia and loss of consciousness; and still later amaurosis of both eyes, and paresis of the extremities. In another case a slight attack of hemiplegia preceded the athetosis by five years. The disease may be looked upon as a variety of symptomatic chorea.

(*b*.) *Morbid Anatomy.*—The lesion is supposed to be of embolic origin in most cases, but never so sufficiently severe as to abolish voluntary power of movement. Dr. Gowers holds with Hammond and with Charcot, that the cerebral ganglia, and not the spinal cord, are the seat of the lesion, from clinical considerations, confirmed by the evidence of one *post-mortem* examination. A case is also recorded in which a woman, aged 32, had suffered for thirty years from the characteristic movements of athetosis, the result of a great fright. A lesion was found in the extra-ventricular nucleus of the left restiform body. It was occupied in its anterior portion by a focus, which contained in its centre a calculus of the size of a French bean. The left cerebral peduncle was smaller than the right, but the lobe was undiminished in size. The inflammatory process had not gone beyond the lenticular nucleus, and no other lesion could be traced in the brain.

Symptoms.—The disease is rare. Its name, like that of chorea, is a general term comprising varieties which are very different in point of symptoms and progress. It may be unilateral (*hemiathetosis*), or it may be double or general. A clinical study of thirty-seven cases by Dr. P. Oulmont yields the following conclusions:—(1.) Hemiathetosis consists of slow, exaggerated, involuntary movements limited to the foot and hand of one side of the body, and now and then occupying the corresponding half of the face and neck. (2.) To these movements are generally added transitory contractions, or intermittent spasms, which are simple modifications of athetotic movements—a sort of intermediate stage between the mobility of athetosis and the rigidity of post-hemiplegic contracture. They may attack all parts of the upper extremity, but in the lower extremity they rarely pass the instep. (3.) The movements are involuntary, little modified by the will, and often exaggerated by it. They persist during rest, often even during sleep, at least to the degree of fixing the limb in an abnormal position. (4.) Hemiathetosis appears nearly always on the paralysed side during the course of motor-hemiplegia. (5.) It coincides, in the great majority of cases, with more or less complete hemianæsthesia of the same side. (6.) The other symptoms which may accompany it—namely, permanent contracture, rigidity, and atrophy, with laxity of the articular ligaments, do not depend on the athetosis, but on the hemiplegia itself. Articular relaxation is specially marked. (7.) Hemiathetosis resembles hemichorea; like it, it is the symptom of a cerebral lesion, doubtless in the neighbourhood of the lesion which produces hemichorea—*i.e.*, the fibres in front and outside of the sensory bundles at the lower part of the *corona radiata* (of Reil). In cases where motor or sensory hemiplegia, or both, are absent, it may be admitted that there is such a tendency to concentration, that it attacks the fasciculi at a place where the sensory and motor bundles, united at the lower part of the internal capsule, are already dissociated. (8.) Hemiathetosis and hemichorea, distinct varieties of post-hemiplegic disorders, may be united by various forms of actual tremblings, transitional states in which the characters of both are blended. (9.) Double athetosis presents the same clinical aspects as hemiathetosis, except that the movements exist on both sides of the body. The face seems to be attacked more constantly and more severely than in the unilateral form; and although its nature is unknown, there seems to be the same relation between athetosis and hemiathetosis, as there is between chorea and hemichorea (*London Med. Record*, July 15, 1878, p. 292).

Ophthalmoscopic examination has shown, in one case, œdema of the right pupil, combined with grey peripheric degeneration and white atrophy of the left pupil.

Treatment.—Beyond improving the general health, any medicinal treatment can only be determined upon by a special study of each particular case. If syphilis has been an antecedent, its appropriate remedies must be had recourse to.

(4.) *Motor Paralysis.*—*Palsy, paralysis, weakness*, or *paresis*, are terms commonly restricted to those affections where the power of voluntary motion is lost or diminished (diminution of motility), in which the motor fibres are no longer acted upon by volition—*acinesis* (from *a*, privative, and κινησις, motion). The terms imply "the more or less complete loss of that power which the higher centres should exert over the movements of the muscles;" while the word "*paresis*" is now used to denote the slighter form of paralysis. "Muscle is the instrument and not the producer of force, the genetic factor of all muscular power being in the nervous system. Hence, damage to the nervous centres is followed by derangement of muscular function, diminished or total loss of motor power (*paralysis*) being one of the most common consequences of cerebral and spinal disease." The term *anæsthesia* implies a palsy of the nerves of sensation. The various forms of palsy, or paralysis, are rather symptoms of a lesion than specific diseases. Palsy of a part is thus a very constant symptom of structural disease of the brain or of the spinal cord, but it occasionally occurs from a diseased state of a nerve-trunk itself. It may affect one limb only (*monoplegia*), or merely a part of one, and it is also limited to the muscles of certain regions. It may be limited to the lower limbs only, or to the parts below the knee; to the arms or the forearms, as in cases of infantile paralysis, lead-poisoning, wasting palsy, and traumatic paralysis. Paralysis of the muscles of expression may also exist without any other affection; it is usually one-sided, and occasionally double, involving lesion of the *portio dura* on one or both sides. Its further distribution is implied by the terms "hemiplegia" and "paraplegia." "Hemiplegia" is the name given to a paralysis of one lateral half of the body. It usually implicates one arm, one leg, and one half of the face—the paralysis of the face being usually on the same side as that of the paralysed limbs. When the paralysis of the face is on the side opposite to that of the paralysed limbs, it is now called a "crossed" or "alternate" paralysis (as from lesions situated within the *medulla oblongata* or *pons Varolii*). "Paraplegia" is the name given to a paralysis which implicates the lower part of the body—*i.e.*, the legs, and a portion of the lower part of the trunk to various heights, accompanied by disturbances of sensibility, palsy of the bladder and rectum, and bedsores. It is the characteristic form of a spinal palsy. There are also cases of paralysis of single nerves—*e.g.*, of the oculo-motor nerves, fifth nerve, *portio dura*, and musculo-spinal nerve; also from lesion of special limited regions, such as in "bulbar" paralysis, when nerves and muscles of a system are implicated together, as the spinal accessory and hypoglossal, with impairment of the functions of deglutition; or in cases of paralysis of the deltoid; and the muscles of the ball of the thumb in muscular atrophy, and of the extensors of the forearm (excepting supinator longus) in cases of lead palsy. Paralysis may also be "general," as usually associated with certain forms of insanity, but not limited to that association. The paralysis when general is for the most part slight, and indicated by feebleness of the muscles all over the body, including also those of expression, mastication, and deglutition; and when put into action they are usually tremulous.

The following conditions give rise to paralysis of motion:—(1.) Lesion of a nerve in some part of its course destroys its power of transmitting that force which is expressed by a contraction of the muscle into which the nerve is distributed. (2.) A lesion of some part of those centres of the nervous system whence the nerve takes its origin, or with which it may be connected directly or indirectly. And, as a correlative statement, it may be written that whatever interferes materially with the conducting power of nerve-fibre, or the generating power of nerve-cell, will constitute a paralysing lesion. Poisoning of the nervous matter will operate in this way. *Chloroform*, *ether*, *opium*, the poison of *lead* and of *mercury*, applied directly to the nerve-fibre of a living animal, suspends its power of transmitting the nervous force so long as the influence of the poison lasts. Poisons formed or retained in the living body operate in a similar way, such as the retained *urinary* and *biliary* principles, as in Bright's disease; the poisons of *rheumatism*, *gout*, and probably also *syphilis*, in some of its more severe tertiary effects. Whatever, in short, impairs the natural structure of the nerve-matter, such as *inflammation*, *atrophy*, *condensation*, *softening* (spinal, as in the form of *tabes dorsalis*), *solution of continuity*, either by simply cutting the trunk of a nerve, or by the deliquescence of the nerve-fibres, as a result of disease, such as *white softening*, a *sanguineous* (*spinal apoplexy*) or *serous effusion*, *pressure on a nerve or a nervous centre*, are causes which will produce more or less complete paralysis. Of this there is abundant proof—*e.g.*, the inclusion of a nerve in a ligature, compression of a nerve by a tumor, a depressed piece of bone in fracture of the skull, or, an apoplectic clot on the exterior of the brain. (3.) Lesions within the cranium, more or less localised, as in the *general paralysis* of the insane, where the cortex, chiefly in the præfrontal lobes, is the seat of a parenchymatous inflammation; also in *hemiplegia.*

The diagnosis of the part of the motor apparatus affected by the paralysing lesion is also of importance to be determined; and also whether the paralysis is *neuropathic* or *myopathic.* In the latter the paralysis usually commences in particular muscles, and gradually spreads to others; also, atrophy precedes complete paralysis with fibrillar contractions and pain in the muscles; diminution of electrical excitability, as the volume of muscle diminishes; and then usually a local cause. In *neuropathic* paralysis it is practically of importance to determine the seat of the paralysing influence, and especially to determine the existence of—(1.) a *peripheral* paralysis; or, (2.) a *spinal* paralysis; or, (3.) a *cerebral* paralysis.

A *peripheral* paralysis is due to lesion at any point in the course of the nerves after their emergence from the brain or spinal cord to their terminations in the muscles. They are probably always paralyses of conduction. They are limited to the region supplied by one or more nerve-trunks. Anæsthesia is almost always coincidently present, and is precisely limited to the region of distribution of the mixed nerves affected. Reflex automatic and associated movements are completely absent. When spasms occur from central disease, they do not extend to the paralysed muscles. There is generally *marked* vaso-motor and trophic disturbances, and eventually the "reaction of degeneration." If no reaction takes place on electric excitation of the central segment of the nerve, while it is easily produced when the peripheral segment is excited, the paralysis is peripheral. No morbid phenomena indicating disease of spinal cord or brain are present, except as accidental complications.

A *spinal* paralysis is due to a lesion of any segment of the spinal cord. Its characteristic feature is the occurrence of a nearly uniform ascending paralysis of both sides—paraplegic. But this is not absolute proof of its spinal origin, since similar cases have been met with arising from paralysis of the *cauda equina,* or from some lesion of the brain (cerebral paraplegia). In accordance with the height which the disease has reached in the cord (lumbar, dorsal, or cervical region), the symmetrical groups of muscles belonging to the lower extremities, trunk, and upper extremities, are paralysed progressively from below upwards. Disturbances of sensibility are common, but not always equal in extent and intensity to those of mobility. They are often limited to numbness or formication in the feet, and retardation of the conduction of sensation. Pain in the back, and a sensation of constriction round the body, are of common occurrence. Disturbances in the functions of the bladder and of the sexual functions are characteristic symptoms. Reflex movements are for the most part preserved, and are often increased in intensity; but when there is uniform degeneration of the whole medulla, they are abolished. Convulsive movements proceeding from the brain (*e.g.,* epilepsy) do not extend to the paralysed parts; but muscular tension and spasms are not unfrequently seen in them. In certain forms of spinal disease (e.g., *tabes*), affections of the optic nerves and of the nerves of the muscles of the eye are extremely common. No positive conclusions can be drawn from electrical testing. Hemiplegia may occur from spinal disease—the unilateral lesion described by Brown-Séquard.

A *cerebral* paralysis, resulting from disease within the cranium, presents a great variety of symptoms, which render their characteristics more complex and their diagnosis more difficult. In ordinary well-marked cases (extravasations, embolism, tumors), *hemiplegia* occurs on the opposite side of the body, the arm being usually most affected, the face and tongue least. There may be also isolated forms of paralysis, as of particular nerves, because different motor nerves lie in close contiguity with each other. Reflex actions are almost always preserved, and frequently increased in energy. Epileptic convulsions and those caused by strychnine occur in the paralysed parts, and are often more violent there than in the healthy parts. Vaso-motor disturbances are very common; trophic disturbances are rare. Atrophy of muscles scarcely ever occur (except in paralysis of the pons). Disorders of the functions of the organs of sense, of the different cerebral nerves, and especially of the intellectual powers, are highly characteristic, as well as the not unfrequent occurrence of disturbance of speech, whether intellectual (*aphasia*) or peripheral (*alalia*). Alteration in the size of the pupils, headache, vertigo, and vomiting are also significant of cerebral origin of paralysis. Electrical investigation supplies much information. There is usually slight and uniform increase in the excitability, and a normal reaction when the disease has been of very long duration. Distinct and strongly marked diminution occurs only in paralysis resulting from disease of one of the peduncles of the brain (ERB).

The use of electricity is of importance both as a means of diagnosis and as a curative agent in paralysis. It is employed in the forms of :—

(*a.*) *Faradisation*, as when the "*induced*" or "*interrupted*" *electric* current from an *electro-magnetic* or *electro-galvanic* battery of sufficient power is passed through a healthy muscle, contractions are excited, showing that it is susceptible to the electric current, and possesses *electro-muscular contractility*. This current also causes more or less pain of a peculiar kind, not due to the contraction merely; and hence it indicates *electro-muscular sensibility*. They are usually spoken of as the "Faradic," "induced," or "interrupted currents." They are instantaneous, and pass alternately in opposite directions; and the currents may be arranged to be rapid or slow as desired. They are of momentary duration, but of high tension. It affects healthy muscles powerfully. This induced current is sometimes also called "Faradaism." The induced current (when contrasted with the continuous current), (1.) gives rise to no perceptible chemical action, but produces marked muscular contractions; (2.) It diffuses itself but little when passed in the most direct manner between any two points of the body; (3.) It does not produce warmth so much as a numbness and jarring sensations; (4.) It is felt in its full force at the first application, as it readily penetrates the skin.

(*b.*) The galvanic current is obtained from a galvanic battery or voltaic pile, which consists of a series of plates or cylinders modified in various ways.. The force of electricity obtained is called the "galvanic, voltaic, primary battery current;" also "continuous" or "constant current," which latter term implies not merely the absence of interruptions, but constancy in the strength of the current for definite purposes, although for variable periods. The galvanic current is continuous in one direction only, and is usually of comparatively low tension. "Voltaism," and not "galvanism," is the proper term for the constant current. Contrasted with the induced current, the continuous current (1.) produces constant chemical action, as shown by its decomposition of water, *hydrogen* being given off at the "*cathode*," or negative electrode, oxygen at the "*anode*," or positive. (2.) It has considerable diffusibility when applied to the body, so that during applications to the legs a flash may be produced in the retina. (3.) It produces a sensation of warmth, and in palsied limbs really increases the temperature. (4.) It becomes increasingly felt with the continuance of the application, owing to some delay in penetrating the skin. (5.) It relieves muscular fatigue (POORE). (6.) Ordinary nerve-irritability is modified by the constant current, so that it is markedly *increased* at the *cathode* (negative pole), and lessened at the *anode*. The former causes more burning and smarting.

(*c.*) *Frictional, static,* or Franklinic electricity is a form now rarely used, on account of the clumsiness of the apparatus.

In *muscular paralysis* depending on *intracranial* causes, the paralysed muscles respond more rather than less to the electric current, up to the time when they begin to degenerate. *Electro-muscular contractility* is in excess. In such cases there is generally early rigidity of the muscles and an irritative lesion of the brain. In another class of cases excitability is diminished, the muscles are flaccid, and nervous excitability is depressed. In others there is no difference on the two sides.

In *muscular paralysis* due to intra-spinal affections, injury to nerves, or traumatic causes affecting the muscles, *electro-muscular contractility* (faradic excitability) is *impaired* or *lost*, irrespective of degeneration, while the galvanic excitability of the muscles may be preserved or increased.

In *paralysis* from lead, *electro-muscular contractility* and *sensibility* are both impaired—faradic excitability being always much diminished and often entirely lost, not only where atrophy has come on, but where the bulk of the muscles is only slightly diminished. In *progressive locomotor ataxy*, muscular response is generally quite normal, although the bulk of the muscles may have decreased. In paralysis from disease of the *antero-lateral* columns of the spinal cord, *electro-muscular* contractility is either diminished or gone. In *hysterical paralysis* the electric excitability of the muscles is normal, while *electro-muscular* sensibility is nearly or totally gone. In *rheumatic paralysis, electro-muscular* contractility is normal, while sensibility is stronger in the suffering muscles than in the healthy parts. In *progressive muscular* atrophy *faradic* excitability is lessened as the bulk of the muscles are diminished. The electric appliances are also of great value in detecting *malingering,* as after injury or railway accidents, when paralysis of certain parts may be pretended to exist. Malingerers believe that no paralysed muscles ought to contract with any current, and do their best to control such contraction (BUZZARD). Faradisation will at once decide the presence or absence of peripheral paralysis.

The lesion causing paralysis may have various locations in the spinal cord as well as in the brain; such as, in the anterior roots, within or without the cord; in the large (motor) ganglion cells of the anterior cornua and their immediate offshoots; or, finally, in the paths which lead up in the antero-lateral columns to the brain. The lesion may extend through a great part of the length of the cord. It is the combination of the paralysis with other symptoms which aids in fixing an accurate localisation to the lesion. These are, especially, the presence or absence of reflex actions, secondary muscular atrophy, muscular tension and contractive changes in electrical excitability, atrophy, degeneration, and trophic disturbances. (1.) Disease of the anterior roots rarely, but of the grey anterior cornua more frequently, is indicated by a paralysis which is rapidly followed by a marked degree of atrophy, and the reaction characteristic of degeneration. (2.) An affection of the lateral columns is indicated by a paralysis with tension and contracture of muscles without atrophy. (3.) An affection of the paths which ascend to the brain, outside of the grey substance, or at least outside the ganglia of the anterior cornua, is suggested by a paralysis without loss of reflex function and without atrophy. (4.) An affection of grey substance is indicated by a paralysis with trophic disturbances. (5.) A widely diffused lesion of the anterior grey substance is suggested by a very extensive palsy with atrophy, the reaction of degeneration, and absence of reflex acts. (6.) A strictly localised affection of roots or lesion of the grey anterior cornua is indicated by paralysis in the districts of certain pairs of roots (*e.g.*, in those of the upper extremities or both crural nerves).

The usual plan of making the patient grasp the physician's hand gives an imperfect indication of impairment of muscular power, nor does it allow of any correct estimation of changes for better or for worse in the progress of palsy. To measure the strength of paralysed muscles of the upper extremity, the *dynamometer* of Mathieu, a surgical cutler of Paris, will be found a useful instrument. It consists of an elliptical steel spring, to which is attached a semicircle of gilt brass, upon which a scale is marked. An indicator, with a small cog-wheel at one end, may be moved freely around the arc of the circle by a steel arm, upon one side of which cogs are cut, fitting into those of the indicator. The lower end of this arm touches the elliptical spring, when the indicator points to zero on the scale; a brass sheath on the under side of the scale keeps this arm in place, at the same time letting it move freely (Fig. 18).

"When the dynamometer is taken into the hand and presssd, the two sides of the spring are approximated, and the steel-arm, the cogs being pushed by the lower side of the spring, turns the indicator. When pressure is taken off, the indicator does not return to 0, but remains at the point to which it has been carried by the muscular power of the patient.

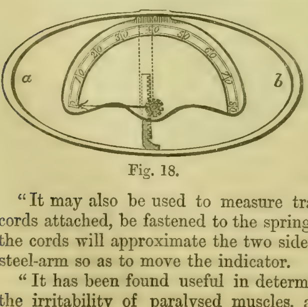

Fig. 18.

"It may also be used to measure tractile force, for if two hooks, with cords attached, be fastened to the spring at the points *a* and *b*, traction on the cords will approximate the two sides of the ellipse, and thus push the steel-arm so as to move the indicator.

"It has been found useful in determining the effect of electricity upon the irritability of paralysed muscles, and so enabling the physician to decide whether the strength of the muscles is increased or diminished after the application of this agent."

Four different conditions of the muscles are to be observed in cases of paralysis, namely,—(1.) As to *tone,* a condition little different from that of health, but less firm, less excitable by the galvanic stimulus, when the paralysing lesion is not of an irritative kind. (2.) Complete relaxation or limpness of the muscles, characterised by softness, imperfect nourishment, and rapid wasting—so rapid, that in a few days the size of the limb experiences a marked diminution. Such muscles scarcely, if at all, respond to the galvanic stimulus. This condition is usual when the paralysis is sudden in its onset, and the destroying lesion extensive. (3.) Contraction of the muscles, with rigidity and wasting (the flexors being always more rigid than the extensors)—a condition which is due to a chronic shortening of the muscles themselves, and generally associated with some irritation (inflammatory) going on at the site of the lesion. It is then acute, and comes on early. In old cases it comes on gradually with muscular atrophy. (4.) Nutrition not impaired, constant firmness and rigidity, incomplete paralysis, increased susceptibility to galvanic stimulus. Hence it is necessary to inquire as to the existence of, and whether this is associated with *tonic* or *clonic* spasm, permanent contraction of the parts (*contracture*); also as to the occurrence of movements beyond control, and to tremors.

The practical inferences to be drawn from these conditions are of great value. Thus, early rigidity indicates a lesion causing irritation of the motor ganglia; and it is especially suggestive of lesions of the cortex, including the meninges of the brain, and certain forms of spinal meningitis. Late rigidity is suggestive of changes from contraction of nervous tissue during some cicatricial process. As to *contractility,* it is usually retained, under the influence of the galvanic current, in cerebral and in ordinary spinal paralysis. In both, and especially in the latter, it is generally exalted. Hence, the paralysed muscles respond by contracting to a weak current more readily than the muscles which are healthy. When the muscles have been long diseased from paralysis they gradually cease to respond to electric stimulation; and when degeneration advances contractility is impaired in proportion to the amount of destruction of the muscles. When paralysis is due to disease involving nerve nuclei, or nerves going to the paralysed muscles, the absence of response to electric stimulation is marked and rapid; it is not unfrequently abolished in from five to ten days (BRISTOWE, l. c., p. 927).

Where contractility to the induced current remains unimpaired, we may infer that there is no serious lesion of the muscles themselves, or of the nerve trunk supplying them, or of the nervous centre at its point of junction with the nerves in question. *Abolition* or *diminution* of the contractility to the induced current occurs with great rapidity in paralysis from such serious lesions of the nerves as result from wounds or compression of the nerve trunk from tumor or exostosis; or, certain exudations (as in the cases of the osseous canal through which the facial nerve passes), which thus give rise to its compression. Caries may also lead to the destruction of a nerve. In infantile paralysis, and in other forms of spinal disease with destruction of the cord at the roots of the nerves, or of the cells connected with them in the anterior cornua, this contractility is lost or diminished, and also in paralysis from lead palsy. In all these the diminution or loss of contractility to the induced current usually exists from the very time the paralysis declares itself, thus differing from the loss of contractility which is developed slowly in connection with nerve disease or atrophy. *Preservation of contractility to galvanic current, and loss of contractility to induced current,* is commonly observed in the earlier stages of the lesions just mentioned: and it frequently happens in cases of facial paralysis especially, also in infantile paralysis and in lead palsy, that the response to the continuous current is even livelier on the paralysed than on the sound side—a reaction which is usually concurrent with degeneration of the nerve trunk or the nerve root involved. *Loss of muscular contractility to both currents* is found in advanced paralysis from any cause which has led to the destruction of muscular tissue, or to its fatty degeneration. This is especially the result in cases of old spinal paralysis with atrophy, in the last stage of pseudo-hypertrophic, muscular paralysis and wasting palsy, and also in lead paralysis. It also occurs rapidly in serious mechanical lesions of nerve-trunks from wounds and injuries (FINLAYSON, *Clinical Manual*, p. 225).

As to *nutrition* of paralysed muscles, they may retain bulk and texture, or may waste from mere disease; but the tissue remains to resume its function as soon as the paralysing lesion disappears. As to *reflex movements*, the most striking are those which occur in cases of spinal paralysis, in which some circumscribed lesion cuts off all nervous connection between the brain and the paralysed limbs, leaving the portion of the cord with which they are connected in a healthy condition. In these cases, sometimes under the influence of defæcation or micturition, sometimes from the irritation of bed-clothes, but more strikingly from touching or tickling the soles of the feet, the limbs may be made to execute violent and repeated movements (BRISTOWE, l. c., p. 929).[*]

[*] The following summary (rearranged from the writings of Professor W. H. ERB, of Heidelberg, Baden, in *Ziemssen's Cyclopædia*) may be of use as a guide to the interpretation and recognition of complicated pathological processes in the spinal cord, as it embodies the principles deduced from physiological and pathological experience :—

I. *Implication of Posterior Columns and Roots:*—(1.) Section or limited lesion of the *posterior columns* destroys the sense of touch in parts posterior to the lesion; but the sense of pain remains. (2.) Disease or destruction of the entering posterior root-fibre (or of the network of fibres directly formed by them) impairs the sense of touch equally with that of pain and the other classes of sensation. (3.) Injury or disease of the posterior column, *at the level of the lumbar cord*, leads to a diminution of the sense of touch at the anus and perineum generally; while the sensibility and motility of the lower extremities remain unimpaired. (4.) Destruction of the entire extent of the posterior columns (inclusive of the sensitive root-fibres passing through them) must be followed by anæsthesia of a corresponding extent. (5.) An *irritation* affecting a limited *longitudinal extent* of the posterior columns (inflammatory hyperæmia) produces a spontaneous pain in only those roots which traverse the diseased spot (*girdle pain*). Subjective sensations of touch (formication, prickling, numbness, sensation of heat and cold), and some degree of hyperæsthesia, occur in the parts situated posteriorly. (6.) *A lesion producing paralysis* affecting the posterior columns, in the same way gives rise to a girdle of complete insensibility corresponding to the district supplied by the paralysed nerve roots. Below this girdle the so-called sensations of touch are absent, or greatly impaired; the sensation of pain is retained, but is badly localised. (7.) If an affection which at first *irritates*, and afterwards *paralyses*, progresses upwards, the painful girdle travels upwards, and leaves behind it a girdle of anæsthesia which gradually increases in width. In the parts situated behind this, Clinically, it is necessary to determine—(1.) If the paralysis is due to inflammation, or some specific lesion in the track of a nerve, within the cranium or within the spinal canal. (2.) What are the nature and extent of the lesion? (3.) Is it due to blood-poisoning, *lead, arsenic, copper, mercury,* or poison from food? (4.) Is it functional or reflex? (5.) The

the sense of touch is gone, but subjective sensations of touch (formication, numbness) may be present. (8.) When the power of movement is unimpaired, and a girdle of pain, without aberration of the sense of touch, is present, then only the nerve roots, within or without the cord, are affected. (9.) Contractures and convulsions of the lower extremities also occur in affections of the segments of the cord above the lumbar region; they are then a consequence of an implication of the posterior columns, and arise reflexly. In the same way, in disease of the posterior columns spasmodic symptoms occur in the parts situated near the head.

II. *Implication of Grey Substance:*—(10.) Disturbance of the conductive power of the grey substance for *a limited longitudinal* extent suspends the sense of pain, but the sense of touch remains (*analgesia*). (11.) When the grey substance is partially destroyed in the transverse direction, and the posterior columns are also affected, the conduction of sensory impressions is retarded, in a degree proportional to the smallness of the transverse piece of grey matter that remains. But if the conductive power of the posterior columns is retained, this retardation appears to extend only to the sensation of pain; while the conduction of the sensation of touch takes place with normal rapidity. (12.) Limited destruction of the grey substance and of the entire transverse extent of the posterior columns is followed by complete anæsthesia of the portions of the body lying posteriorly, with weakness of motion and partial paralysis. (13.) In diseases of the grey substance, and of the posterior columns, the parts behind the lesion may experience only changes in the sense of touch, without any eccentric pains. If the latter occur, they point to an implication of those nerve roots which are situated further back. (14.) Disorganisation of the entire grey substance to a considerable distance must be followed by anæsthesia and paralysis in the posterior part of the body. If the lesion is limited to one place, the sensory and motor paralysis may be partial. (15.) In proportion as the grey substance remains normal the nutrition of peripheral parts (skin, muscle, nerve, bones, joints) remains intact in the various diseases of the spinal cord.

III. *Implication of Anterior and Lateral Columns and Grey Substance:*—(16.) Disorganisation of an anterior and a lateral column, and of the greatest part of the grey substance, produces paralysis of the same side. (17.) Limited destruction of the entire transverse section of the anterior (and lateral) columns and of the grey substance is followed by complete paralysis, also analgesia, but the sense of touch remains. (18.) Affections of the antero-lateral columns and the corresponding grey substance produce contractions or convulsions *only* in the muscles immediately dependent on the diseased spot and its motor roots; but contractures of muscles supplied by the roots given off behind the affected spot are not produced.

IV. *Implication of the Antero-Lateral Columns:*—(19.) Destruction of the anterior and lateral columns in their entire transverse section (inclusive of the motor nerve roots passing through them) is followed by a paralysis of a corresponding extent. (20.) Disease of the antero-lateral columns and the kinesodic substance alone produces paralysis without lesion of sensibility. (21.) If the movements of respiration are entirely intact in an affection of the cervical cord which paralyses the extremities and trunk, then the lateral columns are not involved.

V. *Implication of Motor Ganglia and Cervical Medulla:*—(22.) Disease of the motor ganglia, into which the motor roots first enter, produces paralysis in the region of the related nerves, without disturbance of sensibility, but with trophic disturbances. (23.) Conditions of irritation in the cervical medulla will produce dilatation of the pupil; paralytic conditions contraction.

VI. *Implication of Reflex Action:*—(24.) Complete compression or division of the spinal cord exaggerates the reflex acts in the region lying posteriorly to the lesion. (25.) In limited destruction of the dorsal medulla, the reflex acts which are performed through the lumbar cord (evacuation of urine and fæces, vascular tonus, &c.) go on with very little alteration, only they can no longer be modified by the will.

VII. Generally, and in conclusion:—(26.) Slight pressure on the cord may bring on paralysis of the extensors and secondary contractures in flexion, but this is never severe. (27.) Unilateral lesion of the cord is followed by almost total paralysis and increased sensory excitability on the injured side, with very slight disturbances of motion and loss of sensibility on the opposite side.

state of the system as to nutrition, debility, anæmia, or as to the history and existence of any specific or constitutional disease or family tendency.

The different forms of paralysis of common occurrence are due—(1.) To disease of the brain or spinal cord, the result of destructive lesions, such as occur in *apoplexy, minute hæmorrhages, softening, renal disease, induration, and syphilitis*—the *epileptic* or *choreic* state; (2.) To pressure upon or injury to a nerve; (3.) To hysteria; (4.) To the influence of poisons, such as *lead, arsenic, mercury,* and some kinds of food-grains in a diseased state, as from *lathyrus sativus.*

The typical forms of such paralyses are,—

A. CEREBRAL PARALYSES, comprehending, *Paralysis of the Insane,* or *General Paralysis; Hemiplegia.*

B. MEDULLA OBLONGATA PARALYSES: *Bulbar Paralysis,* or *Glosso-labio-laryngeal Palsy* (acute and chronic).

C. SPINAL PARALYSES: *Paraplegia; Reflex Paralysis; Hemiparaplegia; Infantile Paralysis, or Acute Spinal Paralysis; Locomotor Ataxy,* or *Tabes dorsalis; Wasting Palsy,* or *Progressive Muscular Atrophy.*

D. LOCAL PARALYSES: (*a.*) *of the 3rd, 4th, and 6th oculo-motor nerves;* (*b.*) *of the 5th nerve;* (*c.*) *Facial Palsy; Paralysis of portio dura; Bell's Paralysis;* (*d.*) *Paralysis of Spinal Nerves.* The circumflex, the radial or musculo-spinal nerve and spinal accessory.

E. OTHER KINDS OF PALSY: *Paralysis from Blood-poisons; Paralysis from Lead-poisoning,* or other poisons in food or drink, or specific disease, such as *Diphtheritic Paralysis; Paralysis from Lathyrus Sativus.*

A.—*Cerebral Paralyses.*

GENERAL PARALYSIS—Syn., PARALYSIS OF THE INSANE.

LATIN EQ., *Paralysis Insanorum*—Idem valet, *Paralysis ex toto;* FRENCH EQ., *Paralysie des Aliénés*—Syn., *Paralysie Générale;* GERMAN EQ., *Algemeine Lähmung der Wahnsinnigen;* ITALIAN EQ., *Paralisi Generale de' pazzi.*

Definition.—*An incessantly progressive form of general paralysis, in which the cineritious substance of the brain is the seat of cloudy swelling of its minute elements—the ganglionic brain-cells—with lesion of minute blood-vessels and increase of connective tissue, associated with peculiar disorder of the intellect, general failure of nerve power, muscular debility, frequent blood extravasation (hæmatoma), convulsions of the nature of apoplexy and epilepsy combined, and tending to involvement of the whole brain in degeneration, softening, and atrophy, so that general and complete paralysis of body and mind results.*

Pathology.—(*a.*) *Causation.*—Conflicting statements abound regarding the cause of this disease. It may result from an injury to a previously perfectly healthy brain. Men in the prime of life are chiefly attacked, whose activity of brain-life and brain-circulation is in excess; who take a strong flesh diet, much meat and drink; who fully taste life's troubles and joys, excitements and delights; whose brain is much irritated, somatically and physically, and whose powers of resistance are weakened by hereditary taint, illness, or alcoholism (MESCHEDE). Such cases may be generalised as due to "abusive functional activity of the brain"—so that some excessive expenditure of brain-power sets the morbid process going (Dr. CRICHTON BROWNE, l. c., Vol. VI., p. 175). It is rarely met with under twenty-five, most frequently about thirty-five to forty-one and a half years (the age which just precedes the fullest brain development), and a time at which there appears to be always great nutritive excitement. It rarely occurs after sixty, and never after seventy. Males are more liable than females, in the proportion of fifty to fifteen (BLANDFORD). When it does not result from injury, the direct causes seem to be prolonged over brain-work, probably combined with sexual excesses in those who lead "a fast life." Dr. Sankey found hereditary predisposition only in 14·5 per cent. of the cases; and Dr. Wilks considers that the evidence of its being hereditary fails in this disease more often than in any other class of disorders of the intellect. This he justly considers is in favour of the view that general paralysis may affect a previously good and healthy brain. Its immediate cause is often an accident or injury; hence, probably, its greater frequency in men than in women, and also its extreme rarity in ladies of the upper class of life. A number of other cerebral diseases produces similar symptoms to those of general paralysis. These are, especially, tumors, hæmorrhages, pachymeningitis, sclerosis in patches, chronic alcoholism, and especially syphilis (LEIDESDORF). Dr. Broadbent has also published a series of cases illustrative of syphilitic affections of the brain, in two of which most of the symptoms of general paralysis, mental and bodily, were present in a very pronounced manner, but (contrary to the usual history of cases of general paralysis) the patients recovered under treatment by *iodide of potassium and biniodide of mercury.* Pathologically, we have to do with a chronic inflammatory process affecting the grey matter and meninges of the brain.

(*b.*) *Morbid Anatomy.*—Dr. Crichton Browne, while in charge of the West Riding Lunatic Asylum, examined the brains of fifty-two patients who died of general paralysis, and the following is a summary of the morbid appearances, which he fully describes (*Reports*, l. c., Vol. VI., p., 170 1876). In the early stage of the disease the whole of the brain of patients examined after death has been seen to be congested; while the cortical substance in certain districts, as well as the pia mater, was swollen and engorged. Throughout the brain, or in some districts of it, minute red spots have been seen in greater or lesser numbers; and incipient adhesions of the pia mater, but not of the usually dense character. The grey matter in these cases is of a reddish colour, and, together with the medullary substance, looks as if it had been sprinkled with wine, which has caused blotches of discolouration. The vinous tinge is most visible in the middle layer of the cineritious substance. A viscid gummy substance has also in this stage been detected, smearing or permeating the cortical substance. Later on, when the patient dies at the end of the third stage in the ordinary course of the disease, the brain with its envelopes present characteristic appearances, which are more easily recognised by the experienced than described. When they are described one by one, they cease to be distinctive, for each of them may be seen in other forms of brain disease having no alliance with general paralysis. It is in the grouping and combination of the morbid appearances that the distinctiveness consists:—(1.) The shape and consistence of the brain is abnormal. It is somewhat attenuated in the frontal region, and it lies as if it were flattened and collapsed. It has an uneven outline with lateral bulgings, due to the system of its texture, which is greatly in excess of what is ordinarily seen, and which renders it unable to preserve its proper form when deprived of the support of the cranial walls. (2.) The brain is not only softened, but it is atrophied. The volume of the entire cerebral mass is decreased; and it is bathed by a large amount of serous fluid. In no form of disease, except in the dementia of brain wasting, is atrophy of the brain so advanced as in cases of general paralysis—all the more remarkable because death usually happens at that period of life when the weight of the brain is at its greatest. (3.) The *arachnoid* is greatly thickened. In many places it has lost its transparency, and become opaque and white. Along the margins of the great median cleft, it has become a tough glistening membrane; and over the frontal and parietal lobes it is in many places cloudy and opalescent. Over the temporo-sphenoidal lobes and cerebellum scattered patches of opacity are generally to be noticed. Over the occipital lobes it retains its natural aspect. These opaque patches are best seen over the sulci, and not on the summits of the gyri. Similar white opacities, in lines or belts, may also be noticed running parallel to vessels on each side of them, conforming to all their sinuosities. (4.) The *pia mater* is remarkably thickened. It is coarse and tenacious, having a swollen and somewhat gelatinous appearance, due to the infiltration of its nerves with the serous fluid. It is, at the same time, irregularly hyperæmic, presenting reticulations of dilated vessels amidst its œdematous expanse, with more or less injection. These changes in the pia mater correspond with those in the arachnoid; they do not extend to the occipital lobe, and are most marked over the frontal and parietal lobes. (5.) The convolutions, as seen through the membranes, are much atrophied—the wasting being most advanced in the frontal and parietal lobes, to some extent in the upper part of the temporo-sphenoidal lobes, but not so in the occipital lobes or orbital lobules; and where the wasting is very far advanced much œdematous effusion, diffused throughout the meshes of the pia mater, is collected in hollows and channels. These outward appearances of the brain in general paralysis are sufficient to the practised eye to reveal the nature of the disease. But there are other appearances in the outer coverings, and in the texture of the brain, that are also characteristic of the disease. (6.) The skull bones are generally thickened, but not dense, sometimes decidedly soft when the thickening is considerable, with more or less deep grooves for the meningeal vessels. (7.) The dura mater is thickened and adherent to the skull, most so in the frontal region. Its inner surface is of a dirty colour throughout, and its sinuses contain dark-coloured blood and clots. There is also staining of a rusty brownish-yellow colour, often forming a distinct film, which can always be scraped off (*hæmatoma*). It is most distinct and extensive when there is an arachnoid cyst, and it may occur in any region, but it is chiefly to be seen in the temporo-sphenoidal fossa. Remains of filiform bands may also be seen, which have passed between the dura mater and the pia mater.

(8.) But of all the morbid appearances to be met with in general paralysis, the most constant and characteristic are the adhesions of the pia mater to the cortical substance of the summits of the gyri. The *pia mater* is glued to the surface of certain gyri, like a glove that has dried into an open sore; and if it is forcibly torn away, it removes some of the grey matter, and leaves a rough eroded surface beneath. The points or areas of adhesion may be few or numerous, large or small, distinct or confluent, but they are invariably confined to the summits of the gyri, and do not spread down their sides, nor occur at the bottom of the sulci. They have an irregular outline and jagged edges, and perhaps incline to an oval shape, having their long diameters parallel to that of the convolution on which they are situated. When the pia mater is stripped off where adhesions are abundant, the denuded and abraded convolutions look as if they had been gnawed and eaten away by a caterpillar. Other convolutions appear granular or porous, as if perforated by numerous minute holes. When the pia mater is removed, the wasting of the brain becomes more obvious. The gyri are seen to be attenuated and the sulci greatly enlarged. The adhesion of the pia mater to the cortical substance was first noticed and described by Bayle, in 1822, and was regarded by him as evidence of the existence during life of a chronic meningitis—a view the significance of which Dr. C. Browne thinks has since been overlooked. It is not to be set aside, as heretofore, as an accessory or inessential feature. On the contrary, these adhesions are characteristic of the disease in these respects, that,—(*a.*) They constitute the most constant pathological change, occurring in 80 per cent. of all cases of general paralysis; (*b.*) they explain the essential nature of the morbid process in that disease; (*c.*) they will, when minutely studied, explain its symptoms and progress. The adhesions are always the most plentiful and firmest in the most typical and acute cases of the disease. Adhesions of the pia mater to the cortical substance are also to be met with in the following diseases:—tubercular meningitis, encephalitis, meningitis acute and chronic, atrophy of the brain, chronic alcoholism, syphilitic cerebral degeneration. But with regard to the adhesions in these diseases, Dr. C. Browne points out the following three considerations as necessary to be borne in mind:—(*a.*) In all of them there is a combination of mental and motor symptoms to some extent resembling general paralysis; (*b.*) in all of them the morbid process from which the adhesions resulted was of an inflammatory kind; and, (*c.*) in all of them there is something connected with the adhesions which distinguishes them from those of general paralysis. The most characteristic differences between the adhesions in general paralysis and in chronic meningitis are that, in the latter there are deposits of pus in their vicinity, which do not occur in general paralysis; and that the adhesions are widely diffused in chronic meningitis, while in general paralysis they are more isolated, in small areas, and scattered. These appearances favour the view that the scattered patches and numerous isolated areas of adhesion, so constant in general paralysis, are in all probability due to inflammatory changes commencing in the cerebral substance. These changes are slow and insidious in their progress, and chronic in their character, taking months and even years to spread over the cerebrum, involving probably only one part of it at a time. These adhesions in general paralysis are confined to the anterior three-fourths of the brain, and are not to be seen on the posterior fourth. The frontal lobe is undoubtedly their favourite site. Here the adhesions are most numerous and most firm, so that when they are torn through a considerable depth of grey matter is removed, and a proportionally deep abrasion, with steep rough edges, is left behind. Adhesions especially abound over its superior external and internal aspects, and over the orbital lobule. In the marginal gyrus, where it forms the border of the great median fissure, both on its outer and inner aspect, they are generally very numerous, most so at its anterior extremity. In the superior middle and inferior frontal gyri (Figs. 3, 4, pp. 19, 20, *ante*), they are also numerous and extensive. The anterior of the extremities of these gyri, forming the tops of the frontal lobes, constitute the areas where adhesions in cases of general paralysis are most invariably found, and are of most marked character. In the parietal lobes, the adhesions affect all its four gyri. In the temporo-sphenoidal lobe the adhesions occur very irregularly. Both hemispheres are affected to nearly an equal extent, but generally they are more numerous over the right than left hemispheres, and a considerable degree of symmetry may be observed in their distribution.

(9.) On slicing the brain its quality is found to be generally deteriorated. The grey matter has a sallow and faded appearance. It is traversed by vessels which are coarse and prominent; and in some places it has a violet or vinous tinge. It is softened and watery, as is also the medullary substance, which is of a dirty greyish-white colour, presenting an excessive number of coarse vascular points, and a varying number of blotches of vinous staining. The ventricles, abnormally capacious, contain an excessive amount of serous fluid. The choroid plexuses are œdematous, and the ependyma often granular. The basal ganglia are all more or less atrophied, and the cerebral nerves have a preternaturally white and glistening appearance.

These crude appearances in the morbid anatomy of general paralysis are more or less corroborative of its inflammatory character. The thickening of the skull bones result from the often recurring hyperæmia of the head; the thickening of the dura mater, its discolouration, and the false membranes which line it, are the vestiges of a past pachymeningitis. The torn bands passing between the dura mater and the arachnoid are the remains or shreds of lymph or blood-vessels proceeding to a false membrane, into which they break up into stellate ramifications. The thickening and opacity of the arachnoid, the thickening and œdema of the pia mater, and particularly the double streaks of whitish-yellow thickening, following the vessels, are very characteristic of a past meningitis. The general atrophy of the brain, the pappy and soft condition of its substance, the pallor of the cortex, the dilated and tortuous vessels with thickened walls, and the copious effusion of serum, are the changes to be expected in a brain that has been dilapidated by slow inflammatory encroachments.

The microscopic appearances have a similar pathological significance. These consist mainly in a parenchymatous inflammation of the cortical substance of the hemispheres, beginning in the inner layer, and spreading from thence, causing slow but progressive destruction of the ganglion brain-cells, as shown by Dr. Franz Meschede, whose valuable paper, in Virchow's *Archives* for 1865, has been translated by Dr. G. F. Blandford in the *Journal of Mental Science*, for October, 1866. These cell changes constitute the real source of the malady. The changes in these cells correspond with the various stages and exacerbations of the disease; and it is the gradual destruction of these cells that ultimately involves the gradual abolition of mental life. In acute cases running a rapid course there are hyperæmia and parenchymatous swelling of the inner layer—a congestive turgescence and succulence—causing it to appear darker and wetter on section, such an appearance as attends the "*cloudy swelling*" of parenchymatous inflammation. (See Vol. I., pp. 65, 87.) The alteration of the cells is found in different degrees, from mere parenchymatous swelling to their reduction to molecular detritus. The inner layer of the cortical substance has a bright red look, sometimes extending to the surface, chiefly expressed in the anterior lobes, on the convexity along the longitudinal fissure, and tolerably constant in the convolution of the temporal lobes, much less on the basilar surface, and least of all on the posterior lobes. The capillary network is greatly developed, filled to excess with blood-corpuscles, sometimes with points of extravasations here and there, the vessels being tortuous and elongated. In chronic cases this inner layer appears dark, dull, and yellowish, with hardening, so that its consistence is firmer than in the normal state (*sclerosis*). The brain cells of this layer are in different stages of degeneration: at first turgid and swollen, they become cloudy from aggregation of fatty molecules, which sometimes obliterate the form of the cells, till they seem mere heaps of granules. It thus really seems to be a diffuse chronic inflammation of the peripheral cortical layer of the brain ("*periencéphalitis chronica diffusa*" of Calmeil).

On the other hand, M. Luys expresses his belief that the lesions are characterised by a general hyperplasia of the woof of the neuroglia, which thus creates a condition similar to that found in cirrhosis of the liver. They start from different points indifferently—sometimes commencing in the white substance, sometimes in the cortex, and sometimes in the submeningeal regions of the spinal marrow. Hence the different forms which general paralysis may assume at its commencement (*London Med. Rec.*, Jan. 15, 1878, p. 15).

There is distinct lamination of the two widely separated layers of the *pia mater*, distension of the minute vessels, in some instances hypertrophy of their muscular coat, and the presence of a fine hyaline substance around them, with deposits of hæmatoidin, or the *débris* of extravasations. In the nerve substance there is dilatation and tortuosity of vessels, great distinctness of the hyaline sheaths, abundant deposits of pigment and hæmatoidin, enlargement or shrinking and disorganisation of cells, with accumulations of molecular *débris* round their nuclei, and particles of molecular degeneration in the white substance. These appearances are trustworthy records of former hyperæmia, long continued, often repeated, merging into inflammation of a low, non-purulent type, and ending in degeneration. An exudation subsequently glues together the *pia mater* and the tops of the gyri, and in this exudation certain changes are gradually wrought, by which it becomes vascular, and transformed from embryonic into fibrous connective tissue. In the brain substance there is increase of perivascular protoplasm, thickening of the vascular walls, increased nuclear germination, and the development of anastomotic networks of connective-tissue corpuscles, causing impaired nutrition, and more or less disintegration of the nerve elements. For practical purposes the thickening and adhesion of the *pia mater* in general paralysis may be regarded as an index of the changes in the cortex beneath, where that form of "cloudy swelling" of the minute elements of tissue takes place—in the ganglionic brain-cells of the cortical substance—to which Virchow has given the name of *parenchymatous inflammation*, the tendency of which is directly to degenerate and degrade texture, the minute cerebral capillaries being also implicated in the change.

Symptoms.—There are in general paralysis invariably certain *motor* symptoms, involving, in different degrees, and in various orders of succession, the muscles of the face, tongue, hand, arm, leg, and the like; and we have seen, from the experiments and observations of Hitzig, Ferrier, and Hughlings Jackson (p. 63, *ante*), that certain areas of the cortex of the hemispheres are shown to be centres which represent movements of the face, tongue, hand, arm, leg, and the like. Adhesions almost invariably occur over these areas in cases of general paralysis; and thus they become the legible records of a series of experiments of the most instructive kind, performed upon the human brain by disease. The nature of the morbid process is such that in its course it imitates, as it were, the experiments of the physiologist upon the brain. In travelling over the hemispheres the disease-process first stimulates and then breaks up a centre or centres; it first produces a discharging and then a destroying lesion. It shows the effects of—(1.) comparatively slow, feeble, incontinent discharge of certain tracts and areas of grey matter; of (2.) the sudden, excessive, and explosive discharge of wider tracts and areas; and of (3.) the gradual loss of functional activity of the areas and tracts which have been so irritated and discharged. General paralysis, therefore, if watched throughout its whole course, will afford peculiar facilities for ascertaining in which regions of the cerebrum certain groups of movements are most represented, and in what order the movements are grouped and arranged. Accurate records of symptoms, and accurate charts of the lesions* found after death are still much required.

The invasion is generally gradual, mental feebleness and muscular weakness being so slight as scarcely to attract notice. In a few cases, however, the attack is sudden, and preceded by intense energy of body and mind, violence of manner, and increased bodily temperature, rapidly followed by commencing *imbecility*, advancing to *dementia*, with incipient paralysis of speech and limb. The impairment of speech very much resembles that which indicates incipient alcoholic intoxication; and the mental condition, that of the effects of Indian hemp (*cannabis sativa*), taken in the form of *hashish*, "a placid, self-complacent vanity is developed, which makes the subject of it feel himself the greatest being, physically and mentally, in the universe." There is an inordinate expansion of *self-feeling*, comparable by Meschede to the pleasing mental sensations induced by wine—believed to be a characteristic result of *turgescence of the cortical substance* or *mind organ*. Whether the disease commences suddenly or gradually, one of the earliest phenomena is a feeling of weariness of the lower extremities—weariedness after little exertion. The gait also soon becomes peculiar and characteristic, as the patient walks "to and fro"—"to and fro"—without any definite object, but an expression of restlessness, which indicates the desire to continue the movements. As the disease advances, more attention is paid to the walk, so that the centre of gravity is maintained with great care, and the patient moves with caution and studied attention, looking neither to the right hand nor to the left. The step is characteristic. The foot has no elasticity of motion, but comes down flat upon the ground. The steps are shuffling and short, while the legs are thrown outwards and apart, and the patient straddles rather than walks, with a shambling and tottering gait. Perfect co-ordination of the muscles of the lips first begins to fail, indicated by slight tremulousness; and a feeling of stiffness in the lips may be complained of. As a consequence there is hesitation in articulating words, and particularly of words with labial letters. Afterwards, as the disease progresses, any attempt at speech induces convulsive movements of the corners of the lips, twitches or quivering of the upper lip, and contractions of the chin. The head at the same time is nodded shortly and sharply, as if to aid expression. The speech soon progresses in difficulty; it becomes thick, like that of a drunken man, until articulation is impossible. The face becomes devoid of all expression. It is mask-like, or like a curtain—a perfect blank of thought and feeling. If the patient is asked to put out the tongue, he involuntarily raises his hand to his head, as if to aid the effort; but the mouth is then merely opened, and if the tongue is protruded, it is done in a jerking way, and it trembles greatly. When the patient is made to stand erect, he appears to balance his weight on both legs as equally as possible. He cannot "stand at ease," and the position of the arms and hands is constrained. In sitting, the attitude is square, squat, and graceless, the head droops slightly, the thighs are held parallel, and the knees bent at a right angle, each hand resting on a knee or on the elbows of a chair (SANKEY).

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* The lesions are best seen and investigated in brains which have been preserved in *nitric acid*. By steeping the brain for a few weeks in a mixture of one part of strong nitric acid to eight or ten of water, the membranes are completely removed, while spots where adhesions had existed are left rough, eroded, and readily recognisable. It is better not to wait for the slow destruction of the *pia mater* by the acid, but to peel it off whenever it has become black and the brain substance hard. It then separates with ease, and leaves the abraded areas distinctly marked out (*West Riding Asylum Reports*, l. c., p. 203).

It sometimes happens that muscular feebleness exists for some time before mental imbecility betrays itself. This muscular feebleness has sometimes been spoken of as the "*pulse of insanity*," and when the disorder of intellect manifests itself, general paralysis is characterised by delusions of a peculiar kind, such as the possession of good fortune, great wealth, high birth, grandeur, prowess or personal excellence. The animal spirits are exuberant—there is general contentment and good humour, except when thwarted or contradicted, when an outburst of ungovernable passion is apt to be induced. The temper is extremely irritable and uncertain, while the views held for the time and expressed vary from day to day.

The delusions also are apt to be characterised by suspicion, with continual fear of assassination or injury. In some cases hallucination and illusions of sight and hearing prevail, somewhat similar to those in *delirium tremens.* When the invasion has been gradual, the intellect becomes slowly weaker and weaker; the will becomes feeble and purposeless; the memory becomes treacherous and fails, so that words are omitted in speaking and writing, or sentences are repeated. The pupils may be contracted at first, with subsequent irregularity, and the *irides* variously susceptible to light. Anæsthesia may exist, with impairment of tactile sensibility. (See example in Dr. WILKS' excellent paper in *Guy's Hospital Reports,* Vol. XVI.). The handwriting is greatly altered; needlework is clumsily done, and with much fumbling; the thread is got and held with difficulty, and often dropped. All acts of delicate manipulation requiring a keen sense of touch become impossible. The patients are apt to fall, and to be much knocked about without feeling pain, and sometimes clumsily pull the features of their face with their hands (SANKEY).

The morbid change on which the disease depends progresses slowly through the brain, and as it does not affect it all at once, the symptoms vary in different cases. When it begins in the cineritious substance, the mental powers often fail, while much bodily power remains. Thus every lunatic asylum contains many patients suffering from this disorder who can walk well, who can deal a vigorous blow with their fist, and who enter into the games of the institution, or are engaged in labour. That the guiding will may be almost absent, although the motive powers connected with the central ganglia are perfect, is sometimes remarkably shown by cases of patients who have considerable difficulty in starting to walk, but who, when once the machinery is set agoing, will continue to walk until exhausted, having scarce any power to stop. Such a case is the very opposite of one in which, from disease of the centres or spine, all power is lost, though the mind and will are intact (WILKS). The mental symptoms alone are believed by Dr. Blandford to be sufficient to denote that general paralysis exists; and he believes the defect connected with articulate speech to be in the nerve-centres, which supply volitional power, because the patient, by a strong effort, in which he forces all his nervous energy in the direction, can correct it. This, he considers, would account for the absence of unilateral symptoms, which are often absent throughout (*Journal of Mental Science,* Oct., 1866).

At first the patient has the appearance of being well nourished; the appetite and digestion are good. The body-temperature at the beginning does not vary from that of health, but tends rather to be under the normal, with a difference of about 7° Fahr. between morning and evening temperatures. It is not till after the disorder has existed for some time, and when these peculiar convulsions set in—something of the nature of epilepsy and apoplexy combined—that the temperature is materially increased, and exhibits very marked fluctuations with great daily variations. The temperature also shows a decided fall over two or three days before the occurrence of a convulsive seizure. At the same time observations as to temperature in the insane must be taken with caution, and with the knowledge that the patient may at the same time be suffering from deposit of tubercle in the lungs and other parts, to which they are greatly liable, and which would very materially elevate the temperature. This general paralysis of the insane steadily advances from bad to worse, but with occasional remissions in the symptoms; and hence it has also been named "*progressive general paresis.*"

The earliest evidence of impaired excito-motory functions consists in imperfect deglutition, the mouth being filled and the food kept there, or rolled from side to side. There is danger also of its becoming impacted in the pharynx, and so choking the patient. That reflex action is also weakened, is shown by the fact that the soles of the feet may be tickled without causing reflex muscular movements. The sensitive irritability of muscles to electricity is sensibly impaired or altogether absent. In the last stage of the disease there is a constant tendency to gather up the bed-clothes and roll them over. All instinct of decency is lost. Death generally takes place suddenly, as from meningeal apoplexy, during or after one of the convulsive attacks; or by asphyxia, when the respiratory muscles are invaded. If the patient survives such an attack—and, indeed, as these attacks of convulsions between epilepsy and apoplexy are frequent—layers of effused blood become organised; and *hematoma* of the dura mater are often met with after death in this disease (see *Hœmatoma*), and hence a *cystic hœmatoma* seen in such cases is most probably an accidental complication.

Diagnosis.—It is still an obscure disease, "uncertain as to its origin, indeterminate in its nature, and intractable under treatment." It has symptoms in common with—(1.) Wasting palsy, wasting muscular atrophy, or locomotor ataxy; (2.) Chronic, lead, and mercurial paralysis; (3.) Forms of palsy from cerebral or spinal disease; (4.) Chronic alcoholism,—all of which lead to the final production of an atrophy essentially the same as that which occurs in the later stages of *general paralysis.* It is also sometimes confounded with epilepsy and hysteria, tumors of the brain, syphilis, and encephalitis. It has been regarded by some as a special form of disorder of the intellect. The essential and characteristic symptoms distinguishing the disease from all these morbid states are, delusions regarding grandeur especially, elation of spirits, a joyous and happy state of mind, extravagant notions ("*Paralysi,*" or "*folie ambitieuse*"), constant mental exaltation. These delusions are quite different from those which obtain in chronic alcoholism, and which are just the opposite, especially constant mental depression, dread of bodily harm and tendency to suicide, a sense of shame, degradation, and timidity. Alcoholic tremor in the upper extremities is wanting in the general paralysis of the insane, and so are the usual hepatic and other visceral disturbances that attend on chronic alcoholism. The pupils are nearly always dilated from chronic alcoholism; in the insane paralytic they are irregular. But the broad distinction has to be made between this disease and other forms of disorder of the intellect. It can only be distinguished from them, and from the other diseases already mentioned, by the surroundings of the individual case. It cannot be identified generally by the symptoms nor by any group of them, independent of *post-mortem* proof of change in the grey matter. Wilks gives authentic cases of tumors of the brain causing similar symptoms; and the mental exaltation usually met with, and considered by some as pathognomonic, is not absolutely constant, neither is the condition of the pupils, so that the points of distinctive *history* are sometimes wanting or indecisive. Under the term "*general paralysis of the insane*," a very characteristic form of disease, when seen in its typical form, is no doubt recognisable; but if modified forms and early stages of the disease be included, the term is equally applicable to other affections. The disease is as tangible as Bright's disease of the kidneys, and probably much resembles it (WILKS). In many cases the diagnosis of the disease from syphilis is very difficult.

General *paralysis* is therefore a disease which has an appreciable morbid anatomy; and in consequence, when cerebral diseases are classed on a pathological basis, it comes under the domain of the ordinary physician; but since the mind suffers in a more chronic manner than in most of the other affections which are seen by such practitioners, this complaint is in practice treated chiefly by the alienist. There are, however, many reasons for drawing a line between it and other mental affections. Thus, it sometimes arises from a definite cause, such as an injury, in a person not predisposed to insanity; it runs a certain course of not many years' duration, and it may attack a brain previously sound. Dr. Wilks gives cases of the disease where the general paralysis followed—(1.) injury; (2.) dementia; (3.) where it was not attended with any exalted ideas; (4.) where it existed without mental symptoms; (5.) where maniacal symptoms accompanied the general paralysis. On the other hand, purely mental diseases are generally dynamic or functional: they depend upon some natural and inherent failing, and they show themselves by peculiarities of manner, habits, and feelings. Then he goes on to inquire whether the peculiar symptoms are sufficient to characterise the disease—many of the symptoms belong simply to an atrophy of the brain, being present even when this atrophy is from *alcoholismus*, also chronic lead and mercury poisoning, and simple old age. Hence, it must be asked, "Whether the peculiar form of delusion, which is certainly one of the most striking features in these cases, is sufficient to characterise the complaint, and whether its absence is enough to negative the diagnosis of general paralysis?" (WILKS, in *Guy's Hospital Reports*, Vol. XII., 1866, p. 207.)

The evidence, *clinical* and *post-mortem*, tends to prove that *general paralysis* of the insane differs essentialy from all other forms of disorders of the intellect; whether the symptoms during life or the appearances after death are regarded, the cases have many well-marked characters peculiar to themselves. The symptoms connected with the intellect are distinct, as are those also of the bodily functions; the mode of attack is peculiar, while in persons liable, the cause and duration of the malady all differ from other disorders of the intellect. The lesions seen after death are also peculiar. It seems to me to be a distinct species of paralysis, rather than a mere variety of disorder of the intellect, and hence it finds its place here.

Prognosis.—The average duration of the disease is about thirteen months (CALMEIL); few survive three years (ESQUIROL); and it is seldom protracted beyond four or five (WILKS). Death is the usual end; although some cases of recovery are recorded, as if by crisis, expressed by the formation of boils or abscesses (SANKEY). An authentic case is also fully reported by Dr. Domett Stone, in the *Lancet* of February 2, 1867. The patient got well, under abstinence from mental work, good diet, constant exercise, varied amusement, diversion of the mind, and a restorative drug treatment by *iron, cod-liver oil, phosphorus, nux vomica,* and the like. As a rule, however, general paralysis is intractable. It is continuously progressive to a fatal end, and may almost be regarded as a malignant disease of the grey matter of the hemispheres (BLANDFORD). Remissions in the course of the disease are recorded by Böttger and Gauster. The former describes three kinds of remissions, lasting from several months to a year:—(1.) Cases in which motor symptoms disappear, but in which the mental weakness remains. (2.) Those in which mental improvement takes place, though the paralysis persists. (3.) The rare instances in which both mental and motor symptoms disappear together. Remissions must be regarded as exceptional, occurring only in early stages. In all cases of improvement or apparent recovery there remains a weakness of intellect, in the shallowness and instability of mental impressions, frequent changes between cheerfulness and depression without adequate cause, marked alteration in the patient's original character, especially a disposition to unmitigated and purposeless lying. The patient does not appreciate his condition, and a certain shyness and disinclination to speak of the past always remain. After the remission, the renewed progress of the disease is generally ushered in by an epileptiform or apoplectic seizure, which is not unfrequently rapidly followed by death—or the patient goes through the usual stages of the disease.

It is therefore necessary to speak with reserve as to cures of cases of general paralysis, as remissions may simulate complete recovery. A favourable prognosis might, however, be made if the intermissions are of long duration; and when during their continuance the patient's nutrition improves, and to the exclusion of severe paralysis and mental weakness. The cases of cure after the use of iodide of potassium are probably cases due to syphilis.

Treatment.—Regarding the disease from the commencement as essentially a chronic form of parenchymatous inflammation, confined at first to the convexity of the fore-half of the hemispheres, and frequently tending to meningitis more or less complete, Dr. Meyer (following Jacobs of Siegburg) has adopted the method of counter-irritation in its treatment. He keeps open for some months a suppurating surface. For this purpose, the head is to be shaven of hair over the centre of the fore part of the skull-cap for a space as large as half the palm of the hand. On the central portion of this shaven space as much *tartar-emetic ointment* as will cover a shilling is to be rubbed in efficiently, and with some energy, with a linen compress or with the ball of the thumb. On a second application caution and gentleness are necessary, in order that the already loosened epidermis may not be torn away, for the deprivation of the cuticular layers causes exquisite pain. Considerable swelling very soon extends over the frontal region and the face in the course of three or four days, which may even advance as far as the back of the head and neck. By this method an eruption of pustules does not take place; but a large scab comes off in one piece by the frequent application of warm poultices, as soon as the place shows signs of swelling. As soon after the establishment of this suppurating process as the patient's strength will permit, the internal use of *iodide of potassium* is commenced. The diet must be digestible and of a highly nourishing character. The patient is to be sent out as much as possible for work in the open air, but only in the early and evening hours in the summer months. Exposure of the head to the sun or other source of heat is to be avoided.

HEMIPLEGIA.

LATIN EQ., *Hemiplegia;* FRENCH EQ., *Hémiplégie;* GERMAN EQ., *Hemiplegie*—Syn., *Halbseitige Lähmung;* ITALIAN EQ., *Emiplegia.*

Definition.—*A form of paralysis affecting one lateral half of the body.*

Pathology.—*Causation and Morbid Anatomy.*—Any morbid condition which affects any part of either of the hemispheres of the cerebrum may be attended with more or less paralysis of the opposite side of the body. When both the corpus striatum and optic thalamus on one side of the brain are involved in a destructive lesion, the result is a loss of sensation in, and of voluntary power over, the opposite side of the body and face—a hemiplegia which may be absolutely complete without any impairment whatever of the intellectual faculties. This is one of the best established facts in human pathology. To still more circumscribe the destructive lesion, it is found that lesions of the *lenticular nucleus* invariably cause hemiplegia (pp. 30 to 68, *ante*); and generally a typical hemiplegia results from lesions involving one corpus striatum and some portion of the white substance in its immediate vicinity. But while it involves the opposite side of the body, it may not involve all parts of it; and the differences in the clinical symptoms will depend on the extent and position of the lesion in and about the basal ganglia. For example, the effects of lesion of the optic thalamus on vision will vary according to the position of the lesion. We should expect hemiopia of both eyes from a lesion occupying the region of the *corpora geniculata;* total unilateral blindness of the opposite eye if the lesion is central; and if the lesion is very extensive, perhaps total loss of vision in the opposite eye, and nasal hemiopia on the same side (FERRIER, l. c., p. 246).

Damage also to the posterior part of the left third frontal convolution and its immediate neighbourhood involves, as a rule, more or less impairment of the faculty of speech; while certain irritative and destructive lesions of the cerebral cortex seem to be specially related with convulsive movements in the lower animals, and with some local and general paralysis, probably in man, when the cortical lesions are extensive. With regard to lesions in the optic thalamus producing hemiplegia, Dr. Crichton Browne's experience has convinced him that whenever the substance of one optic thalamus is broken down, or softened to any considerable extent, peripheral irritation fails to induce not only signs of sensory stimulation, but even responsive movements in the limbs subtending that thalamus. By the light of that experience he has been able, in several cases of hemiplegia, to predict with accuracy the situation in which the clot would be found after death. Whenever ordinary paralysis of one side (hemiplegia), of a pronounced character, has associated with it annulment of reflex action and of sensibility in the powerless extremities, he does not hesitate to conclude that the central lesion is localised in, or extends into, the optic thalamus; and, whenever hemiplegia coexists with unimpaired or but slightly enfeebled reflex activity and sensibility, he feels tolerably certain that the morbid change is confined to the region of the corpus striatum. The enfeeblement or abolition of reflex excitability in those cases in which the optic thalamus are implicated may affect both the upper and lower limbs, but almost invariably it is found to exist in the feet and legs to a much more marked degree than in the hands and arms. Pinching and squeezing the toes, and applying heat, cold, and electricity to them, will sometimes fail to cause the slightest movement, while the same treatment of the fingers will be tardily followed by muscular contractions (*West Riding Asylum Reports*, Vol. V., p. 231).

In further illustration of the localisation of lesion causing hemiplegia, O. Kohler and A. Pick record certain motor phenomena occurring in a man about six months after an apoplectic attack, which caused paralysis and complete anæsthesia of the right half of the body (*hemiplegia*, including the sense organs), and purposeless movements of the finger and hand on the paralysed right side. Later on there occurred violent swinging of the right arm and uncontrolled movements of the toes. These movements ceased during sleep (*Athetosis*, see p. 331, *ante*). The hand always assumed a peculiar position (super-extension and abduction of single fingers). After death, an opening at the posterior end of the left corpus striatum led into an apoplectic cyst as large as a farthing. The same was found in the external part of the optic thalamus, and it sent a prolongation outwards through the inner capsule. On vertical section through the middle of the thalamus, the apoplectic cicatrix was seen in the outer half, extending as a reddish-brown colouration through the whole breadth of the inner capsule to the nucleus lenticularis. This place lay immediately behind and bounding that which, according to Flechsig, contains the compact bundle of the fibres of the pyramids. These authors consider the lesion of the compact bundle of the fibres of the pyramids, which ascends between the optic thalamus and the posterior part of the nucleus lenticularis, to have been the cause of the pathological phenomena; and, that it is also possible that lesions of the fibres of the pyramids in other places (as the inner part of the capsule) may be followed by the same or similar results (*London Med. Record*, Feb. 15, 1880, p. 42). A case of hemiplegia and hemianæsthesia, after a fall, is recorded by Dr. Wilks, in which recovery took place, and which he considers a good example of *functional* hemiplegia arising from a blow. The age (fifty) and character of the patient (a female) removed the case from the class of hysterical affections. She had perfect hemiplegia, with aphasia, hemianæsthesia, and loss of all the special senses. So complete a paralysis is rarely seen. If injury produced such a paralysis, Dr. Wilks considers that a very large portion of the brain must have been involved in the lesion; and if this had occurred, there must in all probability have existed a perfect insensibility, and very speedy death in consequence. But "if it be conjectured that the lesion was slight, then it must be supposed that there is a spot in the brain where all the fibres of the motor tract, of the sensory tract, as well as those of all the special senses, converge, and that this particular spot was injured. So circumscribed, however, must be this locality, and when exposed to harm be so readily restored to perfect integrity, that the most complete paralysis which results from its injury may be recovered from without leaving a trace of mischief behind. It cannot, however, be thought possible that so complete a paralysis of so many functions could have arisen from an organic lesion; and, therefore, the only explanation of the phenomena seemed to Dr. Wilks to be that which he has been in the habit of offering—namely, a temporary cessation of the functionising process, or of the working of one hemisphere of the brain. This arrest of function may arise from a physical or moral shock, either of these being competent to cause a stunning of the senses, rendering the patient insensible; and in the same way, but in a lesser degree, the shock may affect half of the brain, producing only semi-inertness and insensibility" (*Brit. Med. Journal*, April 26, 1879, p. 427). Hemiplegia is also described as due to idea only (ERB).

In a case of hemiplegia affecting the right arm only, somewhat intermittent in the sense that it was at times complete, and at other times less absolute, related by M. F. Raymond, and reported by him in *Le Progrés Medical* of April 22, 1876, there was no other lesion than tubercular meningitis, circumscribed and localised on two convolutions, the anterior and posterior marginal, near the paracentral lobe (see p. 19, *et seq., ante*)—*i.e.*, in Ferrier's region of the motor centre of the arm (Dr. CRICHTON BROWNE, in *London Med. Record*, July 15, 1876).

In typical hemiplegia "the cerebral motor nerves are affected comparatively little; and their involvement increases, as a rule, from before backwards. Thus, the movements affected by the third, fourth, fifth, and sixth nerves are rarely if ever impaired, so that the motions of the eyeball on the affected side continue, for the most part, perfect. The motor root of the fifth nerve suffers, as a rule, but little; on the other hand, the *portio dura* is generally distinctly involved, but only slightly and unequally. Thus the expression of the face is usually more or less blank on the affected side, the muscular wrinkles more or less effaced, the mouth drawn to the opposite side, the eye a little more open than its fellow, and winking a little less rapidly performed. On the other hand, the muscles of the arms and leg are always chiefly affected. If the case be severe, they are alike motionless; but if there be a difference between them, it is that the leg retains a greater degree of motor power than the arm; that it is the last to be involved and the first to recover (BRISTOWE, l. c., p. 923).

The nature of the lesions which give rise to hemiplegia are of the following kinds:—(1.) *Hemiplegia* typical of a diseased brain depends on a softening clot, abscess, tumors, (such as aneurisms of arteries at the base of the brain,) or exudation, involving or compressing some considerable portion of the *corpus striatum* or *optic thalamus*, or in the immediate vicinity of those parts. Unless pressure be produced, or the fibres otherwise interfered with, paralysis does not result. The region "reaches from the *corpora striata* in the brain down the entire length of the anterior horns of grey matter of the spinal cord, and includes the *locus niger* in the *crus cerebri*, and much of the vesicular matter of the *mesocephalon* and of the *medulla oblongata*." Disease of any part of this range of structure is capable of producing paralysis; and the palsy is on the side of the body opposite the lesion. (2.) The intracranial portion of this range exercises the greatest and most extended influence in the production of voluntary movements, and the most extended and complete paralysis takes place from disease of the *intracranial portion.* (3.) In cases of cerebral disease it must be remembered that the intracranial portion of the centre of volition for the left side of the body is situated on the right side, and that for the right side is situated on the left side of the cranium, while the *intraspinal* portions maintain relatively their respective sides. These two *intracranial* portions are connected by the oblique fibres coming from the anterior pyramidal column of the *medulla oblongata,* which (crossing from right to left) decussate with similar fibres proceeding from left to right. (4.) Exudations which are the result of inflammatory or other diseased states of the membranes of the brain, have hitherto been considered to cause paralysis by pressure; but Dr. Ferrier's experiments show that destructive lesions of the cortex will produce paralysis. (5.) Morbid states which affect or destroy fibres of deeper-seated parts, such as the *crura cerebri,* or of the *cerebellum* in its crura (because a connection exists between the hemispheres of the cerebellum and the fibres of the pyramids in the *pons Varolii*) cause paralysis. (6.) The slow accession of paralysis following symptoms of irritation indicates a gradual morbid change, such as from exudations slowly taking place. (7.) An important feature in paralysis is due to the condition of the muscles, as to whether they are rigid or relaxed. (8.) Rigidity, whether supervening or occurring simultaneously with the paralysis, indicates irritative disease within the cranium. (9.) In cases where the rigid condition of the muscles does not come on till after a long period of paralysis, and after the muscles are perhaps wasted from atrophy, such a condition indicates loss of substance in the brain, and that the cicatrix is undergoing contraction. (10.) In *hemiplegia* typical of epilepsy, the lesion is transient, the palsy in general remaining only a few hours, or at most a few days, after the epileptic attack. It is termed *Epileptic Hemiplegia.* (11.) The *hemiplegia* associated with chorea occurs during acute attacks of that disease, and is termed *Choreic Hemiplegia.* (12.) The *hemiplegia* associated with hysteria is also of transient endurance. (13.) There is a form of *hemiplegia* where the morbid phenomena seems to spread from the periphery to the central parts. (14.) It may be associated also with disease of the surface—e.g., *meningitis arachnitis.* (See WILKS, *Guy's Reports,* Vol. XII.)

In all these forms of hemiplegia the paralysis is a paralysis of motion, more or less complete. In general, however, sensation is also more or less impaired. In estimating the condition of the sentient functions, the same method is to be adopted which Weber devised in comparing the sensibility of the surface of the skin in different parts of the body. It consists in ascertaining how near the sharp points of a pair of compasses may be approximated, and yet be distinctly felt as two points by the patient. (See page 139, *ante.*)

The special lesions of the brain causing hemiplegia are, chiefly—(1.) Obstruction of a principal cerebral artery by a plug of fibrine detached from an excrescence on one of the aortic or other valves of the heart,—the result of a former endocarditis (KIRKES and VIRCHOW). (2.) A coagulum formed in an artery, resulting from some altered nutrition of its wall, and connected in general with a rheumatic or other morbid state of the blood. (3.) A softened state of the brain, such as the condition known as white softening, which follows the retardation and diminution of cerebral circulation by diseased arteries, or by the complete stoppage of an artery by a plug. (4.) Hæmorrhage, induration, or tumors—*e.g.*, tubercle or cancer in the parts indicated above.

Symptoms.—It is one of the commonest forms of paralysis, and one to which the name of "*paralytic stroke*" is commonly applied. Either half of the body may be affected; and the parts which are actually involved are generally the upper and lower extremities, the muscles of mastication, and the muscles of the tongue on one side; the respiratory process is not interfered with. The paralysis may be either complete or incomplete as regards motor power. Consciousness may or may not be perfectly retained; and whether it is so or not, the patient, when seized, if standing, falls to the ground, because the power of maintaining his equilibrium is destroyed by the failure of the antagonising muscles of one-half of the body. The affected arm and leg lie as if lifeless by the side, all power of motion in them being lost. Stimulation, however, of the extremities of the sentient nerves, by slight titillation with the fingers, sometimes gives rise to active movements. The combined effect of such stimulation and the resulting movements is to cause considerable pain. These excited motions, to which the name of "*reflex action*" has been given, occur almost exclusively in the lower extremities. Other involuntary movements of the paralysed limbs occur simultaneously with the action of yawning, or result from emotions of surprise, joy, pleasure, grief, laughter, crying.

When the *fifth* nerve is implicated in the hemiplegia, the proper masticatory movements are unequal on the two sides, in consequence of paralysis of the temporal, masseter, and pterygoid muscles of the affected side; but the buccinator escapes. There is a want of force in the masseter muscles of the paralysed side; and there is therefore apt to be lateral displacement or obliquity of the inferior maxilla, either when at rest or during mastication. The mesial line between the lower incisors is thus also apt not to correspond to that in the upper jaw.

In cases of hemiplegia the temperature of the paralysed side is raised by half a degree to one degree. It is rarely normal, and never lessened. The thermometric equilibrium is restored on the cure of the paralysis. The general body-heat of hemiplegic patients is not as a rule increased, and is usually below the normal—92·5° Fahr. During the last hours of life it may rise to 96° Fahr. Well-marked paralytic atrophy is attended by lowering of temperature in the parts affected. Becquerel's discs, in connection with a galvanometer, are the best means to determine differences of body temperature in paralytic parts. They are composed of two thin plates of *copper*, soldered to a thin rod of *bismuth*, contained in a small tube of hard rubber, furnished with a handle. One disc is placed on the sound limb, and the other on the corresponding part of the paralysed limb. Both are in connection, by means of delicate silk-covered wires, with the poles of a galvanometer. If the temperature of both limbs be the same, the needle of the galvanometer remains at rest. If the heat of either limb is raised, the needle is deflected to the north or south pole, according as one or other limb has the highest temperature. A difference of less than $\frac{1}{100}$ of a degree can thus be determined.

Sometimes the *third* nerve may be paralysed (indicating lesion of the *crus cerebri*), in which case the upper eyelid drops, and there is inability to raise it, combined with outward squint (*lateral diversion*), and dilated pupil.

The protrusion of the tongue is also characteristic in hemiplegia. It is pushed out towards the side affected, and on being retracted it is drawn towards the healthy side. Imperfect articulation may exist in *hemiplegia*, and results from the palsy of the *ninth* and *fifth nerve;* and where the power of speech is wholly lost, or utterance is limited to monosyllables, the sign is not favourable, but denotes, with other symptoms, extensive lesion of the brain, superficial as well as deep. When deglutition is impaired, serious and extensive lesion of the brain, connected with the *vagus* or *glosso-pharyngeal nerve*, is denoted.

Treatment.—The object to be aimed at in the early treatment of hemiplegia is to keep down the frequency and force of the heart's action. For this purpose strict maintenance of the horizontal position is necessary; and when consciousness exists, let the mind be kept tranquil by every means. Remove any local impediment to the easy flow of blood, and let the head be slightly raised, in a room of equal temperature. Let the bowels be cleared out, so that no irritation from them may operate injuriously on the brain. *Enemata* ought to be employed; and *castor-oil* or *calomel*, with *compound jalap powder*, may effect an efficient evacuation. Food should be nutritious and easy of digestion, and not too abundant.

The gradual removal of blood may be of service, by relieving congestion of the right side of the heart, as shown by engorgement of the veins of the head and neck, and by lividity of surface (BRISTOWE, WATSON). The circumstances under which its use is *inadmissible* are thus defined by Dr. Todd:—If the patient be cold and collapsed; if the heart's action be feeble and intermittent; if there be an anæmic state; if the patient be of advanced age; if there is evidence of extensive disease of the arterial system or of the heart; or, lastly, if it can be ascertained that already a large amount of hæmorrhage has taken place into the brain;—these, singly or conjointly, are reasons why bleeding *ought* NOT *to be resorted to.* If none of these objections exist, it is to be considered whether any of the indications noticed require to be fulfilled, and whether they can be fulfilled by a local or general blood-letting. Modern investigations show that the brain is not generally in a hyperæmic state; so that it is chiefly to check or to prevent hæmorrhage that bleeding is to be resorted to in such cases. The sudden or rapid abstraction of a moderate quantity of blood, either from the arm or temple, or by skilful cupping, may check hæmorrhage, but the quantity taken should be small; and so, likewise, the quantity drawn ought to be moderate, if it is desired merely to lessen the frequency and force of the heart's action. "Generally, Dr. Todd came to the conclusion that, in cases of white softening, you are less likely to err by omitting than by adopting the practice." The rigidity of the muscles, which comes on very early, and which indicates an inflammatory process going on round the clot, is to be combated by urinary and alvine evacuants, and by counter-irritation. It is not desirable to interfere in the *later* forms of muscular rigidity. With regard to the use of expedients for promoting the restoration of the paralysed limbs to their normal condition, Dr. Todd's experience led him to write that nothing is of more decided benefit than a regulated system of exercise—active when the patient is capable of it, passive when he is not.

Any congestion of the spinal cord apt to supervene on cerebral hemiplegia must be diminished. The patient should not lie on his back, but, if possible, flat on his belly, the arms and legs being encased in flannel, and placed at a lower level than the spine. A hot douche may also be applied to the spine, the water falling from a height of at least *four* or *five* feet, through a tube *three-quarters* of an inch or *one inch* in diameter; and its temperature should be between 98° and 100° Fahr. The application should be continued two or three minutes, and continued daily for some time. *Dry-cupping* over various parts of the spine may also be of service, and so also may blisters, moxas, and cauteries (BROWN-SÉQUARD).

The most useful internal remedies are those which tend to lessen congestion—namely, *belladonna* and *ergot of rye.* The dose of *ergot* in powder may be at first *three grains twice a day,* and gradually *the dose may be increased till it reaches six grains twice a day. Belladonna* may be applied to the spine in the form of a plaster *four inches wide and six or seven inches long;* and if no amelioration of the symptoms follows in a few weeks, the *extract of belladonna* may be given in doses of *a quarter or a third of a grain twice daily;* and if after six or eight weeks of this treatment no improvement is observed, *iodide of potassium,* in doses of *five* or *six grains twice a day,* may be given in addition to the *belladonna* (Dr. BROWN-SÉQUARD). If meningitis is believed to exist along with chronic myelitis, then the *iodide of potassium* should be given from the very commencement, along with *belladonna,* or with *secale cornutum* (Dr. MERYON). It has been given in doses of x. to xviii. grains daily for two months with success, in a case where loss of feeling over the whole of one side was complete for two years (BRIQUET). Spermatorrhœa may be relieved by pressure applied over the region of the prostate (THOMAS and MERYON). Electricity must not be used, even for purposes of diagnosis, in a recent attack. It may only be used after a long period, to prevent atrophy of paralysed limbs, when the application of galvanic currents and the use of the flesh-brush are recommended. When œdema of the limbs exists, a warm bath to the part every night is of service. The bowels must be kept open; and if anodynes are required, *opium* should be avoided, and *hyoscyamus, conium,* or *Indian hemp,* should be used instead. *Iodide of potassium* ought to be taken before food in the morning, and an hour before dinner, so as to avoid its presumed decomposition by the gastric juice.

The nutrition of the spinal cord ought to be improved by the daily use of the cold douche; and sea-bathing may be of service. If symptoms of irritation do not exist, *belladonna* is not to be given; it is only in cases of congestion that it may be useful in diminishing paralysis; and the same rule applies to *ergot of rye. Strychnine* increases the amount of blood in the spinal cord. It may be employed in paralysis only when there is no sign of irritation, and ought to be avoided when there are signs of congestion or irritation (BROWN-SÉQUARD).

B.—*Medulla Oblongata Paralyses.*

BULBAR PARALYSIS—Syn., LABIO-GLOSSO-LARYNGEAL PARALYSIS; DUCHENNE'S DISEASE.*

Definition.—*A chronic and progressive affection of the bulbar neuclei, in which there is diminution and subsequent loss of the motor power of the tongue, soft palate, and lips, associated with structural changes in the central nuclei and roots of the motor nerves which supply the affected muscles. The disease progresses always more or less rapidly to a fatal termination.*

Pathology.—(*a.*) *History.*—In this country the disease is best known by the name of "*labio-glosso*-laryngeal paralysis." The shorter and better name of "bulbar paralysis," which was introduced by Wachsmuth, directs the attention at once to the seat of the lesion in the nervous system—namely, the *medulla oblongata,* from which the facial, trigeminal, glosso-pharyngeal, pneumogastric, and spinal accessory nerves take their origin, whose functions at some part or other in the course of the disease are seriously compromised. The disease was first differentiated by Trousseau in 1841; and twenty years later he and Duchenne, of Boulogne, more accurately described its clinical phenomena. Duchenne believed it to be a muscular disease; but Trousseau, from *post-mortem* examination, came to the conclusion that it was a disease of nerve roots. Since 1860 *post-mortem* examinations and careful microscopic investigations into the morbid parts have been made by Charcot, Jeffrey, Kussmaul, Kesteven, and others, after Lockhart Clarke's method; and the following account is given mainly from Kussmaul and those other authorities.

(*b.*) *Causation.*—It seems to prevail more commonly among males than females, in the proportion of two to one. It does not seem to recognise the influence of inheritance. Otherwise its causes seem to be numerous, and may be described as direct and indirect (Dr. DOWSE, *Brit. Med. Journ.,* Nov. 4, 1876, p. 580).

A. *Direct.*—(1.) Progressive interstitial neuritis; (2.) Thrombosis of medullary vessels; (3.) Hæmorrhage; (4.) Morbid growths—syphilitic or other; (5.) Vascular spasm. B. *Indirect.*—(1.) Reflex action from peripheral irritation; (2.) Inhibition from shock to central cerebral ganglia. It has not been observed under thirty years of age; but the tendency to the disease appears to increase as age goes on. It has been seen in all ranks of life; and rather more often among the better situated classes. Its origin has been assigned to exposure of the neck to cold, falls on the head, inveterate rheumatism. It has been frequently found associated with syphilis. Violent muscular exertion, with prolonged exposure to cold, is the cause assigned in one remarkable case, in which the exertion consisted in blowing two wind instruments (clarionet and bombardon) on the same night in two villages far apart from each other. In the interval a march was made of many hours in severe cold (STEIN, quoted by Professor A. KUSSMAUL).

(*c.*) *Morbid Anatomy.*—The anatomical lesion involves the centres of the

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* Not recognised by the College of Physicians.

pneumogastric, spinal accessory, hypoglossal, and *facial* nerves; and there is *atrophy of the central neuclei* or *motor roots of these nerves, sometimes extending to the anterior roots of several of the upper spinal nerves, with similar changes in the roots of the vagi and motor roots of the fifth pair, attended with more or less paralysis of limbs and incipient muscular atrophy.* Concurrent paralysis of the lips, tongue, *velum, palate,* and vocal cords, together with the associated muscular movements of deglutition, have recently been recognised as more or less capable of explanation by the close anatomical connection between the muscles supplied by the *vagus,* the *spinal accessory,* and the *lingual* nerves (see p. 74, *ante*). The *lower* rootlets of the spinal accessory nerve (forming the external branch) arise, in common with the anterior roots of the spinal nerves in the cervical and brachial region, from the anterior grey substance of the spinal cord; while the *upper* rootlets (forming the internal branch) have a totally different and a double origin—one from a special nucleus continuous with that of the pneumogastric behind the central canal, and the other from the proper nucleus of the hypoglossal, in front of the canal. Some of the fibres of the hypoglossal seem to take their origin from the spinal accessory nucleus (Lockhart Clarke, *Beale's Archiv.*, No. 3).

The recognition of the cerebral character of the affection was made by three German physicians—Barwinkel in Leipsic (1860), Wachsmuth in Dorpat, and Schulz in Vienna (1864). Theoretically they all arrived at the conclusion that Duchenne's paralysis emanated from the medulla oblongata, and subsequent minute anatomical examinations have justified the name of progressive bulbar paralysis, and established the fact, that the affection emanates from the motor grey regions of the *bulbus medulla.* It is only by a use of the microscope that the affected portions of the medulla can be recognised.*

Careful *post-mortem* examinations have thus demonstrated the basis of the disease, the central region whence it starts, and the morbid process which induces it. The lesion is always an affection of the motor nuclei of the rhombic fossa, leading to a degenerate atrophy of the ganglion cells of those nuclei; and sometimes there is a chronic myelitis going on to sclerosis. This morbid process attacked the motor nuclei at the same time as the other motor regions, particularly the anterior pyramids; only in exceptional cases the olivary bodies; but hitherto never the restiform bodies and their grey substance. In one case the nuclei exhibited pigment atrophy of the cells without change in the neuroglia, but with marked sclerosis of the pyramids. The destruction of the ganglion cells in the motor nuclei must be regarded as the essential element of the morbid process which lies at the root of the progressive bulbar paralysis. The degeneration of the ganglion cells is that of a yellow pigment atrophy. Their colour becomes darker, more yellow, often intense yellow, yellowish-brown, or reddish-yellow. They are sometimes diminished in size, and their prolongations become indistinct, or quite disappear. By the side of smaller granules large brown grains, and clusters of grains, like mulberry-shaped cells, make their appearance. In the form of myelitis tending to sclerosis, there are countless conglomerate fat granules in the fresh medullary substance, sometimes separate and sometimes in masses. In hardened preparations an increase of the neuroglia was shown by a great extension of the interspaces between the nerve-fibres. The connective substance was granular or filamentous. The walls of the vessels were thickened, and had a broad sheath of connective tissue, in which fat-granules and brilliant disciform bodies were found. Of the grey medullary masses of the medulla oblongata which correspond to the anterior columns of the spinal cord (see p. 39, *ante*), which always exhibit the lesions, the nucleus of the hypoglossus was always the one most affected by the degeneration; the number of ganglion cells in the atrophied nucleus being one-tenth or one-twelfth of what it should be. Degeneration has also been constantly found in the nuclei of the spinal accessory; as a rule, also, in the nucleus of the facial, and by Duchenne in the nucleus of the motor portion of the fifth. The olivary bodies appear only in exceptional cases to be involved in the process of degeneration. Degeneration of nuclei near the raphe is sufficient for the production of progressive paralysis of deglutition and speech. Degenerative changes similar to those occurring in the bulbus medullæ have been found in the spinal cord in cases where paralysis has invaded spinal muscular groups. This myelitic tendency to sclerosis has been found in the anterior cornua, anterior and lateral strands, together with atrophy of the ganglion cells.

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* It is not sufficient to examine fresh preparations. The parts must be hardened in *chromic acid* and chromate of potash solutions, which render the nerve substance not only hard for fine sections, but preserve the elements of the tissue, and particularly the delicate ganglion cells and their prolongations. The sections are then to be rendered transparent by Canada balsam, glycerine, very dilute solutions of soda and the like, and then some colouring materials are to be made use of which have a peculiar affinity for this or that element of the tissues without injuring the structure of the parts,—carmine, logwood, and the various analine dyes. By these means we may bring into view, fully and sharply, nerve tubes, ganglion cells, and the *neuroglia* of these nervous elements. Where degenerative myelitis has taken place with germination of connective tissue, sections are prepared in this way, by imparting a different colour to the normal and degenerated portions, so as to differentiate the healthy from the diseased structures, even to the unaided eye. The healthy portion becomes of a tawny yellow colour, the degenerate portion remains clear. If a thin section be similarly coloured with carmine, a reverse condition of things is exhibited. The clear degenerate parts appear of a dull red colour, while those already coloured by the chromic acid admit the red colour material with greater difficulty. The carmine also causes the ganglion cells and their prolongations to stand out red in relief while the nerve tubes remain uncoloured, forming a strong contrast to the red connective substance. Several weeks are required for a satisfactory hardening by means of the chromic acid solutions. Even the most skilful anatomist requires from two to three months to bring to a conclusion minute investigations of such cases of disease (Kussmaul, in *German Clin. Lect. Syden Soc.*, 1876, p. 26).

To the naked eye, the roots of the motor bulbar nerves frequently appear atrophied. Most commonly the roots of the hypoglossus were found attenuated and transformed into such fine threads that they could scarcely be discerned. Also the vagi, accessory, and facial were thinner than normal (KUSSMAUL, l. c.). The *post-mortem* evidence also shows the close alliance of progressive bulbar paralysis with progressive muscular atrophy. Glosso-pharyngeal paralysis and progressive muscular atrophy are found to be attended with the same nerve-lesions—namely, atrophy of motor roots of nerves, cranial as well as spinal; and in all patients suffering at first from glosso-pharyngeal paralysis there is a tendency for the paralysis to become general (TROUSSEAU). Gradual disappearance of the nerve-elements, and profuse germination of connective-tissue from the neurilemma, with intense fatty atrophy, are the characteristic anatomical lesions at the roots of the affected nerves. Hence, in a large number of cases, bulbar paralysis "is not unfrequently a fragment of some widely diffused nervous disease," such as a complication of disseminated sclerosis. Its supervention constitutes, almost without exception, the last stage of lateral sclerosis, and it is not uncommonly associated with progressive muscular atrophy, sometimes coming on late and sometimes constituting an early complication (BRISTOWE, l. c., p. 1005).

In some cases diffuse sclerosis of the *medulla oblongata* has been found (NIEMEYER). According to Wachsmuth the atrophy of the nerves is secondary to "a cerebral affection of the *medulla oblongata*, having its seat in the olivary bodies, and in the grey matter lying far back between the diverging lateral and posterior columns forming the floor of the fourth ventricle."

Symptoms.—The commencement can scarcely be noticed. The disease steals upon its victim imperceptibly, and reveals itself at first by a slight difficulty in articulation, subsequently and speedily followed by a difficulty in swallowing. The patients feel themselves quite well in other respects. The tongue only feels heavier. More force seems required to use it and set it in motion for speaking or eating. Both of these actions cause a sense of great fatigue. The earliest and most noticeable symptoms to the patients' friends are those which are due to palsy of the muscles of the tongue, the soft palate, and the lips, those of the larynx and pharynx becoming implicated at a later period. The tongue is first altered, and later come in turn the lips, the larynx, the pharynx, and the palate. The commencing paresis of the lips sometimes reveals itself by a sensation of slight stiffness, such as might be caused by frost. These slighter symptoms of paralysis remain in rare cases stationary for months, or even years, until they may be suddenly and rapidly developed after a violent cold or other serious febrile illness, or without any ascribable cause. On the other hand, the commencement has been sudden, as if by apoplexy, or by a sudden attack of dyspnœa. If initial symptoms are noted, they are usually—headache, pains in the neck, feelings of constriction in the throat, and of tightness of the chest—all which Professor Kussmaul regards as symptoms of excitation consequent on hyperæmia of the medulla oblongata. As the disease becomes further developed, the patient cannot blow, whistle, nor spit, nor pucker up his mouth, and a copious secretion of saliva precedes the paralysis of deglutition, and saliva runs from the mouth involuntarily. Embarrassment of speech attracts attention. The tongue seems less supple, and the utterance becomes more and more thick. The food is apt to lodge between the teeth and the cheek, the cause of this being different from that which obtains in Bell's paralysis. In such facial paralysis it is due to paralysis of the buccinator muscle; here it is due to the circumstance that the tongue being more or less paralytic, awkward, and incapable at the tip, the patient is obliged to use his fingers to remove the food from between the teeth and the cheek, and so replace it on the tongue. Pronunciation of certain words is made through the nose. If the lip articulation is the first to be lost, the vowels *o* and *u* cannot be properly sounded, on account of the deficient contractility of the *orbicularis oris* muscle; but the order of succession in which the vocal sounds are lost to the patient's alphabet is determined by the order of succession in which the muscular regions of articulation—the lips, the tongue, and the palate —are invaded by the paralysis. Later on *i* and *e* cannot be pronounced. *A* remains generally as long as power of phonation exists. When the tongue is paralysed before the lips, saliva is apt to dribble from the lips and corners of the mouth. The paralysis continuing to progress, the tongue at last lies motionless in the hollow of the mouth, behind the lower teeth. Its apex and base are equally motionless, and not a word can be articulated. The power of uttering grunting sounds alone remains. The shape of the tongue is also altered. It has sunk down in the centre, presenting a hollow in the middle line, with its edges raised. The soft palate also droops, and the tip of the uvula rests upon the tongue, and is generally callous or insensible to irritants. The first stage of deglutition thus becomes impossible. The morsels are swallowed by holding the head backwards, and facilitating their gliding down by fluids. Sometimes only a small quantity of the food gets into the œsophagus, the remainder being propelled upwards through the mouth and nostrils, and sometimes small portions of food will find their way into the larynx, causing great distress. The appetite remaining good, but swallowing being thus impossible, constant hunger aggravates the distress (TROUSSEAU).

In cases where paralysis of the lips is sufficiently extensive, the orifice of the mouth is increased in breadth by the action of the antagonistic muscles. The naso-labial furrows are deepened, and a whining aspect is given to the physiognomy. Later on the mouth hangs loosely down, the lower lip is everted downwards, and the saliva runs over it without intermission. The expression of the countenance is blank and strange. Excessive weakness of the respiratory movements is soon superadded to these already serious symptoms. The walls of the chest and the diaphragm scarcely move. If the patient be asked to blow out a candle he cannot do it. The flame will be scarcely agitated by his utmost efforts to blow upon it. Coughing is equally inefficient; so that if catarrh should supervene, there is great difficulty in expelling the increased secretion of mucus. Asphyxia is thus apt to prove fatal. The heart's action becomes abnormally rapid. General debility now makes rapid progress, and the patient rather inclines to remain in bed, sitting up with his head supported on pillows, inclined to one side sufficiently to allow of the saliva flowing away, which he is now unable to swallow. There may be symptoms of paralysis of the vagus, causing attacks of dyspnœa and syncope, which occur in the advanced stages of the disease. They are particularly likely to happen after exertion, though sometimes they occur at night when the patient is in bed. Sleep is disturbed by paroxysms of suffocation, and death is apt to ensue suddenly by cessation of the heart's action, unaccompanied by pain or noise. These attacks are found associated with great acceleration of the pulse—130 to 150 in the minute—and with the sensation of a total failure of strength (TROUSSEAU; see also WILKS in *Medical Times*, 1868, p. 281).

Diagnosis.—From the general paralysis of the insane, it is distinguished by the intellect of the patient remaining perfectly clear; and the gravity of the complaint impresses itself on the mind of the patient. The convulsive movements of the lips are also absent in general paralysis. In cases of hemiplegia the palsy is unilateral. In double facial paralysis—a rare affection—the movements of the tongue are free, and deglutition remains efficient, all the muscles of the face are paralysed, so that expression is as blank and fixed as marble, the patient laughs or cries as if from behind a mask (DUCHENNE); but in *glosso-laryngeal paralysis* the lower part of the face alone remains motionless. Groups of symptoms closely resembling those of bulbar paralysis may also be caused by effusions of blood into the *pons* or *medulla oblongata*, or by syphilitic gummata or other disease of the same part; or similar symptoms may even arise in connection with degenerative descending lesions connected with chronic forms of cerebral disease (BRISTOWE, l. c., 1005).

Prognosis is unfavourable. The disease has always proved fatal, with rare exceptions. Its progress is generally rapid and continuous, although sometimes tedious. From three to six months it may not seem to make so rapid a progress as it does later; but as soon as deglutition becomes imperfect, death is rapid. It generally kills more rapidly than progressive locomotor ataxy, because the parts affected are more essential to life; and while ataxy may be protracted over twenty years, progressive bulbar paralysis rarely lasts longer than two or three years, and often not so long.

Treatment.—Faradisation is of doubtful efficacy at the commencement, but gives relief at later periods, by temporarily restoring function to the affected muscles, to the lessening of the trouble of deglutition, and by exciting the respiratory acts.

Dr. Dowse relates one case of cure under his care (*Brit. Med. Journal*, March 11, 1876, p. 321). The bulbar paralysis came on after a severe epileptic attack. The paralysed parts were brought under the influence of galvanism, and injections of one-sixth of a grain of *strychnia*, and one-twentieth of a grain of *atropia* were daily used to the lower extremities. At first the induced current from a two-celled Stohrer's battery was several times applied to the tongue without any result; after this the continuous current from six cells of Stohrer's battery was applied, and with the best results, for the tongue was quickly protruded, and as sharply retracted. For some days, however, he had no voluntary power over it; but the use of the daily galvanic stimulus shortly ensured this, and articulation soon became perfect. It was for some time almost inaudible, and the letter R was the most inarticulate of all the consonants. With this the power of swallowing soon returned, so that he had complete co-ordinate voluntary power over those parts supplied by the bulbar nerves. The last to regain power and sensation were the lower limbs. The muscular atrophy here was so extreme, that nothing but the tibiæ and integuments appeared left; but galvanism, frictions, and injections gave excellent effects. He also took *cod-liver oil, quinine,* and *phosphorus*. Instead of his being a mute, helpless, and emaciated creature, he had complete voluntary power over every part of the body.

When initial pains in the head and neck point to the commencement of myelitic changes, Professor Kussmaul recommends the repeated application of dry-cupping glasses to the nape of the neck every five days, and in full-blooded strong persons he would order blood to be abstracted at the first cupping. Afterwards he would permit the cautious tentative use of the shower douche, and even the jet douche for strong persons. Food and drink of a stimulating kind must be avoided during the first months.

As to medicines, *nitrate of silver*, administered early, is deserving of most confidence. He also recommends galvanisation. It may be necessary to administer food through an œsophagus tube, or by nutrient enemata.

C.—*Spinal Paralyses.*

PARAPLEGIA.

LATIN EQ., *Paraplegia;* FRENCH EQ., *Paraplégie;* GERMAN EQ., *Paraplegie;* ITALIAN EQ., *Paraplegia.*

Definition.—*A form of paralysis affecting the lower parts of the body, in which both legs, and perhaps also some of the muscles of the bladder and rectum, are paralysed.*

Pathology.—*Causation and Morbid Anatomy.*—For this kind of paralysis there are many causes assigned; and the most different disorders have been comprehended under this name. The conditions which lead to it have been described, (*a.*) as Functional, and (*b.*) as Organic. (1.) Of functional conditions may be mentioned *anæmia, dyscrasiæ, hysteria, reflex irritation.* (2.) Of organic sources the following may be specified as causes:—primary disease of the cord, or secondary disease of the spine (spinal curvature); organic disease of the cord, accompanied by rapid wasting of muscles and loss of voltaic irritability; hæmorrhages into the cord or its membranes; congestion of the cord; acute and chronic myelitis; tumors; sclerosis. In all these organic affections, and in injuries of the spine from wounds and contusions, some differences in the symptoms have been observed, according to the seat of the injury. Disorganisation of the substance of the cord entails a condition of paralysis more or less extensive according to the seat and the extent of the inflammation. Every part of the body which receives its nerves from the spinal cord below or posterior to the upper level of the structural disorganisation is paralysed; consequently, when destructive *myelitis* extends through a segment of the cord at the *fifth* pair of cervical nerves, the upper extremities are paralysed, and all those parts which receive nerve-power from a lower level are paralysed too.

In dealing with the phenomena of spinal paralysis, the following fundamental physiological data must be kept in remembrance:—(1.) The spinal cord (including the medulla oblongata) is the instrument through which the power of motion is generated and expressed, and the co-ordination of movements effected; through which sensation is transmitted to the brain and to the grey cerebro-spinal matter of the cerebro-spinal centres. (2.) Complete division of the spinal cord abolishes sensation and voluntary motion in all those parts of the body supplied with spinal nerves from below the seat of injury. Any lesion of the nerve-substance which results from a disease-process may do this, if it destroys completely the nerve-matter at the seat of lesion. According to the region in which such a lesion may be situated, so are the different forms characteristic of this loss of power. The nearer the brain and *medulla oblongata*, the more immediately fatal to life. If at the junction of the cord with the *medulla* *oblongata* such an injury were to happen, immediate death would ensue, as may be seen when an animal is "*pithed*." Generally speaking, from the head downwards the parts of the body are supplied, *seriatim*, by the nerves coming off from the spinal cord; and according as the injury to the cord is situated lower and lower down, so does the paralysis affect less and less of the body from below upwards to the seat of injury or disease. If, again, the spinal cord be lacerated or divided *above* the origin of the phrenic nerves, or above the third cervical vertebra, death is the immediate consequence, the nervous influence being no longer transmitted with sufficient completeness to the diaphragm and other muscles of respiration. Petit gives two remarkable instances of this. The only son of a working man went into the shop of a neighbour, who in play raised the child from the ground by putting one hand under his chin and the other at the back of his head. The child, only six or seven years old, struggled, dislocated his head, and died immediately. There are a few cases, however, in which disease of these parts has not been immediately fatal. Thus, the *odontoid process* has been destroyed by caries, or the second cervical vertebra has been dislocated, and yet the patient has continued to live for some months, or even some years. A remarkable case of a diminished area of the occipital foramen, whence resulted great pressure on the cord, is related by Mr. Holberton in *The Medico-Chirurgical Transactions*, Vol. XXIV., p. 108. The patient lived more than two years, the most remarkable symptom being an extremely slow pulse. In these chronic cases the formation of the lesion is slow, so that the cord becomes accustomed to the gradually increasing pressure, and the respiration consequently still continues to be carried on principally, though feebly, by the muscles of the neck and shoulders, the diaphragm and intercostal muscles being more or less palsied.

In the cervical region, *below* the origin of the phrenic nerve, at the level of the fifth and sixth cervical vertebræ, when the lesion is throughout a complete segment of the cord, and above the origin of the superior intercostal nerves, breathing is performed only by the diaphragm and abdominal muscles, while the intercostal muscles cease to act, and the ribs cease to rise and fall. In this condition the patient may live a few days, seldom a week, and never a month (WATSON). The inspiration is free, but the expiration is laborious; for the intercostal and abdominal muscles are paralysed, and incapable of assisting in that process. The patient can yawn, for that is an act accompanied by inspiration; but he cannot sneeze, for that is an act accompanied by expiration. At this point, also, the upper extremities are still palsied, both as relates to motion and to sensation. When the palsy of motion and of sensation is complete, the patient, during the short remaining period of his life, presents a living head, with its sensibility and muscular powers unimpaired, attached to a trunk and extremities of whose existence he is only conscious by the sense of sight (BRODIE). The circulation of the blood is affected, and the action of the iris of both eyes, through the medium of the sympathetic nerves (MERYON). Another very common symptom connected with injuries of the upper portion of the cord is *priapism*, which may show itself about the second or third day after the accident, and generally subsides after the first fortnight. It sometimes occurs even when all sensation in the part itself is destroyed, so that the patient is not sensible of the introduction of the catheter.

If the injury be in the situation of the sixth and seventh cervical vertebræ, the palsy of motion and of sensation of the upper extremities is frequently imperfect, while it is complete in the trunk and lower extremities. If the lesion of the segment occurs below the cervical region—in the upper dorsal portion, for example—the breathing is not affected, or but slightly, while the digestive functions become impaired, and paralysis of the trunk and lower limbs is complete. Such a condition is technically called *paraplegia*. It implies palsy and loss of feeling in the lower limbs, hips, loins, and trunk, according as the injury is higher or lower in the dorsal or lumbar region of the cord. A person in this condition may live a long time, depending greatly on the seat of lesion or injury; the higher up generally the sooner fatal.

When the spinal cord has been injured in the part corresponding to the first dorsal vertebra, the upper extremities may still suffer from an incomplete palsy, either of motion or of sensation, or both. When, however, the seat of the lesion is in a line with the second dorsal vertebra, the sensation and motion of the upper extremities remain unimpaired, but the respiration is still difficult, from the palsy of the intercostal and abdominal muscles. If the paralysing influence do not extend through the entire thickness of the cord, then the lower extremities may preserve their sensation and motion, although the arms hang powerless, owing to the disease having dissected out, as it were, the groups of ganglionic cells which determine the action of certain sets of muscles, whilst the conductors of the will for the movements of the legs pass by unscathed (MERYON). An illustrative case, occurring in the practice of M. Broussais, is recorded by Ollivier. The patient was a medical student, aged twenty-one, who had, as a result of acute myelitis, complete paralysis of the upper limbs, while the legs, as well as the bladder and rectum, retained their healthy power. He died on the eighth day after the attack. There was some increased vascularity of parts of the encephalon, considerable congestion of the sinuses of the cord with fluid blood, and much sanguineous effusion between the dura mater and the vertebral arches opposite the brachial enlargement of the cord, as well as a considerable quantity of red serum between the pia mater and arachnoid at the lower part. Four minute cartilaginous laminæ were found about the centre of the dorso-lumbar enlargement; and the opposite surfaces of the arachnoid were adherent at several points over the brachial enlargement, while part of the cord, especially the grey substance, was soft for about two inches. The remaining part of the cord below was somewhat softened.

When the disease occurs in the dorsal region between the two enlargements of the cord, the respiratory muscles, which are under the influence of the dorsal spinal nerves, are frequently agitated by violent spasms, and the breathing is accomplished by short and painful efforts. If the disease extend to either enlargement, the arms or legs may participate in the spasmodic movements. But, as the work of disorganisation goes on, anæsthesia of the surface and paralysis of the muscles, above alluded to, follow in the train of symptoms;—abdominal respiration, disturbed circulation, embarrassed digestion, difficult defecation, inefficient micturition, and all the consequences of these respective functional disturbances ensue.

The symptoms, when the injury is in the lumbar region, are not dissimilar to those of the dorsal region, except that the respiration is unaffected. When the lumbar region is the seat of the disease, the sound introduced remaining in the bladder is generally covered with incrustations, and the patient also more commonly suffers from ulceration of the nates. These symptoms, perhaps, may result only in consequence of the patient surviving for a longer period than when the superior portions of the cord are affected. When the *myelitis* is limited to the lumbar enlargement of the spinal marrow, the convulsive movements occur at an early period of the disease, and cease *pari passu* with the disorganisation of the cord. For a time the electro-muscular contractility is retained; but eventually it is almost always lost. The urine generally becomes alkaline, from retention by spasmodic contraction of the sphincters of the rectum and bladder; and *priapism* not unfrequently results as a reflex action from a distended bladder—a state which soon gives place to a negative condition, ushered in by reflex spasms of the legs during defecation and micturition (Meryon, l. c., p. 35).

In chronic affections of the cord the palsied limbs usually waste, and become atrophied. In cases in which a limb has suffered from palsy, both of sensation and of motion, some singular phenomena of reflex action still remain. When a stimulus is applied to the palsied limb, it may occasion involuntary contraction of the muscles of that limb. Thus, when a feather is passed lightly over the hollow of the foot, as in tickling, convulsions occur in the limb, although the patient is quite unconscious that anything is touching his foot. These movements are quite independent of volition, and vary in extent and force inversely with the degree of voluntary power possessed by the affected limb, being most forcible when the loss of voluntary power is most complete, and diminishes gradually in extent and force as that power is increased. In some instances, by irritating one leg, movements were caused not only in that leg, but also in the other leg; and similar phenomena have been observed in decapitated animals, showing that (consistent with the anatomical observations already referred to) a portion of the cord may furnish a supply of nervous energy after disease has interrupted its connection with the brain.

In all cases where the lesion of the cord is of such a nature as to intercept the transmission of the influence of the will from the brain, convulsive movements are apt to occur in the legs, and to continue for a long time, even after the arms have become completely paralysed. These phenomena are doubtless owing to the excitement of disease reflected from the spinal marrow to the motor nerves of the lower extremities (Dr. Meryon). Similar involuntary movements may be produced artificially by tickling the soles of the feet, whose nervous connection with the brain is cut off by the destruction of a portion of the cord. The influence of the stimulus is transmitted to the spine by the incident nerves, and is reflected back by the motor nerves, thereby producing spasmodic contractions of the limb (Budd, in *Med.-Chir. Trans.*, Vol. XXII.).

Reflex paraplegia, or *reflex paralysis*, are terms first proposed and used by

Brown-Séquard. It has also been proposed to describe the phenomena as *paralysis from peripheral irritation* (JACCOUD).

The following forms of paralysis have been described as due to reflex action, namely—(1.) *Emotional paralysis.* A case of this kind is described by Dr. Meryon, in his work already referred to, p. 172. (2.) Pregnancy is apt to induce such *reflex paraplegia,* and sometimes *hemiplegia* or *amaurosis* (CHURCHILL). (3.) *Neurolytic paralysis,* in which, from no adequate cause, the functions of the cord seem suspended for a time, associated generally with exposure to cold and wet. (4.) From the *irritation of worms* in the intestinal canal (DAVAINE, MERYON); and also subsequent to *dysentery.* (5.) From the *irritation of teething* in children. (6.) *Urinary paralysis.* (7.) From *uterine disease,* as from *dysmenorrhœa,* or *metritis.* (8.) *Mechanical injury* of one part may give rise to reflex paralysis in another part; as in cases of gunshot wounds recorded by Dr. S. W. Mitchell, Morehouse, and Keen of the United States Army (War Department, Surgeon-General's Office, *Circular* No. 6). As examples of this form of paralysis, they record the following cases:—(1.) A wound involving the muscles of the right thigh is followed by reflex paralysis of the right arm and left leg; (2.) a wound of the right thigh is followed by paralysis of the right arm; and several others; but in all, however great the loss of motion or sensation at first, the power of movement and sensation began to return early, and continued to improve till the part had nearly recovered all its normal powers; but in nearly all some amount of paralysis continues permanent, and the part remains weak.

Whatever the correct interpretation of the physiological phenomena may be, it is certain that physicians and surgeons, at different times, have met with and recorded cases of paralysis which the amount of disease present in the nervous centre or its covering after death would not account for, which blood-poisoning would not account for, but which were found to be uniformly associated with injuries or disease of parts or organs remote, and not directly contiguous to the spinal marrow, and which ceased after their cure or relief. Such cases were assumed to be cases of "reflex paralysis." They have been long recognised and described by various observers. Mr. Stanley, in 1833, published a paper in *The Medico-Chirurgical Transactions,* "On Irritation of the Spinal Cord and its Nerves in connection with Disease of the Kidneys." He there records cases of paraplegia in which no morbid appearances were detected in the cerebro-spinal axis, with the exception of a moderate hyperæmia, but where inflammation of the bladder or kidneys, or gonorrhœa, had existed for some time. Similar cases of "Urinary Paraplegia" have been recorded by Romberg, Graves, Henoch, Rayer, and several other writers. Mr. Spencer Wells recorded, in *The Medical Times and Gazette,* cases of this kind in 1857. Graves' theory was, that pain, convulsion, and loss of motion may be produced by causes which act on the peripheral ends of the nerves; that such actions might be propagated to the central organs, and thence produce, by reflex irritation, similar phenomena in other parts. But the cord had not been subjected to microscopic observation; and as it has often been found that spinal cords which are much diseased appear to the naked eye perfectly normal, confirmation of this reflex theory was not established. Dr. Brown-Séquard, in a series of four lectures, published in 1861, relates the grounds of diagnosis and the principles of treatment of these various forms of paraplegia.

The views entertained by these several writers regarding the existence of a "reflex paraplegia" have been called in question by many able pathologists, and chiefly by Sir William Gull, Drs. Nasse and Valentiner. Sir William Gull is of opinion that the inflammation of the urinary passages (for example) is propagated *per continuo* to the cord; and he describes a case in which paraplegia occurred in consequence of gonorrhœa and syphilis. On *post-mortem* examination the cord appeared to be normal, but on careful microscopic examination pretty extensive fatty degeneration was found beneath the level of the sixth dorsal nerve. Remak also refers the *paraplegia urogenitalis* to a lumbo-sacral neuritis.

But the facts on which the belief in "reflex paraplegia" rests, and which show that "a paralysis of the lower limbs may be caused by some alteration in the *periphery*, or the trunk of various sensitive nerves," may be shortly stated as follows:—(1.) A very rapid cure of the paraplegia following the removal or cure of the primary disease which involved the peripheric sensitive nerves. Such rapid cures do not result in cases of paraplegia when the spinal cord or its membranes are primarily diseased. (2.) There are certain characteristics of *reflex paralysis* which tend to show how distinct it is from the cases of paralysis depending on organic alteration of the nervous centres. These characteristics are,—(*a.*) An outside excitation connected with some morbidly sensitive surface or nerve, and which exists for some time before the paralysis comes on—*e.g.*, *stricture of the urethra*, *gonorrhœa*, *disease of the kidney*, *prolapse of the womb*, *dysentery*, and the like. (*b.*) Variations in the degree of this excitation are followed by variations in the paralysis. (*c.*) The cure of the paralysis follows the subsidence of the primary disease. Cases of "reflex paralysis" are also well known to occur in all the upper parts of the body—*e.g.*, paralysis of the optic nerve is sometimes due to injury of the frontal nerve; amaurosis from the action of cold on the facial branches of the fifth; paralysis of the auditory nerve is sometimes due to neuralgia of the face; local palsy, as of the eye, the neck, the trunk, the bladder, or the rectum, is sometimes due to teething, worms, or other sources of excitement of the sensitive nerves, terminating in the mucous membranes or the skin; and the cure of amaurosis after the expulsion of worms is by no means uncommon.

Cases of paraplegia following diseases of the womb, and cured after the cure of this disease, are recorded by Romberg, Hunt, Wolf, Brown-Séquard, and others. Cases of paraplegia due to a disease of the urethra are on record by Graves, Wells, and others. Cases of paralysis consequent on inflammation of the bladder—gonorrhœal cystitis—have been related by Rayer and Brown-Séquard. Cases of reflex paralysis are also due to diseases of the prostate or kidney, to enteritis, dysentery, diphtheria, and affections of the lungs and pleura. The paralysis which attends teething has been known to increase and decrease as the molars are cut. Irritation of the nerves of the skin, generally following cold and wet, has been known to induce so-called reflex paraplegia (*neurolytic paralysis* of Handfield Jones); and so also has disease of the knee-joint. In all these instances the paralysis follows the primary disease which is the cause of the local "peripheric excitation." Increase or decrease of this excitation and of the attendant paralysis, according as the cause persists or is suppressed, is among the recognised phenomena of such cases.

There are at least two ways by which the reflex action from the outside irritation may induce paraplegia, namely—First, *the reflex contraction of blood-vessels.* Brown-Séquard has the merit of having actually demonstrated this. He has shown that reflex contractions of the blood-vessels of the medulla spinalis takes place, and that paralysis results from blood-deprivation—*paralysis from reflex ischæmia, i.e.,* from *reflex vascular contraction producing ischæmia.* The blood-vessels can contract with energy on the application of a stimulus or excitant, sometimes to the extent of a real and prolonged spasm. The nervous system is amply supplied with blood-vessels in *three* different places—namely, (1.) *In the spinal cord;* (2.) *in the motor nerves;* (3.) *in the muscles;*—and a contraction of these blood-vessels is found to cause "paraplegia." In the vessels of the pia mater of the spinal cord Dr. Brown-Séquard has shown how the vessels contract when a ligature is applied on the *hilus* of the kidney, or when a similar operation is performed on the blood-vessels and nerves of the *supra-renal* capsules. Generally, in those cases the contraction is much more evident on the side of the cord corresponding to the side of the irritated nerve. These demonstrations also coincide with other physiological evidence. More than half a century ago, Comhaire extirpated the kidney from living dogs, with the effect of instantly producing paralytic weakness of the hind leg of the side operated on. Brown-Séquard found the same result on extirpating either a kidney or a supra-renal capsule. Dr. Lewisson, of Berlin, repeated Comhaire's experiments, but without being able to confirm them. He found, however, that by pressing the kidney firmly between the fingers he induced a complete paralysis with loss of reflex irritability, which lasted as long as the pressure continued. It is, therefore, quite legitimate to conclude that irritations of these parts from disease may produce a paraplegia, by causing contractions of the blood-vessels of the cord similar to those which took place in these experiments. There are other reasons which show that such paralysis is due to reflex action and probable contraction of blood-vessels—namely, the fact familiar to surgeons, that chills, tremors, and even convulsions, are often connected with catheterism.

The present position of our knowledge of the structure and functions of the spinal cord hardly entitles us to assert, without careful microscopic examination, whether alteration of the cord does or does not exist in cases of *reflex paralysis.* The spinal cord to the naked eye may appear perfectly healthy. Under the microscope, however, marked atrophic changes in the nervous element have been found, associated with considerable germination of the connective tissue: so that many, if not all, of the fatal cases of so-called reflex paraplegia in which the cord is reported to be quite healthy, would probably give the same result if microscopic investigation of the cord had been adequately carried out (LAVERAN, YEO). In a considerable number of cases of *paraplegia urinaria,* an anatomical affection of the cord has been proven in the form of a myelitis which begins in a circumscribed lesion of the upper part of the lumbar enlargement. From this fact, and from the symptoms of lumbo-sacral neuritis which not unfrequently precede, it appears probable that the inflammation of the bladder first passes to the nerves, and induces there a progressive neuritis, which may lead to myelitis; and the same explanation applies to the other forms of paraplegia, such as after dysentery and affections of the uterus. At the same time the explanation does not exclude the possible occurrence of an actual reflex paralysis, occurring suddenly. The existence of such a true reflex paralysis is rendered probable by physiological experiment, but is not yet proven by the records of pathology; the most frequent mode of origin of these affections being the progressive inflammation of nerves, which not unfrequently passes to the spinal cord.

Paralysis due to *myelitis* may be usefully contrasted here, in a tabular form, with the leading phenomena of the two diseases, as given by Dr. Brown-Séquard in his admirable lectures *On Paralysis of the Lower Extremities;* and which, to some extent, are subscribed to by Dr. Edward Meryon.

PARAPLEGIA DUE TO

(A.) URINARY REFLEX IRRITATION.	(B.) MYELITIS.
1. *Preceded* by an affection of bladder, kidneys, or prostate.	1. Usually no disease of the urinary organs, except as a consequence of the paralysis.
2. Usually lower limbs alone paralysed.	2. Usually other parts paralysed besides the lower limbs.
3. No gradual extension of the paralysis upwards.	3. Most frequently a gradual extension of the paralysis upwards.
4. The paralysis is usually incomplete —an extreme debility or weakness of the limbs rather than paralysis.	4. Very frequently the paralysis is complete.
5. Some muscles more paralysed than others.	5. The degree of paralysis is the same in the various muscles of the lower limbs.
6. Reflex power neither much increased nor completely lost.	6. Reflex power often lost, or sometimes much increased.
7. Bladder and rectum rarely paralysed, or at least only slightly so; *sphincter ani* weak.	7. Bladder and rectum usually paralysed completely, or nearly so.
8. Spasms in paralysed muscles extremely rare.	8. Always spasms, or at least twitchings.
9. Very rarely pains in the spine, either spontaneously or on the application of pressure, percussion, or a hot moist sponge, or ice.	9. Always some degree of pain, existing spontaneously, or caused by external excitations.
10. No feeling of pain or constriction round the abdomen or the chest.	10. Usually a feeling as if a cord were tied tightly round the body at the upper limit of the paralysis.
11. No formication, pricking, nor disagreeable sensations of cold or heat.	11. Always formications, or pricking, or both, and very often sensations of heat or cold.
12. Anæsthesia rare; the tactile sensibility being but slightly, if at all, impaired, but the muscular sense may be almost lost.	12. Anæsthesia very frequent, and always at least numbness.
13. Usually obstinate gastric derangement.	13. Gastric digestion good, unless the myelitis has extended high up in the cord.
14. Variations in the degree of the paralysis corresponding with changes in the primary disease.	14. Ameliorations very rare, and not following changes in the condition of the urinary organs.

15. Urine is usually acid, unless the urinary organs are diseased.	15. Urine almost always alkaline.
16. Cure of the paralysis frequently and rapidly obtained, or taking place spontaneously, after a notable amelioration or cure of the urinary affection.	16. Frequently a slow and gradual progress towards a fatal issue; very rarely a complete cure.
17. *Usually* muscles do not become atrophied, and temperature is little lowered.	17. Atrophy of the muscles of the paralysed parts.

It is necessary, therefore, to look for some primary disease to account for *reflex paraplegia*, believing at the same time that such primary disease may ultimately establish a myelitis, while a diagnosis of *reflex paraplegia* is mainly based on a contrast of the phenomena which attend the various forms of paraplegia due to other causes.

In addition to the characters in the table, there are some symptoms peculiar and almost pathognomonic of forms of paralysis due to other local lesions—*e.g.*, meningitis of the spinal cord induces rigid spasms of the muscles of the back; intense pain on motion of lower limbs or of the spine; spontaneous acute pains that radiate from the spine to the lower extremities (similar to rheumatism); frequency of cramps; and pressure on the cord by a tumor or diseased bone. They may and often do produce a myelitis. Pressure causes a feeling of tightness and pseudo-neuralgic pains. A tumor in the grey matter of the cord is attended with anæsthesia from the very first, which may reach a higher degree than the power of motion; and reflex power below the lesion becomes then extremely exalted. In hæmorrhage into the cord there is pain, then sudden paralysis, and often convulsions.

Treatment.—In cases of *reflex paralysis* means must be taken, first—To diminish the external or peripheral irritation which causes the paralysis; second, To improve the nutrition of the spinal cord; third, To prevent the ill effects of rest on the paralysed nerves and muscles.

In cases of disease of the urethra or prostate, *an injection of a solution of one grain of the extract of belladonna, in twenty drops of laudanum, is to be made into the urethra, and the injection should be retained for half an hour, or even an hour, after which some emollient decoction should be used, such as lintseed tea, to wash out the passage.* Every two or three days this operation should be repeated. If the bladder be diseased, Dr. Brown-Séquard advises a solution of *one grain of the extract of belladonna*, in twenty drops of laudanum, to be used as an injection, after a complete emission of urine. Injections of *carbolic acid* are also of use in counteracting decomposition of urine and epithelium in the bladder. If the prostate is enlarged, a suppository ought to be put at times into the rectum. One of the best suppositories for this purpose is composed of the following ingredients, namely:—*White sugar, white soap, and gum-arabic in powder, of each three grains; opium, in powder, a grain and a half; or belladonna extract, one grain; or both combined. These being mixed together, the mass is to be formed into a conical shape, and being dipped in melted wax, is ready to be introduced, when required, into the rectum* (Simpson). When the irritation causing reflex paraplegia starts from the vagina or uterus, a pill of half a grain of the *extract of belladonna*, with a grain of *opium*, surrounded by a piece of cotton wool, is to be introduced into the vagina, and made to surround the neck of the womb. By means of a thread it may be withdrawn so soon as the pain ceases or diminishes (TROUSSEAU, BROWN-SÉQUARD). *Belladonna* ought not to be used constantly. *Opium,* combined with *strychnia,* is of greater use in reflex paralysis; and of all remedies, *strychnia* is best suited for promoting the second indication, which points to increasing the nutrition of the spinal cord. The dose combined with opium must be a small one—namely, *one-fortieth* to *one-thirtieth of a grain daily;* and when used alone, its dose may be *one-twentieth of a grain;* and when employed together with *belladonna,* its dose must be still larger. In cases where no congestion nor inflammation of the spinal cord exists, *strychnia* ought to be persistently employed; but its use ought to be suspended at once whenever it produces spasms, or even numbness of the feet in getting out of bed in the morning (BROWN-SÉQUARD). *Sulphur baths* are also productive of great benefit.

The third indication—namely, to prevent the ill effects of rest on the paralysed nerves and muscles—is best met by the employment of Voltaism and shampooing of the paralysed limbs. Two or three applications, of ten minutes each, in a week, are sufficient; but of all things it is necessary that the primary disease (causing by its persistent existence the reflex paralysis) should be cured or mitigated.

SPINAL HEMIPLEGIA; HEMIPARAPLEGIA SPINALIS—Syn., UNILATERAL LESION OF THE SPINAL CORD; BROWN-SÉQUARD'S SPINAL PARALYSIS.

Definition.—*The localisation of lesion in one lateral half of the spinal cord at any height, involving more or less complete destruction of one entire lateral half of a transverse section of the cord, the longitudinal extent of which may vary greatly.*

Pathology.—The phenomena of this condition are of frequent occurrence; and the peculiar group of crossed sensory and motor paralysis are due to the decussation of the sensory fibres in the cord (BROWN-SEQUARD). Clinically, the cases are characterised,—By unilateral motor paralysis—hemiparaplegia or hemiplegia—on the side of the lesion; by crossed anæsthesia on the opposite side of the body; by symptoms referable to the roots of the nerves corresponding exactly with the height at which the lesion is situated; and by a series of other manifestations which may vary very much according to the seat and extent of the lesion, but which are also severally very characteristic of the different portions of the spinal cord which may be affected (ERB).

(*a.*) *Causation and Morbid Anatomy.*—The most frequent cause of *unilateral* lesion is to be found in traumatic injuries, which thus perform the experiment of cutting one lateral half of the cord in man. The paralysis may result from compression of the cord from without, as from meningeal tumors, or intra-meningeal effusion of blood; from falling on the back heavily, or from a height; or from the result of hæmorrhage within the cord itself, or as the result of unilateral circumscribed *sclerosis;* or of a *chronic myelitis* from cold or concussion of the spine; or as the result of intra-medullary tumor; or from syphilitic affections.

Symptoms.—There is motor paralysis, confined to the side of the lesion (unilateral), which may involve one leg merely (hemiparaplegia); or, in proportion to the height of the lesion in the cord, it may involve the arm of the same side (spinal hemiplegia). On the side opposite to the seat of lesion in the cord, there is either entire freedom from all motor paralysis, or it may be affected only to a slight and subordinate degree. On the side of the lesion there is also evidence of vaso-motor paralysis, especially if the lesion is of rapid development. The temperature of the paralysed limbs may be 1·8° Fahr. above the normal. Muscular sense and muscular sensibility are diminished on the affected side; while there is hyperæsthesia of the skin for all qualities of sensation over the entire paralysed side. The hyperæsthetic region is generally bounded above by a narrow anæsthetic belt corresponding exactly with the height and longitudinal extent of the lesion in the cord. The conditions in reflex action vary.

On the side opposite the seat of lesion, the following condition exists:—There are no motor disturbances, while the sensitiveness of the muscles and electro-muscular sensibility are retained. But there is more or less complete anæsthesia of the skin to a height corresponding with that of the lesion, and extending exactly to the median line of the body, as may be demonstrated on the trunk and scrotum. There is no vaso-motor paralysis. In individual instances there are less constant and less characteristic manifestations; such as, a painful feeling of constriction corresponding to the level of the lesion in the cord; and, with painful sensations on the anæsthetic side, or on the paralysed side, or on both sides; disturbances in the evacuation of the bladder and bowels; sexual powers more or less diminished; emaciation of the paralysed side setting in early; lowering of faradic irritability on the paralysed side; occurrence of gangrenous bedsores on the anæsthetised side; development of an inflammatory knee-joint affection on the paralysed leg; and, lastly, the occurrence of well-marked *ataxy*.

Certain differences of symptoms occur according to the height within the cord at which the lesion exists:—(1.) At the *lumbar enlargement*, in addition to the symptoms already noticed, a *unilateral lesion* is attended by an anæsthetic zone on the paralysed side, corresponding to the area of distribution of one or more lumbar nerves; and may be situated on the pelvis, or the abdomen, or region of the groin, or anterior surface of the thigh. (2.) In the *dorsal region a unilateral lesion* produces a marked distinction in the condition of the two lower extremities as regards their motor and sensory disturbances. The hyperæsthesia and paralysis of the one contrast with the anæsthesia and complete mobility of the other. The difference extends to the trunk, ascending to a variable height, where the belt-like anæsthesia of the paralysed side becomes prominent, sometimes bounded by a narrow hyperæsthetic zone above. (3.) In the *cervical portion* of the cord the phenomena vary much, according as the lesion involves the upper or lower roots of the brachial plexus. In the *lower extremities*, the same conditions exist as in lesion of the dorsal portion. In the *upper extremities*, on the side of the lesion, there are more or fewer muscles and nerve domains paralysed; hyperæsthesia exists in some or in all parts, mingled with anæsthesia of certain regions of skin, or for certain varieties of sensation. On the opposite there is no paralysis, but more or less complete anæsthesia exists in all or in individual parts. The *neck and head* on the side of the lesion may show anæsthesia and hyperæsthesia of individual portions of the neck—vaso-motor and oculo-papillary paralysis, expressed by a higher temperature of that side of the head and body, heightened sensibility, narrowing of the palpebral fissure, and contraction of the pupil; on the *opposite side* anæsthesia of the neck may exist with a normal condition of the face and eye (Professor W. H. ERB).

Diagnosis lies between this condition and other forms of hemiplegia, or hemiparaplegia. In any form of cerebral hemiplegia there is either no disturbance of sensation, or it is to be found on the same side as the paralysis; there is one-sided paralysis of the face and tongue; there are disturbances of various cranial nerves, and other head symptoms.

Prognosis is governed by the nature and seat of the lesion. Traumatic cases justify a more or less favourable prognosis compared with disease.

Treatment is dictated by the nature of the lesion which causes this special form of paralysis.

ACUTE SPINAL PARALYSIS OF ADULTS; AND, SPINAL INFANTILE PARALYSIS (VON HEINE)—*Syn.*, ACUTE ATROPHIC SPINAL PARALYSIS—ACUTE INFLAMMATION OF THE GREY ANTERIOR HORNS—POLIOMYELITIS ANTERIOR ACUTA (KUSSMAUL)—PARALYSIE ATROPHIQUE DE L'ENFANCE (DUCHENNE).

Definition.—*A sudden feverish attack, with such cerebral phenomena as deafness, coma, delirium, or general convulsions, soon followed by a rapidly developed and complete paralysis, variously distributed over the trunk and extremities; but generally in the form of paraplegia. There is entire relaxation of the paralysed muscles; an absence of any severe disturbances of sensation; and there is no paralysis of the sphincters nor bedsores. The original paralysis is not progressive; but improvement gradually takes place as to the restitution of movement up to a certain point, which is not uniform. In the end the power of movement in some part is lost for ever; a paralytic and withered limb for life is all that can be hoped for* (Professor W. H. ERB).

Pathology.—(*a.*) *History.*—This is one of the most common diseases of the spinal cord; and it is best known by the name of *infantile paralysis*—a name which unfortunately cherished the belief that the disease was unknown in adults. But the advance of knowledge of nervous diseases in recent years, has shown that the disease is by no means confined to children; but that it is met with at almost any period of life. The name of "*infantile paralysis*" has therefore lost its force; and a more strictly scientific name has been devised, which indicates the pathological basis and nature of the lesion, whether occurring in young children or in adults. The inflammation being generally confined to the anterior grey matter, Professor Kussmaul has proposed to call the disease anterior polio-myelitis (from πολιὸς, grey, and μυελος, marrow). Professor W. H. Erb has adopted that name, and described the disease in his article upon it in Vol. XIII. of *Ziemssen's Cyclopædia.* The name has since been adopted in the descriptions of the disease as occurring in adults in this country by

Dr. Althaus, in a paper read before the Medical Society of London, on Feb. 11, 1878 (to which the Silver Medal of the Society was awarded). Cases in the adult have been since described in this country by Dr. Glynn of Liverpool (*Lancet*, Sept. 21, 1878); by Dr. Octavius Sturges (*Lancet*, Jan. 4, and May 3, 1879); and three cases by Dr. W. Allen Sturge (*Brit. Med. Journal*, June 7, 1879). Although the disease has been thus frequently met with, alike in young children and in adults, it was not till 1860 that it was clinically recognised and described by Jacob Von Heine. He especially described the atrophy and deformities of its second period. In 1860, he declared in favour of the *spinal* seat of the disease, by giving it the name of "*spinal* infantile paralysis," the name by which it has since been universally known and designated. He thought it probable that there was an alteration in the grey matter. Duchenne also, from clinical observation, came to the conclusion that the seat of the disease was *spinal;* but up till 1863, the observations had been entirely clinical. No positive evidence of the seat of the disease had been obtained by *post-mortem* examination of the spinal cord, when Cornil was the first to recognise distinct lesions in the spinal cord itself in cases of this disease. After that discovery Prévost and Vulpian, in 1865, made the positive observation that the essential lesions were principally situated in the grey anterior horns of the cord—an observation which was confirmed by Lockhart Clarke in 1868. Since that time, and in recent years, numerous cases have been examined by Charcot, Parrot, Jeffrey, Roth, Leyden, F. Schultze, Vulpian, Erb, and others; with the result that in no case has a *post-mortem* examination been made in this disease without the discovery of a lesion located in the grey anterior horns of the spinal cord. The disease and the seat of the lesion have been shown to be the same, whether occurring in young children or in adults, and any variations in the picture of symptoms is caused by the period of life, the subsequent growth, and the different excitability of the organism of the child as compared with the adult.

(*b.*) *Causation.*—Acute anterior polio-myelitis may occur at all periods of life; but cases are most frequently met with in children between the ages of one and four years. This age of childhood may therefore be regarded as predisposing to the disease; but the causes of this predisposition (unless congenital or inborn) are still undetermined. The disease has also been met with, commencing for the first time, at all ages, in the later periods of life up to sixty years of age; but much less frequently than in children. The cases seem equally divided between the two sexes. As to season there has been observed an increasing tendency to the disease during the summer months. Hence, warm weather has been regarded as a predisposing cause (SINKLER). There is no evidence of hereditary influence or of *neuropathic* tendency; but Erb and Duchenne (fils) have each seen it in a child associated with *tabes* causing the death of the father. The most blooming, robust, and strong children are those most frequently attacked by the disease. No special exciting cause, however, can be assigned. *Cold* has been shown to be an occasional cause, both in young children and in adults. Prolonged bathing in the sea, especially when hot, has been shown to be a powerfully exciting cause (see a case described by Dr. ALTHAUS; also one referred to by Dr. W. H. ERB). The outbreak of the affection has been associated with some acute specific disease, especially in young children, during the progress or soon after the subsidence of measles, scarlet fever, variola, typhus, or malarious fever.

(*c.*) *Morbid Anatomy.*—The essential anatomical changes in this disease are only to be learned—(1.) From cases in which the well-marked clinical characteristics of the disease have been accurately observed and recorded during life; (2.) from cases which are the most recent obtainable. Cases examined from twenty to sixty years after the primary attack, cannot give safe and positive data regarding the pathology of this disease. From anatomical observation already existing, it appears probable that the essential lesion consists in "a more or less diffuse acute myelitis of the anterior grey substance, which reaches its greatest intensity in the lumbar and cervical enlargements of the cord, and, as a rule, leaves no permanent and irremediable alteration, excepting at these two points. In many instances, however, a moderate extension of the process, both in the grey substance and into the neighbouring white (antero-lateral) columns, can be demonstrated;" but Professor Erb suggests that such are only secondary, and not essential. The cases on which these statements are based, Professor Erb divides into two groups, namely:—Those observed soon after the beginning of the illness (two to twenty months), and those observed very long after (17 to 61 years). There are no observations on the earliest stages—*i.e.*, within the first few days or weeks. In spinal cords examined at the earlier periods, nothing abnormal may be discovered by the naked eye; but the substance of the cord may seem tougher at the height of the cervical or lumbar enlargements, and there may be a slight dwindling of the cord, especially at the expense of the antero-lateral columns. A transverse section of such cords shows the markings indistinctly; the anterior grey substance more or less discoloured—whitish, reddish, soft, or diffluent; and sometimes a diminution can be shown in the volume of the grey anterior horns. The anterior roots—corresponding to the main seat of the disease—are grey, translucent, and atrophied. The membranes are generally normal.

But *microscopical* examination only can reveal the exact seat, variety, and extent of the lesion. Its seat is in the grey substance of the anterior horns. It is a diffuse lesion, with regions of greater intensity, particularly at the lumbar enlargement of the cord. Here, usually on both sides, or only on one, an area of softening exists of greater or less *longitudinal* extent ($\frac{4}{10}$ of an inch to $1\frac{1}{5}$ inch), sometimes situated more towards the centre, or more towards the front of the horn, and separated from the surrounding parts by a more or less sharp line of demarcation. Similar areas of lesion may also be found in the cervical enlargement of the cord. These areas of lesion consist of *inflammatory softening.* They are friable, soft, and disseminated with numerous granulation cells, or large bladder-like nucleated cells. There is also an increase in the number of nuclei and in the amount of connective tissue, with more than ordinary fulness of blood-vessels. The *large multipolar ganglion cells have more or less entirely disappeared.* Those remaining are found in all stages of degeneration and atrophy, while others are quite well preserved. The *nerve-fibres* and *axis cylinders* within the area of softening are also no longer to be seen. In the immediate vicinity of the softenings, there is frequently a striking multiplication of nuclei, which, as Professor Erb suggests, may be the commencing of an encapsulating process. The antero-lateral columns may be diminished in volume and the seat of a slight sclerosis.

At later periods, such as seventeen and sixty-one years after the primary paralysing lesion, atrophy and shrivelling of certain portions of the cord are much more evident, even to the naked eye, especially from wasting of the antero-lateral columns and shrinking of the anterior horns, the former being grey and translucent. The anterior roots are atrophied, and the seat of grey degeneration. The lesions are essentially similar to those at the early period, but with this difference, that within the foci or areas of lesion there is a more or less firm, fine-fibred connective tissue, rich in nuclei, especially at the periphery of the zone. There are also thickening and enlargement, and probably multiplication of blood-vessels. Granule-cells are generally absent, but there is a great development of *corpora amylacea*, as well as pigment, all nerve-structures being completely destroyed. Well-preserved ganglion-cells are only to be found outside the foci of lesion. "Clarke's columns" are generally intact. Sclerosis may exist in the antero-lateral columns; and there may be thickening and multiplication of the neuroglia, with or without atrophy of the nerve-fibres.

A difference of opinion exists as to the nature of these changes, namely,—as to whether the affection is originally a parenchymatous, *i.e.*, an acute inflammation of the true nervous tissue, or an interstitial myelitis, *i.e.*, an inflammation of the connective tissue of the cord. The question is still an open one, for material does not yet exist to determine it, from the want of observation of very early cases—*i.e.*, within the first few days or weeks. All agree, however, that the lesion is of the nature of an acute myelitis.

Symptoms.—In children the disease commences with more or less fever, of varying intensity and duration, the intensity of which bears no definite relation to the extent of the disturbances which it leaves behind. It usually lasts but one or two days. It is of an inflammatory nature, secondary to the acute process localised in a large part of the spinal cord, the premonitory manifestations being pains in the back and limbs. Serious initial brain symptoms very soon follow, in the form of congestion of the head, giddiness, confusion of ideas, drowsiness, somnolence, and coma. General convulsions of greater or less extent may also arise once or oftener, but they are generally of short duration. In some rare cases no initial symptoms may be noticed; but the characteristic paralysis may appear in the night, or in the day, when the child seems well; but even when the initial symptoms are most severe, they subside in a short time (in three days at most), when the child's general health seems quite good, were it not that more or less extensive paralysis has supervened. When taken out of bed, or placed on the knee, or in a bath, one limb or all the limbs hang down relaxed, powerless, and incapable of movement. This paralysis progresses more or less rapidly; one leg may be paralysed completely in half an hour, or in a few hours, the other leg following soon, and then the arms. Paralysis may thus become complete within a night, or in from one to two days, and rarely longer than a week. There may be also paralysis of the bladder, also usually incontinence, and more rarely retention of urine, which may last a couple of days, or even weeks, but which eventually disappears. The subsequent progress is uniform in different cases. When the paralysis has thus reached its maximum development, it commences to decrease. In rare cases improvement begins very soon, in from one to three weeks, by single little movements, which gradually extend. But the muscles remain relaxed and soft, yet not becoming much emaciated. In such favourable cases complete recovery may be attained in the course of a few months—temporary spinal paralysis. In the majority of cases, the course is not so favourable. Improvement advances only to a certain point. The arms may become entirely or partially free, or perhaps only partly so. The movements of the shoulders and elbows may return, while the hands remain partially paralysed, or *vice versa.* In the same way partial recovery occurs in the lower extremities; one limb may become entirely movable, while the other remains completely paralysed; or, the thighs may return to their normal condition, while the legs remain paralysed. In a large majority of cases, the larger part of the muscles remain paralysed for a very long time, or permanently, in all of which a high degree of rapidly progressive atrophy is at once set up, and examination by electricity demonstrates degenerative change. With complete paralysis there is entire flaccidity of the muscles; a high degree of atrophy of them with great softness to the touch; total failure of all reflex action in the limbs (alike of skin and tendons), but the sensibility of the skin is everywhere normal. Children may remain for a long time in this condition, but otherwise in excellent health, growth and development of the non-paralysed parts advancing normally, and so they form a contrast to the changes which take place in the paralysed limbs, which are so peculiarly characteristic of the disease. There is progressive emaciation of muscles, till they completely disappear, and only a skeleton condition of a limb remains; but sometimes an exuberant production of fat may hide the atrophy. Contractions occur in most various muscles and groups of muscles. The bones cease to grow. The limbs are thus short and stunted, and withered away. The joints become distorted, lax, and mobile, so that there is displacement of the articular ends of the bones. Thus the most manifold and extreme deformities of limbs, joints, and spinal column are produced.

In adults the phenomena are essentially the same as those in children; but there is less tendency to cerebral disturbance. There is, however, general indisposition, with fever, which is generally introduced by lively backache and pain in the extremities, and with feelings of formication or numbness (*paræsthesia*). Severe headache, dulness, somnolency, and even slight delirium, may occur as well as well-marked gastric symptoms, such as vomiting. The advanced symptoms are exactly similar to those already described in children. There are no disturbances of sensation, and the patient never takes to bed; bladder troubles soon subside, and the sexual functions remain entirely normal. Although the limbs may be withered, there is no shortening, because the limbs are already fully grown.

Diagnosis.—There is a possibility of confounding it with the sequelæ of other forms of disease. It has also to be distinguished from acute, central, or transverse myelitis, in which there is disturbance of sensibility, and of the bladder and rectum, the occurrence of bedsores, and of increased reflex action, and usually no atrophy. Hæmorrhage into the grey matter (*hœmatomyelia*) presents phenomena resembling acute spinal paralysis; but the origin is, like apoplexy, entirely sudden. There is almost invariably disturbance of sensibility, with paralysis of the sphincters and bed-sores. *Myelitis from compression* causes a form of paraplegia with which acute spinal paralysis is not to be confounded. Myelitis from compression causes disturbances of sensibility, heightening of reflex excitability, paralysis of the bladder, spasmodic phenomena in the lower limbs, but without atrophy or evidence of degeneration. It is also to be distinguished from *cerebral hemiplegia*, *progressive muscular atrophy*, and *pseudo-hypertrophy of muscles*, from *hip-joint disease*, and from the stiffness of *rheumatism.*

Prognosis.—The disease does not endanger life, but if recovery of movement does not take place within from three to six months, one or more paralysed and withered limbs will remain for life.

Treatment.—The acute myelitis, if detected, first demands attention. Local abstraction of blood by leeches or cupping should be had recourse to over the cervical and lumbar enlargements. The bowels should be freely moved by cathartics, followed by *iodide of potassium* in appropriate doses. Rubbing in of mercurial ointment and counter-irritation may afterwards be employed the whole length of the spine, or blister, plaster, or painting with the *tincture of iodine.* Dr. Althaus has most confidence in the subcutaneous injection of *ergotine* (Bonjean's solution in diluted water), one-fourth grain for a child from one to two years of age, one-third grain for a child from three to five years, and half a grain for children from five to ten years, and a grain for patients upwards of ten years; to be repeated as symptoms indicate, once or twice a day. When acute symptoms have subsided, the diseased area within the cord should become the main object of treatment, by induction of the catalytic action of the voltaic current. A large electrode, which will cover the entire diseased area, is to be placed over the lumbar or cervical enlargement, while the other is to be applied to the anterior surface of the trunk. First the *anode*, and then the *cathode*, are to act alternately for from one to two minutes at a time, with a moderately strong current. This treatment must be continued for a long time, or repeated at intervals for several years. The faradic current is not to be used at this period. Such are the recommendations of Erb. They differ somewhat from the injunctions of Althaus. "It being most important that the voltaic current should pass through the anterior cornua, it is better to have one pole on the spine and the other one on the front part of the body, than to place both electrodes to the spine. If the cervical cord has to be acted upon, place the positive pole to the nape of the neck, and the negative to the *manubrium sterni.* When the lumbar enlargement is diseased, the positive pole is put to the loins, and the negative a little below the umbilicus; while, when the dorsal cord has to receive the voltaic influence, the positive pole should be slowly guided along the whole dorsal spine, the negative being left stationary at the ensiform cartilage" (ALTHAUS, l. c., p. 53).

If electric contractility remain, much may be hoped for, so long as nerves and muscles have not altogether degenerated. The principles of treatment are the same as have been advocated in analogous diseases—

namely, restorative and not depressant. The general health must be improved by the use of baths, with a strong and abundant diet; the use of cod-liver oil, and prolonged sojourn in mountain air. The systematic, active, and persistent application of the voltaic current of electricity is the best means of preserving the functions of the implicated muscles, and of arresting their atrophy and degeneration, provided all other hygienic arrangements are maintained which have already been insisted on as to food, clothing, ventilation, and open air. The *cathode* is to be applied over all the paralysed muscles and nerves; while the *anode* is best placed upon the vertebral column at the height of the main lesion. The strength of the current should be such as to produce distinct twitchings and lively redness of the skin. The faradic current may, however, still be used when the muscles retain their faradic irritability. The faradic application is to be made with moist electrodes.

Mild purgation may be required; alteratives, especially small doses of *mercury*, continued for a long time, have been of service; and so also have *quinine*, *iodide of potassium*, and *iodide of iron*.

Friction, kneading, squeezing (massage), and shampooing ought also to be persevered in. Three or four small circular wooden balls, about the size of a walnut, shut up in a box, with holes in its cover, so as to expose one-third part of the surface of the balls on a uniform level, makes a good shampooing apparatus.

PROGRESSIVE MUSCULAR ATROPHY.

LATIN EQ., *Atrophia Musculorum Ingravescens;* FRENCH EQ., *Atrophie Musculaire Progressive;* GERMAN EQ., *Progressive Muskel-atrophie;* ITALIAN EQ., *Atrofia Muscolare Progressiva.*

Definition.—*A peculiar wasting of muscles, with atrophy of their substance, associated with lesions of grey matter in the centre of the cord—namely, wasting of the ganglionic cells of the anterior horns, with oleo-granular degeneration, and of the anterior roots of the nerves of the spinal cord, and attended by paralysis.*

Pathology.—(*a.*) *History.*—Numerous cases of wasting of the muscles of one or more limbs, independent of any well-defined cause, have been from time to time observed, and are recorded in various publications; and we are indebted to Dr. William Roberts, of Manchester, for the first systematic treatise on the subject, in 1858. Such cases have hitherto been looked upon as *extraordinary* or *anomalous cases*, and were described as instances of "creeping palsy," "partial" or "local palsy," or "anomalous hemiplegia." In France it has been described by the name of "*atrophie musculaire progressive*," or "*paralysie graduelle du movement par atrophie musculaire*" (DUCHENNE, ARAN, CRUVEILHIER, 1849–50), corresponding to the third form of progressive paralysis described by Meryon—namely, from "granular degeneration of the muscles, and where no disease or lesion is found in the nervous centres." Friedreich of Heidelberg has described it as of the nature of a chronic multiple inflammation of the muscular fibres, and has named the disease "*progressive, chronic poly-myositis.*"

It seems to be still, however, an open question, whether progressive muscular atrophy is really a substantive disease of the muscular texture, or dependent on a structural change in the spinal cord. The College of Physicians have numbered it, and classed it among *diseases of the muscular system.* This is also the view adopted by Niemeyer, who believes that the long dispute as to the nature of this disease has been decided in favour of those who regard progressive muscular atrophy as a primary muscular affection, since almost all observers have agreed in regarding the continuance of excitability in the atrophied muscles, as long as they contain muscular elements, to be the pathognomonic sign of the disease. Dr. William Roberts, in this country, contends that the muscles are the parts essentially diseased, in the first instance, in the cases of "wasting palsy" which he has described. The lesion is of the nature of a degeneration. The nerves and muscles alike retain their excitability till the muscles perish. On the other hand, in addition to the changes in the muscles, Luys was the first to discover and describe alterations of the grey matter of the centre of the cord; and his observations have been confirmed by Lockhart Clarke, Jaccoud, and Charcot. But besides the examination of the spinal cord made by these men, there are those made by Gull, Herard, Virchow, and others, who demonstrate the nervous rather than the muscular seat of the disease. Others, again, place the origin of the disease in the sympathetic system of nerves (SCHNEEVOOGH, JACCOUD, BÄRWINKEL, REMAK, TROUSSEAU). "The opinion seems, however, to be steadily gaining ground, that the nutrition of the muscles is placed under the control of a special set of organic nerves, having upward connections with the sympathetic ganglia and the cerebro-spinal axis, which are by no means identical with the central connections of the motor nerve fibres of the same muscles. Assuming the existence of such nutritive centres, all the clinical phenomena of wasting palsy, and the various findings of the *post-mortem* examinations, admit of easy explanation, on the supposition that these centres, or some of their ganglionic connections, are the primary seat of the disease, and the numerous associations and complications can scarcely be accounted for on any other hypothesis" (DR. WILLIAM ROBERTS).

In all these progressive diseases the muscular tissue is only secondarily affected, and the symptoms depend upon those areas of the cord and medulla oblongata which become involved in the first instance, and upon those which afterwards participate in the pathological processes (ALTHAUS, l. c., p. 48).

(*b.*) *Causation.*—The question to determine is, whether or not the atrophy of the muscle is subsequent to the paralysis. If so, there are good reasons for believing that the wasting of the muscular tissue is an immediate or direct effect of the lesions in the cord; and the paralysis is generally too rapid to be accounted for by mere inactivity of the paralysed muscles. Dr. Roberts believes the disease to be of constitutional origin; and the evidence of this rests upon the facts, that it is transmissible from parent to offspring, and that in its march it exhibits a bilateral symmetry. Over-exertion he traced as a cause in a certain set of cases occurring amongst the working classes in males, in which the right hand and shoulder were more affected than the left. Twenty-five out of sixty-nine cases were of this kind. The disease has been observed to follow cases of fever and sunstroke, severe falls, and blows on the back of the head or spine; and it is well to observe, in all cases, whether or not the brain has received any accidental shock. It is probable that several distinct diseases have been described under the name of "wasting palsy" (W. T. Gairdner).

(c.) *Morbid Anatomy.*—The lesions are to be sought in the nervo-muscular system; and when death occurs in this and other forms of progressive paralysis, it is usually by some disorder of the respiratory apparatus, inducing *bronchitis, broncho-pneumonia, laryngitis,* or simply *apnœa,* from paralysis of respiratory muscles. Lesion of the muscles is constant, and nearly identical in all the cases. They are wasted away, some slightly, some profoundly, even to annihilation, their place being marked merely by origins and insertions, composed of more or less condensed connective tissue. Where muscular tissue is visible, it has lost the red hue of health, appearing of a pale red colour, like the flesh of a frog or fish. The degree of change into fat is various; in some no fat can be found, in others it is abundant, probably as an after-degeneration. Under the microscope the striped elementary muscular fibres are seen to be completely destroyed, the sarcous element being diffused, and in many places converted into oil-globules and granular matter, whilst the sarcolemma, or tunic of the elementary fibre, is broken down and destroyed (Meryon, *Med.-Chir. Trans.,* Vol. XXXV., p. 76). The striæ may be distinct, and between them grey or brilliantly shining molecules are to be seen; and where no striæ exist, long cylinders occupy the place of the primitive fibres (Galliet). Friedreich showed that there is at first germination of the interstitial connective tissue of the internal perimysium between the primitive bundles; the muscular corpuscles are swollen, and their nuclei germinating. The transverse striæ become cloudy and granular.

With regard to the nervous system, the *central organs,* the *spinal roots of the nerves,* and the *peripheral distribution,* have each engaged attention. In thirteen only of the 105 cases recorded by Roberts was the spinal cord examined; and in *four* instances it was found diseased. It is unnecessary to repeat again here how imperfect our information is on this point. Of the lesions recorded, a summary may be stated as follows:—(1.) While the spinal centre was considered sound, the anterior roots and peripheral distribution of the muscular nerves were extensively diseased. (2.) Inflammatory softening of the cord accompanied fatty degeneration and destruction of the anterior roots; and in one instance the peripheral muscular branches were destroyed. (3.) Amyloid degeneration of the cord, confined to the posterior median columns, has been observed in one instance, associated with granular degeneration of peripheral muscular branches, but with preservation of the anterior roots. (4.) In the cases examined by Mr. Lockhart Clarke, in the regions of the spinal cord which supplied the wasted muscles, numerous patches of transparent granular degeneration were found, especially in the grey substance, and generally around or in the vicinity of blood-vessels. The nerve-cells were shrunken and atrophic, and they contained an unusual number of coarse pigment-granules, while *corpora amylacea* were abundant around the central canal of the cord.

The more closely and carefully the spinal cord has been examined after death by competent observers, the more surely has it been found diseased. Progressive muscular atrophy is thus shown to be a disease of the spinal cord, in which a lesion affects chiefly the ganglionic cells of the anterior horns of the grey matter, whence the motor roots emerge, and which preside over the nutrition of the muscles. The number of wasted muscles is fairly proportionate to the degree of atrophy which is found in the anterior cornua (ALTHAUS, l. c., p 323). This sclerosis proceeds in a peculiar manner, affecting some ganglionic cells more and others less completely, some sooner and others later, while some are entirely spared. Hence muscles in different parts of the body seem to be capriciously attacked; and yet there is a tendency for certain sets of muscles, which are either co-ordinated in function or symmetrical, to suffer at the same time. There is nothing specific in the lesion itself, which may be either ordinary grey degeneration, sclerosing myelitis, Clarke's granular disintegration, or pigmentary degeneration of the cells. But the ultimate result is always the same,—namely, atrophy of the ganglionic nerve-cells, which perish one after the other. In all the cases observed the muscles affected are those under the control of the will; hence the course of the disease is easily followed, either by the changes produced in the external form of the parts, the absorption of masses of muscles, the displacement of bones, the abnormal position of joints from loss of their muscular supports, or by the failure of certain movements which contribute to outward expression or inward function; such as, facial physiognomy, deglutition, vocalisation, or respiration—all of which depend on the operation of striped muscles under the influence of the will. When the disease affects the muscles of the limbs, the *disappearance* of the *muscles* causes very notable changes of conformation. The rounded contour gives place to a lean and withered aspect, the bony levers stand out in unaccustomed distinctness, so that the limb has the appearance of a skeleton clothed in skin (ROBERTS). As a general rule, when one limb is attacked, its fellow on the opposite side shares its fate; when the disease is unilateral, the right side is more likely to be its seat than the left. But the disease seems to be extremely capricious and uncertain in its line of attack, scarcely two examples being exactly alike in the combination of muscles implicated, or the relative degree in which they suffer. But when the malady affects the shoulder it scarcely ever fails to include the upper arm; so also the forearm and hand are generally associated in the disease. In the upper limbs the morbid action seems to radiate from two centres,—one in the hand, from which the forearm is invaded; and the other in the shoulder, from which are reached the muscles of the upper arm and those which brace the shoulder-blade to the ribs. When the hand and arm are destroyed, the evil does not then pass up the arm, but starts away to the shoulder, or to the opposite hand. In the same way, when the shoulder is first attacked, the disease does not descend along the upper and forearm to the hand, but, passing over the elbow, it begins afresh in the ball of the thumb, and from that focus spreads up the limb; so that the parts latest reached are those about the elbow, especially the masses that take their rise from the humeral condyles.

Symptoms.—The invasion of wasting palsy is usually slow and insidious. It creeps on unawares; and the victim of its attack only becomes cognizant of the disease when he notices some marked failure in certain muscular powers. The tailor notices that he cannot hold his needle; the shoemaker wonders that he cannot thrust his awl; the mason's hammer has grown too heavy for his strength; the gentleman feels an awkwardness in handling his pen, in pulling out his pocket handkerchief, or in putting on his hat (ROBERTS). On comparing the weakened member with its fellow, it is seen to be wasted, and the failure of power increases; the lifting power is reduced to nothing; the grasp is gone; and at last palsy becomes complete. In the majority of cases the disease commences in the upper extremities; and, if in the legs, it is probable that the atrophy will spread to the trunk. In more than *one-third* of the cases noticed by Dr. Roberts, the hand was the member originally seized, and the exact spot nearly always the ball of the thumb; and the right hand more often than the left. This also is the experience of Dr. Althaus. Next to the hands, one or other shoulder is the favourite starting point. *Loss of power* is a chief phenomenon, and it corresponds to the grade of the atrophy of the muscle. It is only in extreme cases that any part is reduced to absolute immobility. *Muscular vibrations*, consisting of little convulsive twitchings or quiverings of individual muscular bundles, are also early phenomena. They do not impart any movement to the entire muscle; but parts of the muscle seem to spring beneath the skin in quick momentary tremors, undulating over the surface. Tactile sensation generally retains its delicacy in the skin over the affected parts; but in some cases anæsthesia has been noticed with exaggerated facility of reflex movement, chiefly in the skin, over the muscles subject to the quiverings already described. Electric contractility exists, but is less in the affected muscles, and the amount of diminution is in direct proportion to the degree of their atrophy (BENEDIKT, DUCHENNE, REMAK).

Pain is by far the most common of the nervous symptoms. It is present in about half the cases recorded. It is generally transient, and is usually most marked at the commencement. In some cases the pain is sharp and continuous in the joints and along the muscles from the outset, continuing for several months, with pain in the dorsal spine, and in the extremities, and in the bones (REMAK, MEYER, ROBERTS). The advance of the malady has, in one case at least, been attended by an almost childish degree of helplessness, and a most pitiable state of mental irritability and hypochondriac depression (W. T. GAIRDNER, ADAMSON, BELL, GULL, DAY). *Unusual sensitiveness* to low temperature is occasionally a prominent and very annoying symptom; the temperature of the affected parts is always lowered. As a rule, the general health does not seem impaired. Intelligence is clear, judgment firm, and the emotions under control till towards the end, and all the organic functions appear to be performed with regularity.

Prognosis.—The *progressive forms of paralysis* are the most intractable in the domain of Medicine, and the gravity of the prognosis depends upon the disease confining itself to the extremities and the muscles thereto relating, or extending to the trunk and face. The signs of extension to the face are—a diminished mobility of the lips, a slur in the articulatior of words, frequent sighing, and fibrillary muscular tremors on differen parts of the chest, abdomen, or face. Death usually occurs from bronchitis, as the expiratory muscles become involved. The expectoration becomes difficult or impossible. Bulbar paralysis may also supervene. If the disease has been hereditary, there is every fear that it will be fatal; and so also if the lower limbs are first attacked.

The disease runs its course with great slowness. Some cases complete their history in six or eight months, others linger on through many years. Recovery, permanent arrest of the palsy, or death, are its various terminations. The mean duration of the cases ending in recovery has been *one year and two months;* of those ending in permanent arrest of the palsy, the mean duration of the cases has been *two years and three months;* of those terminating fatally, the mean duration was *five years and two months.* The longest duration of a case ending in recovery has been *two and a half years,* and the shortest period *eight months.* The longest case ending in arrest continued active for *seven years,* and the shortest for *four months.* Fatal cases have not been known to terminate under *twelve months,* while one lingered for *twenty-three years,* another for *eight years,* and some beyond *four years* (ROBERTS).

In those cases which terminate by permanent arrest, the wasting of the muscle ceases, and the limb continues for an indefinite period in its maimed condition, neither amending nor deteriorating. The muscles atrophied are not regenerated, but remain *in statu quo;* and those which are not completely atrophied continue to exercise their feeble powers under the influence of the will, but they never regain their former bulk or vigour. After lying torpid for years, the malady may awaken to new and more violent activity (ARAN, VIRCHOW); and temporary lulls in the progress of the atrophy are common enough, but they seldom last long (ROBERTS). The disease is invariably fatal when it invades the trunk. Also, when the physiognomy loses all expression from wasting of the facial muscles, it is a sure sign that the end is near (ALTHAUS, l. c., p. 327).

Treatment of *wasting palsy* ought to be strictly a restorative one (see p. 308, Vol. I.), believing that the disease is one of nutritional deterioration of the nervous centres. Preparations of *wine* and of *cod-liver oil,* with *localised faradisation,* are the most likely agents to improve the condition of the body (ALTHAUS). Local means will aid the hygienic—namely, *methodical exercise* and *douche baths,* or *cold mineral baths;* so also *thermal* or *sulphur baths,* and *galvanism. Frictions,* with stimulating linaments (such as camphor linament), are also favourably spoken of (DUCHENNE, GROSS, MEYER, ROBERTS). "Faradisation" ought to be practised at least three times a week, *for from five to ten minutes each time,* and continued at least a month before it is given up, if negative results are only obtained. Every muscle ought to be faradised in a special manner, according as it has suffered more or less in its electric contractility and nutrition. The power of the current ought never to be strong. When the sensibility of the muscle returns, the intensity of the current may be diminished. Its application should never be protracted beyond ten or fifteen minutes at the most—one minute, on an average, being allowed to each muscle, or distributed over several at a time.

If, on the other hand, it turns out, as the searching examinations of

Mr. Lockhart Clarke and others would tend to show, that these forms of progressive paralysis or "wasting palsy" are always associated with disease of the spinal cord, the efforts of treatment should, in the first instance, be directed to that part of the cord corresponding to the connections of the nerves with the site of lesions—the line of treatment to be pursued being determined by the general symptoms. The application of the *constant* current of electricity—the *positive electrode* being placed in the region of the cervical sympathetic, and the *negative electrode* upon the cervical and upper dorsal regions of the spine—has been followed by long amelioration, if not by a positive cure, in the hands of Remak and Benedikt (CLYMER, in *New York Medical Journal*, 1866, Vol. III.).

PARALYSIS FROM PSEUDO-HYPERTROPHY OF MUSCLES.

Definition.—*A disease in which, with granular degeneration of certain portions of the cord, and germination of connective tissue in the sciatic, tibial, and peroneal nerves, there also occurs germination of the connective and adipose tissue, by which the circumference of the affected limbs is very much increased, with simultaneous wasting of the muscular fibres, which causes loss of motor power in the extremities.*

Pathology.—(*a.*) *History.*—By Friedreich and others this form of disease is looked upon as a form of progressive muscular atrophy, modified by its occurrence during childhood. It was first described, in 1838, by two Neapolitan physicians—Corte and Giojco, and afterwards by Dr. Meryon in 1852. Its relations to muscular atrophy are, however, still obscure.

(*b.*) *Causation.*—The disease occurs particularly in childhood, and almost always in boys, when want of exercise and fatty food tend to induce the morbid changes characteristic of the disease.

(*c.*) *Morbid Anatomy.*—It is not absolutely decided whether and what the alterations of the nervous centres are which are connected with this disease. In a few cases granular disintegration of certain portions of the cord, and germination of connective tissue in the sciatic, tibial, and peroneal nerves, has been discovered. The morbid process in the muscles is probably originally inflammatory, and begins with germination of the connective tissue from the internal perimysium and adventitia of smaller blood-vessels. The new growth becomes fattily degenerate, and fat, interstitial and interfibrillary, continues to accumulate. The primitive muscular fibres then waste by continuous absorption from pressure; but muscular nuclei increase in number, and the fibres show granular cloudiness. The muscle-masses seem to increase in bulk, but the muscles are really atrophic. They appear pale, white, and shining, and scarcely to be distinguished from fatty tissue (ALTHAUS, FRIEDREICH, DUCHENNE).

Symptoms.—The disease shows itself first in the legs, then in the thighs, buttocks, muscles of lumbar spine, and abdomen. The calves and muscles of the thigh stand out conspicuously, like a youthful Hercules; but withal the seeming strength of limb, the patient is unable to stand or walk properly. A steady increase in the circumference of the limbs goes on up to a certain point; but they have a sponge-like fatty feel, quite unlike muscular development. Fibrillary twitchings are occasionally present. Faradic and galvanic muscular excitability are at first diminished, and eventually completely lost, while electro-muscular excitability may be increased. Pain in the back may exist after movement, and generally the patient is unable to sit down or rise up without assistance. No febrile phenomena exist at any period of the illness. The temperature of the affected limbs becomes diminished. Intelligence becomes defective as the disease progresses. (See cases described by Dr. B. Foster, of Birmingham, *Lancet*, May 8, 1869. I have also seen one similar case in the practice of Dr. Lake, of Southampton, in 1873.)

Prognosis.—The disease is not so dangerous to life as progressive muscular atrophy; and in cases which have ended fatally, death has been due to affections of the respiratory organs; and when once distinct enlargement of the muscles takes place, nothing avails to delay the fatal progress of the disease (Bristowe).

Treatment is similar to that described in the previous affection.

PROGRESSIVE LOCOMOTOR ATAXY—Syn., TABES DORSALIS.

Latin Eq., *Ataxia Motus*; French Eq., *Ataxie Locomotrice Progressive* (Duchenne); German Eq., *Ataxie Locomotrice;* Italian Eq., *Atassia Locomotrice.*

Definition.—*A peculiar disease of the spinal cord, characterised by unsteady and disorderly muscular movements (but with muscular power entire), and more or less progressive loss of the faculty of co-ordinating power (voluntary and instinctive). There is sometimes temporary diplopia, with unequal contraction of the pupils. The course of the disease is slowly progressive; and the anatomical lesion is generally a (chronic myelitis in the form of) ribbon-like sclerosis of the white posterior columns and horns of the spinal cord, and posterior roots of the spinal nerves, leading to grey degeneration, sometimes with peripheral structure-change in the cranial nerves, and chiefly the second, third, and sixth pairs, in cases where the sight is affected. The disease generally begins in the lumbar region, and may extend throughout the entire cord, as far as the upper cervical portion, and even into the medulla oblongata.*

Pathology.—(*a.*) *History.*—It has received various names, some suggestive of its progressive nature from bad to worse; such as, *Progressive locomotor ataxy*, *Progressive motorial asynergia* (from α, privative, and συνεργος, joint work, co-operation), *Consumption of the spinal cord*, *Tabes dorsalis, or grey degeneration (degenerative atrophy,* Friedreich) *of the white posterior columns of the spinal cord* (Leyden), *Cerebral paraplegia* of Dr. Gull, and *Leno-myelitis posterior chronica*, or *Sclerosis of the posterior columns* of Erb.

Forms of spinal disease, under the name of *Tabes*, have been long known and described; but it is chiefly to English and German physiologists and pathologists that we owe the recognition of its peculiar form of motor disturbances, namely—those of *inco-ordination* (*ataxy*, from α, privative, and ταξις, order, want of order), as distinguished from *paralysis*. Dr. Todd of King's College, London, was the first to draw this distinction between this disease and paralysis. He stated that two kinds of paralysis might be noticed in the lower limbs, namely—(1.) Simple impairment or loss of voluntary motion; (2.) diminution or total absence of the power of co-ordinating movements, so that while considerable muscular power remained the patient found great difficulty in walking, and the gait became so tottering and uncertain that the centre of gravity of the patient was easily displaced.

(*b.*) *Causation.*—It is one of the most frequent affections of the spinal cord. The disease is one of middle life; most common in the prime of life, between thirty and fifty years of age; it is very rare in youth or old age. It is also more frequent, in a very large proportion, in males than females. The only positive determining causes seem to be prolonged bodily hardships—*e.g.*, exposure to damp, cold, depression of the nervous system from insufficient diet; also, bodily and mental exhaustion, combined with trouble and anxiety; venereal excesses, especially onanism. If there be a hereditary history of various nervous diseases (a *neuropathic tendency*) *motorial asynergia* may be connected with those diseases, and ascribed to a common origin; so that parents may transmit to their children only *the predisposition* to tabes—a certain weakness and irritability of the nervous system, or imperfect development of certain portions of the same. Evidences of tabes are also often developed after typhus fever, articular rheumatism, and acute pneumonia; also, after repeated labours and abortions; serious losses of blood, or too prolonged lactation; and diphtheria.

(*c.*) *Morbid Anatomy.*—To Professor Virchow, Türck, Rokitansky, and Leyden, in Germany, and to Sir William Gull and Lockhart Clarke, in this country, we owe our knowledge of the morbid anatomy of this disease. The essential anatomical lesions are found in the posterior columns of the spinal cord, the posterior roots of the spinal nerves, the peripheral extremities of the cranial nerves, and, exceptionally, in those of the extremities. The membranes of the cord are generally unaffected, or merely congested—sometimes thickened posteriorly by exudations adherent to each other and to the posterior surface of the cord. The pia mater is constantly thickened, opaque, or cloudy, and more or less adherent to the posterior columns. The degeneration and atrophy of the posterior columns of the cord cause the cord to seem flat in its antero-posterior diameter. The posterior median fissure is effaced, or marked only by a whitish line. The wasting consists of an atrophy with disintegration of the nerve fibres (gelatiniform degeneration of Cruveilhier and Virchow), with germination of the connective tissue, giving to the columns a greyish-red transparent aspect. It always commences in the immediate vicinity of the posterior fissure, close under the *pia mater;* and thence it spreads towards the sides and the grey commissure. On section the diseased part always exhibits the shape of a wedge, with the base towards the *pia mater.* *Corpora amylacea* are found imbedded in the connective tissue. The blood-vessels which traverse the columns are loaded on their external coats, and surrounded, to a variable depth, with oil globules and granules of various sizes. The *dorso-lumbar* region of the cord is the most constant site of lesion, which may involve from one or two inches to nearly the whole extent, and may extend in depth to the surface of the lateral columns, and occasionally even along the edges of the anterior columns. Localised areas of some widely-spread degeneration have even affected the extremities of the posterior cornua and deeper parts of the grey substance (LOCKHART CLARKE, *Lancet*, June 10, 1865). The posterior nerve-roots, both within the cord and after leaving its substance, are similarly involved in the degeneration. They are discoloured, grey, translucent, thin, and atrophic, resembling connective-tissue cords rather than nerves—an appearance especially well marked in the posterior roots of the *cauda equina*. The general progress of the disease in the cord seems to indicate a central commencement advancing to the periphery. The dura mater of the cord generally contains a considerable amount of clear or turbid fluid. In the later stages the degenerated parts of the medulla shrink away. The parts become hard and less transparent.

These anatomical lesions (so exactly limited to the posterior columns of the cord, and the roots issuing therefrom) by no means account for all the symptoms which are clinically recognised as distinctive of this disease. The nature of the peculiarly characteristic gait is sometimes referred for explanation to the loss of the "*muscular sense*" of Bell, or the loss of "sense of muscular activity" of Gerdy; or something equivalent. The inco-ordination is essentially a reflex disorder; and the lesions involve the co-ordinating centres (posterior column of spinal cord) of certain muscular acts, such as walking and standing. Some of the phenomena of the disease suggest involvement of the sympathetic—*e.g.*, the vesical phenomena, also the phenomena of irregular contractions of the pupils, and their becoming dilated during paroxysms of pain, or when the legs or arms were pricked or pinched, with local perspiration increased in some parts only of the skin—*e.g.*, forehead, palms of hands, and fingers* (BAZIRE).

The disease, then, is a peculiar one of the nervous system, commencing insidiously with evidence of disorder of some of the *cranial* nerves, perversion of sensibility in different parts of the body, ultimately giving place to inco-ordination of motion—always associated with degeneration of the posterior columns of the spinal cord and posterior nerve-roots, but without loss of muscular power or impairment of the intellect. The connecting links between the initial disorders of the cranial nerves (advancing centrically) and the lesions of the spinal cord (progressing peripherally) have not yet been made out.

In addition to these characteristic *post-mortem* lesions, there is evidence during life, both at the commencement and throughout the course of the disease, of localised congestions, expressed by rachalgia and spinal tenderness.

Symptoms.—The outset of the disease is insidious and slow. Pains, generally ascribed to rheumatism or neuralgia, but now known to be of spinal origin, first attract attention, particularly in damp weather. These pains may be fixed and aching, or lancinating, neuralgiform pains, darting, piercing, and transitory; or lively, stabbing, boring pains, described as shooting through the part like lightning, and lasting for days or nights, or for hours. Affections of the *second, third, fifth,* and *sixth* pairs of cranial nerves, sometimes with headache, giving rise to imperfect or double vision, strabismus, or contractions of the pupil, or dilatations, or deafness, are also insidious phenomena which may attract early notice. These several phenomena generally appear at different times and singly, although sometimes several together; and, after persisting for some time, may pass away, to recur again at intervals of months, and afterwards more frequently, and continuing so for years without further symptoms. Weakness in some of the limbs may supervene, but no actual paralysis. Another distressing harbinger of the disease is incontinence of urine, associated with spermatorrhœa during the night, with a great proclivity to sexual congress, which is no mere impotent desire, but results in effective sexual intercourse. After a period, however (of perhaps two years), the emissions on sexual intercourse become hasty, and a gradual loss of sexual desire and the power of erection supervenes. The bladder becomes irritable. Constipation is occasional, when the pains are severe, and it always aggravates them. Transient tickling sensations prevail in different parts of the body, such as the lips, nose, cheeks, forehead, with occasional numbness of the feet and arms, and the peculiar feelings of so-called "pins and needles, as if they were asleep." After another long interval (say of two more years), undue fatigue after walking is experienced; the legs give way under the weight of the body, and there is a sensation as if the patient walked on a spring-board, and could not keep his balance. These feelings may disappear and return; and eventually cutaneous hyperæsthesia or neuralgia of the skin (one side of the scrotum, testicle, buttock, or external part of the thigh), always unilateral, supervenes. Paralysis takes no part in the phenomena; but rather disturbed co-ordination of muscular movements, as originally pointed out by Dr. Todd, and afterwards described by Duchenne as characterising a new disease. Motorial inco-ordination, however, may not come on till after a period of several years (four or five) of suffering from such nervous symptoms. The pains then occur very irregularly, and last from a few hours to many days. At times they dart from limb to limb, or from one part of a limb to another; or, fixing on a small circumscribed spot, they give a boring, gnawing, or tearing sensation. The pains leave behind them a stiffness and soreness of the part. There may be atrophy of the optic disc, with contracted pupil; cutaneous anæsthesia of the soles of the feet, legs, forearms, and lips; and tactile sensibility seems slowly conducted to the sensory centre. There is decided diminution of cutaneous and muscular sensibility. "The floor is no longer distinctly felt by the feet; the foot seems to rest on wool, soft sand, or on a bladder filled with water. The rider no longer feels the resistance of the stirrup, and desires to shorten the stirrup-leathers. If the patient also does not *see* his movements, the power of co-ordination will be still more uncertain; if, while erect, he closes his eyes, he immediately begins to sway about and totter. If he closes his eyes in the horizontal position, he cannot tell the location of his limbs—he cannot tell whether the right foot or leg is crossed over the left, or the reverse" (ROMBERG). Reflex movements are not excited by tickling the soles of the feet. A painful feeling of constriction, as if by a tight band, is sometimes experienced round the body; and the sensation of "bearing down," in the perineum and rectum, with constipation, prevails. The bladder imperfectly empties itself, and cannot retain the urine after experiencing the desire to void it. The desire to pass it is frequent and imperative, especially during the night. Some degree of paralysis of the bladder is indicated by diminished force in the strain of urine—the jet is not well arched, and drops dribble away after the act is believed to be over. The urine sometimes passes involuntarily. Sexual desire is eventually abolished. The gait is characteristic. The steps are quick, short, and jerking. The leg and foot are well lifted from the ground, but they are thrown spasmodically and forcibly forward, the whole limb being extended. In bringing the foot down, the heel strikes the ground first. As the disease progresses, the limbs are thrown involuntarily to the right or left without purpose, and without the power of restraining in any way their irregular movements. In walking, the eyes are kept fixed on the legs; and a stick is used, over the handle of which a handkerchief may be placed to increase the surface of contact required by the numbness of the hand. The muscular force remains good, so that efforts to bend or extend a limb against the will can be resisted with strength. Involuntary jerkings of the limbs in bed prevent sleep. It is especially at starting that the patient has the greatest difficulty in maintaining equilibrium. When muscular inco-ordination of the upper extremities supervenes, the fingers become numb, and objects are handled with increasing clumsiness. The clothes cannot be buttoned, nor small things removed from the pockets by the fingers. If the patient is set on his legs with his eyes closed, and his feet close together, although he has muscular power to stand, yet he cannot preserve his body from falling, or guide himself in taking a few steps in the dark or with his eyes shut. He has no idea of the position of his lower extremities except from sight.

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* Dilatation of the pupil is known to result from disease of the *third* cranial nerve or its roots; section or lesion of the sympathetic in the cervical portion; intravertebral section of the two upper dorsal nerves, *followed by irritation of the peripheral ends of the cut roots.*

Reflex action of the tendons is also entirely absent in tabes, but whether this takes place before or after ataxy supervenes is still undetermined. Westphal first called attention to this point. He and Professor Erb have found that reflex action may be produced from tendons by giving them a light tap with the finger, most easily seen in the tendon of the *rectus femoris*, at the *ligamentum patellæ* ("patella tendon reflex"); but it may also be produced on the *tendo Achillis* and *triceps* muscle of the arm. Erb considers its absence one of the earliest symptoms of tabes dorsalis, and it is certainly one of the most constant (BUZZARD).

Trophic disturbances are also to be noted in the occurrence of eruptions of herpes, of lichen, and of something like pemphigus; in atrophy of single muscles, or of groups of muscles; also, in disturbances of nutrition of the joints. This joint affection generally manifests itself before ataxy sets in, most frequently in the knee-joint; and after that, in order, in the shoulder-joint, elbow, hip, and wrist. There is sudden and considerable swelling of the joint, from accumulation of fluid, without pain, redness, or heat, and generally also without fever, and sometimes with considerable swelling of the whole limb. Although painless, the affection is generally destructive of the articular extremities of the joint. Subluxation, or spontaneous luxations take place, so that at the knee-joint a peculiarly striking hyper-extension of the leg takes place. Abnormal frability of the bones also occurs.

The prolonged course of the disease is characterised by frequent natural pauses in its progress, sometimes lasting for a long time, before its onward progress is resumed—a point to be borne in mind in the treatment of the disease.

Paresis of the *oculo-motor* and *abducens* occasionally occurs, as shown by *diplopia*, less frequently by *strabismus* and *ptosis* of the upper lid, and lastly by *amaurosis* and *psychical* disturbances. Those symptoms indicate that the disease is progressing to the cerebral centres. Atrophy of the optic nerve has been traced as far as the *corpora quadrigemina*.

Electro-muscular sensibility is generally lessened; and during the early stages of the disease there is neither paralysis of muscles nor wasting of their substance, nor defective *nutrition* of the limbs. The patient may be strong enough to bear and to carry considerable weight; so that there is not muscular weakness, far less paralysis, in the ordinary sense of the term. The seeming paralysis is entirely due to the peculiar deficiency in the power of co-ordinating voluntary movements. But at last the final stage of true paralysis and increasing cachexia supervenes. Motor power diminishes, and finally reaches the stage of complete paraplegia. The legs grow stiff, contractures set in, the muscles become more and more emaciated, and the extremities grow thin and skeleton-like. The appetite diminishes, the bowels become more and more sluggish in function and beyond control. Cystitis with its results supervene. Bedsores set in with increasing febrile phenomena, and death soon terminates a disease of prolonged suffering. Its duration ranges from a few months to thirty years, with a mean duration of *seven* years in 119 cases whose progress has been observed (Dr. CLYMER).

Diagnosis.—In the early stage of the disease the differential diagnosis is between intracranial lesion and the peripheric lesions of the cranial nerves, which characterise the commencement of progressive locomotor ataxy. Here the ophthalmoscope becomes of use. In the amaurosis of intracranial disease there is always evidence of recent or past *neuritis* of the optic disc; but such is not the case in this disease (H. JACKSON). Care must also be taken not to confound the disease with progressive muscular atrophy.

Prognosis is decidedly unfavourable. The lesions may remain stationary for long periods, but recovery rarely, if ever, takes place, and eventually the disease progresses onwards to death; the immediate cause of which are lesions of the brain or spinal marrow, with softening, hæmorrhage, and progressive muscular atrophy; or acute inflammation of the urinary organs, and extensive sloughs over the sacrum. The majority, however, die during the course of intercurrent diseases, not directly connected with the motorial asynergia—namely, of pulmonary consumption, typhoid fever, pneumonia, pericarditis, and dysentery.

Treatment.—No benefit is to be got from antiphlogistic treatment, unless there be marked evidences of inflammatory irritation. Dry cupping may be of use for the relief of pain. *Derivatives* are likewise useless. Thermal baths are also injurious. Cool sponging of the body is of great use; and cold-water treatment generally, especially the use of the cold sponge-bath (ERB).

Phosphorus, in the form of *phosphates of metals* and *salts*, of *diluted phos-*

phoric acid, as a drink in the daily allowance of water, of *phosphate of soda* as an aperient, may be given with benefit. *Strychnine is objectionable. Nitro-muriatic acid* as a tonic, and *cod-liver oil* as a dietetic agent, may also be of use. *Cannabis Indica* and *belladonna* give the greatest relief to the pains. *Nitrate of silver* is recommended by Wunderlich, in doses gradually increasing to half a grain daily. *Phosphuret of iron* is also recommended. Dr. Althaus recommends *sulphur baths* as adjuvants, relieving pains and diminishing numbness.

Flannel should be worn next the skin, and chills from cold and damp carefully avoided, by the use of a chamois leather or spun silk close-fitting jacket over the flannel, and reaching from the height of the clavicles as low as the folds of the *nates* behind. The diet ought to be of the most nutritious materials which the patient can digest.

So long as there is active local hyperæmia, the use of electricity is hurtful. Faradisation may be of use in restoring, to some extent, sensibility to the skin, where local cutaneous anæsthesia has prevailed, after which patients seem to walk better for a time; but when used at all, electricity ought only to be employed during the pauses in the course of the disease, and then with great caution, in the form of a constant current, rather than the induced current, or faradisation. The current should be very weak at first, and the daily application for not longer than from three to six minutes, extended over months. Niemeyer has had no success either with nitrate of silver or the constant current of electricity; but he recommends them to be tried.

D.—*Local Paralyses.*

LATIN EQ., *Paralysis ex Parte;* FRENCH EQ., *Paralysie Locale;* GERMAN EQ., *Oertliche Lähmung;* ITALIAN EQ., *Paralisi Locale.*

Definition.—*Paralyses limited to particular sets or groups of muscles.*

Pathology.—Of this form there are several varieties which require special consideration, namely:—

I. *Paralyses of the Muscles of the Eye*—i.e., *of the muscles supplied by the oculo-motorius, the trochlearis, and abducens.*—Paralyses of these muscles are of frequent occurrence, either alone or as concomitant symptoms of various cerebral and spinal diseases.

(*a.*) *Causation.*—Rheumatic paralysis from cold most frequently affects the abducens and oculo-motor nerves. The trochlearis is seldom affected. Blows on the eye, and penetrating wounds, are the next most frequent cause. Next come diseases of the brain (hæmorrhages and tumors) in the pons and cerebral peduncles, the vicinity of the cerebral ganglia, and near the fourth ventricle; also in bulbar paralysis and paralysis of the cerebral nerves. Affections of the spinal cord, such as tabes and locomotor ataxy, are generally accompanied by paralysis of the muscles of the eye, and indicate anatomical changes progressing upwards from the cord to the nerve centres. Syphilis, diphtheria, and acute diseases, are also to be noted as causes of such paralysis.

Symptoms are dependent on the extreme exactness of the associated and accommodative actions of the ocular muscles in binocular vision. Disturbances in function of any ocular muscle at once produces *disturbance of vision*, *diplopia*, and unsymmetrical position of the globes. There is some defect of the absolute mobility of the eye in the direction of the action of the paralysed muscle, perceptible generally when the two eyes are compared. In many cases an overlapping or confused double image may be observed, the patient complaining of indistinct vision only, the vision being good for each eye alone. There is also false projection of the field of vision, which is displaced in the direction of the action of the paralysed muscle—*e.g.*, in the case of the *abducens* towards the outer side; and in paralysis of the *rectus inferior* downwards. *Giddiness* is a frequent and characteristic symptom.

1. (*a.*) *Complete paralysis in the region of distribution of the oculo-motor nerve* affects the following muscles:—*Levator palpebræ superioris, rectus superior, rectus internus, rectus inferior, obliquus inferior, musculus sphincter iridis,* and *musculus accommodatorius.* Hence, the upper eyelid hangs motionless to the lower border of the cornea, without the power of raising it (*ptosis*). The eye is almost motionless. Movements inwards, upwards, and directly downwards, are completely abolished. Rotation outwards is preserved. Every effort to move the eye causes it to rotate downwards and outwards, owing to the action of the external rectus and superior oblique; and the eye eventually becomes fixed in this position by the gradual contraction of these muscles. The pupil is dilated and immovable. The power of accommodation is greatly diminished. Double images appear in almost the whole field of vision, and fixation is only possible in a downward and outward direction. The eye is generally kept closed by the patient, on account of the giddy feeling superinduced by its attempted use, although vision is prevented by the ptosis.

(*b.*) *Paralysis of the levator palpebræ superioris* (*Ptosis*) may be quite isolated (as from injuries); but it is frequently associated with paralysis of the superior rectus. The upper lid hangs motionless; the fissure of the lids is narrowed. When the eye is directed upwards the lid is not raised.

(*c.*) *Paralysis of the rectus superior* impairs the power of turning the eye upwards. The line of vision may not be in the horizontal plane, or but slightly raised above it. The visual axis is directed more downwards than in the sound eye. The cornea diverges outwards from the action of the inferior oblique. The double images are superimposed vertically, and slightly decussate, that of the paralysed eye being the higher. Objects in the lower half of the field of vision are seen singly. The field of vision of the paralysed eye is projected upwards, and the head is depressed to avoid the double images. Giddiness rarely occurs.

(*d.*) *Paralysis of the rectus internus* impairs the power of rotating the globe inwards. When paralysis is complete no movement can be effected beyond the vertical line. The eye deviates outwards; the axis of the eye inclines outward; there is divergent squint. The double images are side by side, and crossed—the image of the diseased eye being on the healthy side. The lateral distances between the images increases with the movements of the object toward the sound side. Objects appear single in the outer half of the field of vision, provided there is no antagonistic contracture, when diplopia may extend into the outer half of the field of vision.

(*e.*) *Paralysis of the rectus inferior* produces symptoms the reverse of the previous paralysis. There is impaired mobility of the eye downwards. The globe remains at a higher level; the visual axis deviates upwards. The cornea is turned a little outward, owing to the action of the superior oblique. The double images are vertical, slightly oblique, and crossed, the image of the affected eye being lowest. Objects appear single in the upper part of the field of vision. There is secondary deviation of the sound eye downwards and outwards; the head inclines forwards, and giddiness is very marked.

(*f.*) *Paralysis of the obliquus inferior* is often most easily recognised by the secondary deviation upwards and outwards of the healthy eye. Double images are chiefly observed in the upper half of the field of vision, standing not quite vertical to one another; and their vertical distance increasing as the object is moved inwards, while their inclination to each other augments with rotation outwards.

(*g.*) *Paralysis of the sphincter iridis* (*Mydriasis paralyticus*) maintains the pupil in a medium state of dilatation. It is nearly or completely motionless when subjected to the stimulus of light. It contracts very little, or not at all, when the eyes are strongly converged, or when efforts of accommodation are made. Vision is indistinct from circles of dispersion, and bright light is intolerable.

(*h.*) *Paralysis of the musculus accommodatorius*, or *ciliary* muscle, may occur independently, or complicated with mydriasis. Local distance is increased. The patient cannot focus for near objects, nor read small print; hence size and distance are incorrectly estimated. It is one of the most common symptoms of diphtheric paralysis.

2. *Paralysis in the region of distribution of the trochlearis nerve* renders the superior oblique inactive. It is not easily recognised, because of the functional association of the superior oblique with the action of other muscles. Motility of the eye is but slightly affected, and that chiefly in the inner and lower angle of the field of vision. The deviation or squint is inwards and upwards on lowering the object; and simply upwards when it is turned far to the healthy side. Double images appear in the lower half of the field of vision on the inner side, vertically superimposed, somewhat oblique, and their distance increasing towards the healthy side. The feeling of giddiness is often well marked. The head is inclined forwards and turned to the healthy side, so that objects are brought into the upper and outer quadrant of the field of vision. This paralysis is often due to syphilis or injury, and rarely from exposure to cold.

3. *Paralysis of the nervus abducens* affects the external rectus only. Mobility of the eye outwards is impaired. The globe cannot be rotated outwards beyond the middle line. There is inward squint and diplopia in the external half of the field of vision. The double images are lateral, and their distance increases as the object is moved outwards. This paralysis is the commonest from cold—rheumatism, and is sometimes bilateral, as in tabes, and after acute cerebral meningitis. These varied paralyses of the oculo-motor muscles may supervene suddenly, as in the course of a night in the rheumatic and apoplectic forms; or slowly and gradually, as

when they result from syphilis, neuritis, or chronic disease of the cerebral centres.

II. *Paralysis in the region of distribution of the motor portion of the trigeminus—i.e., of the muscles of mastication (masticatory paralysis of the face).*—The muscles most usually impaired in function are the masseter, temporal, external and internal pterygoids, supplied by the motor branch of the fifth nerve. The paralysis is of rare occurrence. It is a symptom of certain forms of bulbar paralysis; and the lesions are usually intracranial, such as periostitis, exostosis, caries, extravasations, aneurisms, and tumors implicating the third division of the fifth.

III. *Paralysis in the region of distribution of the facial nerve—Mimetic facial paralysis—or Bell's paralysis—affects the muscles of expression of the face, usually confined to one side, but sometimes, though rarely, affecting both sides, and apt to be followed by contraction or tonic spasm of the muscles previously paralysed ("Spasmodic Tic" of Marshall Hall).*

Pathology.—(*a.*) *History.*—In editions of this text-book previous to the last one (the Sixth), erroneous doctrines were stated regarding facial paralysis, in accepting and perpetuating the views of Dr. Todd, that *the fifth nerve is more or less involved in the paralysing lesion,* and in mixing up the phenomena of *facial paralysis* depending on *central* or *cerebral lesion,* with *facial paralysis* due simply to lesion of the trunk or branches of the *portio dura.* I am indebted to my friend Professor Sanders, of Edinburgh, for kindly directing my attention to this error, and for references to his important paper in the *Lancet* of October 21, 1865. From his papers on this subject, from the clinical lectures of the late Professor Trousseau on *facial paralysis,* and from Professor Erb's exhaustive article, the following account is given of this affection:—

Causes.—It is one of the commonest forms of paralysis; and one of its most common causes is the action of cold on one side of the face, by exposure to the wind (*coup de vent*), especially to cold when sleeping in a draught or in the open air, or exposure to cold after being in a state of perspiration, or sitting in a railway or other carriage with the side of the face exposed to the wind. "Halla ascribes the increasing frequency of facial paralysis to the railways. People hurry to the station, arrive there warm, enter the carriage, and expose their face to the draught from the window. The result is palsy of the face" (NIEMEYER). There are many ways in which cold may produce these "rheumatic" facial paralyses, or facial paralysis from chill. The influence of *rheumatism* is also conspicuous in many cases, the patient being seized as suddenly as in *lumbago,* acknowledging a similar cause; residence in a damp place, or other exposure to cold and wet, being sufficient to bring on the attack. The influence of mental emotion—a sudden fright or start—has been known to induce the paralysis.

Of traumatic lesion the facial paralysis of new-born infants is not uncommon. It is generally due to the compression of the trunk of the facial nerves by the use of the forceps. If such compression has been severe or excessive, the injury may be permanent; otherwise, it is soon recovered from. It may occur from a box on the ear. Wounds of the *portio dura,* or fractures of the skull, involving lesion to the *aqueduct of Fallopius,* may also be followed by paralysis, and in all these instances the paralysis is sudden. When the facial nerve, during some part of its course, is interrupted in its functions by the secondary influence of advancing organic disease, the nerve becomes compressed gradually, and ultimately altered. In such cases the paralysis comes on slowly. In severe chronic inflammation of the internal ear, with destruction of the tympanum and ossicula, the petrous portion of the temporal bone becoming carious, facial paralysis is apt to supervene, and the lesions may be fatal.

The form of paralysis due to such causes is that known as *peripheral facial hemiplegia;* but the paralysis may acknowledge a *cerebral lesion* as its cause, such as cerebral hæmorrhages, lesions of the *pons Varolii,* implicating the fourth ventricle. In such cases the facial palsy is usually partial, affecting more especially the muscles of the mouth and cheek, leaving the closing movements of the eyelids unaffected. As a rule, also, the paralysis due to a cerebral lesion gives rise to a hemiplegia involving parts beyond the face, such as one or more limbs of the affected side. But there are also instances in which the paralysis due to a cerebral lesion (small cerebral hæmorrhages) has been exclusively limited to the face (GRAVES, DUPLAY). This is in keeping with other forms of hemiplegia (cerebral) in which the paralysis is localised; as when cerebral hæmorrhages or apoplectic-like seizures paralyse the tongue only, or an arm, or distort the features more or less, but generally combined with a hesitation in the movements of the leg, of which the patient is unconscious (TROUSSEAU). The previous existence of peripheral paralysis in such cases may render superadded paralysis from a cerebral lesion very difficult to diagnose. When the cerebral lesion is limited to the *pons Varolii,* to slight injury to the fourth ventricle, the paralysis of the face which results has many of the characters of the peripheral or Bell's paralysis, even to the absence of electric insensibility of the paralysed muscles (VULPIAN, quoted by TROUSSEAU, who, in the course of a very long practice, never saw a case of the kind himself).

Facial paralysis may thus be due to one or other of many causes, namely —(1.) To a lesion of the trunk simply, or of branches of the *portio dura* nerve at some part of its course, generally through the petrous bone, and independent of cerebral disease. This form of facial paralysis is sometimes also known as *Bell's paralysis,* or *peripheral facial hemiplegia,* or *mimic facial palsy.* (2.) *Facial paralysis* may be due to a cerebral lesion—a cause which acts upon the nerve before it enters the internal *auditory meatus*—in which case it is usually accompanied by hemiplegia of the limbs on the same side. This form of facial paralysis is known as *cerebral* or *centric facial hemiplegia.* (3.) A third form of *facial paralysis* is ascribed to reflex paralysis of the *seventh nerve,* consequent on paralysis or lesion of the *fifth pair,* generally an agency involving the peripheral ramifications over the face.

The first affection is that most frequently met with in practice; and although it is not a dangerous form of paralysis, it is one from which recovery is very slow, and in which prognosis, as to complete recovery of symmetry of the face, is uncertain. The nerve may be destroyed in the

Fallopian canal by caries or other lesion of the petrous bone, or by fracture, or gun-shot wound. The second form is the more rare, and is always a very grave disease, and not usually recovered from. In this form the nerve is most frequently pressed upon or atrophied by cerebral tumors; more rarely from exudations or thickenings of the *dura mater* or from *exostosis*. Both kinds of facial paralysis present many points of resemblance and of contrast, of great interest and importance in diagnosis; and the occurrence of paralysis from one of the causes mentioned by no means excludes the other, and therefore cases of both may exhibit extremely complex symptoms.

Symptoms and Phenomena of facial paralysis are confined to the "muscles of expression," including the *buccinator*, and do not involve the masticatory muscles (*masseter, temporal, pterygoid*) supplied by the fifth pair of nerves. A description of the phenomena of *facial paralysis* must therefore have due regard to the course and distribution of the *portio dura* of the *seventh nerve*. The facial nerve, or *portio dura*, of the *seventh pair* is the motor nerve of the face. It emerges from the lateral column of the spinal cord, as that column passes under the *pons Varolii*, and enters the *internal auditory meatus*. At the bottom of this *meatus* it enters the *aqueduct of Fallopius*, and follows the windings of that canal to the lower surface of the skull—namely, to the *stylo-mastoid foramen*—by which it leaves the osseous canal of the temporal bone. It is then continued forwards through the substance of the parotid gland, and separates in the gland, behind the ramus of the lower maxilla, into two primary divisions—the *temporo-facial* and the *cervico-facial*—from which numerous branches spread out over the side of the head, the face, and upper part of the neck, forming what is known as the "*pes anserinus*." Within the temporal bone it is connected with the auditory nerve by filaments of union, and where it swells into its gangliform enlargement, it is joined by the large superficial petrosal branch from the Vedian nerve, and also by the small superficial petrosal nerve. Close to the *stylo-mastoid* foramen it gives off several small branches—namely, the posterior auricular, a branch from the *digastric muscle* and *stylo-hyoid*, and a twig to the *stylo-glossus*. In front of the mastoid process it divides into an *auricular* and an *occipital* portion, and is connected with the *great auricular nerve* of the cervical plexus. It also gives off branches to the *digastric* and *stylo-hyoid* muscles, joining the *glosso-pharyngeal* near the base of the skull, and the plexus of the *sympathetic nerve* on the external carotid artery. The main trunk of the facial then separates into two primary divisions, the larger of the two being the *temporo-facial*, which is directed forwards through the parotid gland. Its ramifications and connections with other nerves form a network over the side of the face, extending as high as the temple, and as low as the mouth. Its branches are arranged in *temporal*, *malar*, and *infra-orbital* sets.* The

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* (*a.*) "The *temporal branches* ascend over the zygoma to the side of the head. Some end in the anterior muscle of the auricle and the integument of the temple, and communicate with the temporal branch of the upper maxillary nerve near the ear, as well as with (according to Meckel) the auriculo-temporal branch of the lower maxillary nerve. Other branches enter the *occipito-frontalis*, the *orbicularis palpebrarum*, and the *corrugator supercilii muscles*, and join offsets from the *supra-orbital* branch of the ophthalmic nerve."

(*b.*) The *malar branches* cross the malar bone to reach the outer side of the orbit, smaller of the two primary divisions—the *cervico-facial*— is directed obliquely through the parotid gland towards the angle of the lower jaw, and gives branches to the face, below those of the *temporo-facial division*, and to the upper part of the neck. Its branches are named the *buccal, supra-maxillary*, and *infra-maxillary*. In the gland this division of the facial is joined by filaments of the *great auricular nerve* of the cervical plexus, and offsets from it penetrate the substance of the gland.*

One very important point to remember is that the *portio dura* of the seventh pair is the only motor nerve of the *buccinator muscle* for all its actions, whether of expression or of mastication; and that the fifth pair supplies it, not with motor, but with sensory fibres (MAYO, VOLKMANN, LONGET). *The buccinator muscle is interrupted in all its functions, whether of expression or of mastication, whenever the portio dura is paralysed; it is unaffected, and all its actions are preserved, in motor paralysis of the* FIFTH PAIR (SANDERS, *Lancet*, October 28, 1865, p. 478).

The threefold functions of the *portio dura* must also be recognised. Considered as a musculo-motor nerve, it contains within its common trunk the following sets of fibres serving different functions:—(1.) *Voluntary motor fibres*, by which the voluntary movements of the features are performed, and by which especially labial and buccal speech and mastication are accomplished. (2.) *Emotional fibres*, by which the features express the passions more or less involuntarily. (3.) *Reflex motor fibres*, which are involuntary, for the act of winking, and for the movements of the nostrils in the acts of respiration. These different sets of fibres are believed to derive their peculiar functions solely from the nature of their

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and supply the orbicular muscle. Some filaments are distributed to both the upper and lower eyelids: those in the upper eyelid join filaments from the lachrymal and supra-orbital nerves; and those in the lower lid are connected with filaments from the upper maxillary nerve. Filaments from this part of the facial nerve communicate with the malar branch of the upper maxillary nerve.

(*c.*) The *infra-orbital branches*, of larger size than the other branches, are almost horizontal in direction, and are distributed between the orbit and mouth. They supply the *buccinator* and *orbicular oris* muscles, *the elevators of the upper lip and angle of the mouth*, and likewise *the integument*. Numerous communications take place with *the fifth nerve*. Beneath the *elevator of the upper lip* these nerves are united in a plexus with the branches of the upper maxillary nerve; on the side of the nerve they communicate with the nasal, and at the inner angle of the orbit with the *infra-trochlear nerve*. The lower branches of this set are connected with those of the *cervico-facial* division. Near its commencement the *temporo-facial* division of the facial is connected with the *auriculo-temporal nerve* of the fifth, by one or two branches of considerable size, which turn round the external carotid artery; and it gives some filaments to the *tragus* of the outer ear (QUAIN'S *Anatomy*, edited by Drs. SHARPEY, THOMSON, and CLELLAND, p. 613).

* (*a.*) The *buccal branches* are directed across the masseter muscle to the angle of the mouth; supplying the muscles (buccinators), they communicate with the *temporo-facial division*, and on the buccinator muscle join with the filaments of the *buccal branch* of the *lower maxillary nerve*.

(*b.*) The *supra-maxillary branch*, sometimes double, gives an offset over the side of the maxilla to the angle of the mouth, and is then directed inwards, beneath the depression of the angle of the mouth, to the muscles and integument between the lip and chin; it joins with the labial branch of the lower dental nerve.

(*c.*) The *infra-maxillary branches* perforate the deep cervical fascia, and placed beneath the platysma muscle, form arches across the side of the neck as low as the hyoid bone. Some branches join the superficial cervical nerve beneath the platysma, others enter that muscle, and a few perforate it, to end in the integument (QUAIN'S *Anatomy*, edited by Drs. SHARPEY, THOMSON, and CLELLAND, p. 615).

origin and place of central connection in the brain or *medulla oblongata.* In lesion, therefore, of the nerve trunk, in which all the fibres indiscriminately are equally liable to be affected, not only voluntary but emotional and also reflex motions will be suspended. But when the cause of the paralysis is cerebral, the origin or course of certain sets of fibres may alone be involved by the lesion, while others may entirely escape injury. The symptoms, therefore, of central paralysis will vary with the special seat of the central lesion. The voluntary and emotional actions (either or both) which have their origin in the cerebrum, will usually suffer; while the fibres for reflex action, which have their source in the *medulla oblongata,* may be expected to retain their power. The play of the features will be lost, and buccal and labial speech and mastication (so far as the buccinator muscle is concerned) impaired; but the natural position of semi-closure, and involuntary winking of the eyelids, will be preserved. Looking, therefore, at the circuitous windings of the *portio dura,* its intricate distribution, and the various textures through which it passes, it can readily be understood how varied are its tendencies to be involved in disease, and even traumatic injury. Tumors, hæmorrhages, or other lesions may involve the nerve within the cranium. While it traverses the circuitous windings of the *aqueduct of Fallopius* it may be injured and pressed upon by the results of necrosis or caries of the bone, or of suppuration or lesions of the fibrous sheath or periosteum; while lesions or tumors, involving the parotid gland, may injure the nerve and paralyse the parts it supplies.

The accession of facial paralysis (when peripheral) is usually sudden, and is generally discovered by the patient when he begins to eat. He feels something peculiar in the act of chewing, and has some difficulty in mastication. When the food gets between the paralysed cheek and the teeth, the cheek is instinctively squeezed or pressed upon by the hand, in order to push the food between the teeth again. The difficulty of mastication only concerns the buccinator muscle, and not the other muscles of mastication. There is no pain. The patient is soon, and often abruptly, told by the first kind friend who happens to meet him, that his mouth is awry, and that it becomes considerably more so when he laughs. He then naturally wishes to see all this for himself; and on looking at his face in the looking-glass, he may verify the observation of his friend, and is then generally greatly frightened and alarmed by the discovery. When the face remains at rest the paralysed side looks slightly flatter, and more flaccid and pendulous, than the sound side. The eye of the paralysed side is also more widely open than the eye on the sound side. When speaking, and still more when laughing, is attempted, the angle of the mouth on the paralysed side remains perfectly motionless, but on the sound side it is immediately drawn upwards and outwards. The eyelids, the cheek, and half of the lip of the paralysed side remaining thus motionless when efforts are made to contract the muscles, a singular and characteristic expression is given to the face. The eyelids remaining motionless on the paralysed side, the patient is unable to shut the eye, but the globe of the eye itself moves perfectly in any direction at will, which shows that the motor muscles of the eye are not affected, and that the paralysis affects exclusively the *orbicularis palpebrarum* muscle, and does not involve the *levator palpebræ superioris.* Sight is unimpaired. The tongue is protruded with ease and regularity; lingual articulation is sufficient; but articulation of certain words is difficult, on account of the paralysis of the cheek. Sometimes the arch formed by the pillars of the fauces is larger on the paralysed side than on the sound one, by the uvula inclining to the sound side. Cutaneous sensibility is unimpaired, and the patient may be in the best of health, the ailment being purely local—namely, paralysis of the facial muscles of expression on one side. There is an absence of all electric excitability of the paralysed muscles supplied by the seventh pair.

Diagnosis of facial paralysis, differentially, is based on a knowledge of the circumstances under which the paralysis sets in, the progress of its development, and the attending phenomena, bearing in mind the anatomical and physiological history of the *portio dura* already given. Three forms of paralysis of the facial nerves ought to be discriminated in considering the diagnosis of central facial hemiplegia, namely,—*Voluntary Motor Paralysis*, *Emotional Paralysis*, and *Reflex Paralysis* (SANDERS, *Lancet*, October 28, 1865, p. 479). Sometimes the paralysis affects both sides; and one important test is given by Marshall Hall by which it may be known whether the cause of double facial paralysis is seated in the brain or in the course of the nerves. If the lesion is central (cerebral) the conducting power of the nerve trunks is retained for an indefinite period, so that by galvanising the trunk and the principal branches of the facial nerves, all the paralysed muscles supplied by the nerves are thrown into contraction, as if the muscles themselves were being galvanised; whilst, when the paralysing lesion is *in the course of the nerves*, the nerves very easily lose their conducting power. If reflex movements be seen in the paralysed muscles, the cause of the paralysis is assuredly a lesion of the nerve centres. The most distinguishing characteristic of paralysis due to a *cerebral lesion* is considered by Trousseau to be the *absence* of complete paralysis of the *orbicularis palpebrarum*. "However complete hemiplegia of cerebral origin may be, I have never seen *complete paralysis of the orbicularis palpebrarum—the eye can always be closed;*" while in Bell's paralysis, the palsy of the *orbicularis palpebrarum* is never absent, and the eye cannot be completely closed (TROUSSEAU). This coincides with the experience of Dr. Sanders. There is, however, a slight modification to be acknowledged here, that "although the patient with cerebral hemiplegia can close both eyes simultaneously, he cannot voluntarily close the eye on the same side as the paralysis while the other remains open" (BAZIRE). But it appears to me that this would only show *some extent* of paralysis, if the patient were known to have been able to close either eye at will previous to his attack—a feat which many, especially females, are unable to perform who are free from any paralysis. From the threefold functions of the *portio dura* already noticed, and the diverse origins of its several sets of fibres,* it will easily be understood how, in very limited and localised cerebral lesions, *all* the branches of the facial nerve may not be affected. The extent of apparent paralysis in cases of cerebral facial hemiplegia is not usually so great, and therefore not so alarming to the patient, as in cases of Bell's paralysis, where the trunk of the nerve only is affected. But the prognosis must be more unfavourable. In ordinary facial paralysis of *cerebral origin*, Dr. Sanders has always found that the muscles of expression, including the buccinator, were more or less affected, while the action of the masseters, temporals, and pterygoids was unimpaired. The usual facial distortion was exhibited to a greater or less degree, according to the amount of the paralysis, but it rarely approached the completeness usually seen in peripheral paralysis from lesion of the nerve trunk. With the exception of the slightness of implication of the *orbicularis palpebrarum* muscle, the phenomena of cerebral hemiplegia of the face are entirely similar to those of facial paralysis produced by lesion of the seventh nerve itself, and, as in the latter, are confined to the muscles of expression, including the buccinator, and do not involve the masticatory muscles (masseter, temporal, pterygoid) supplied by the *motor fifth.*

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* The seventh pair of nerves appear on each side, at the posterior margin of the pons, between the middle and inferior peduncles of the cerebellum, and nearly in a line with the place of attachment of the fifth nerve.

The *portio dura*, or facial nerve, placed a little nearer to the middle line than the *portio mollis*, may be traced to the *medulla oblongata*, between the *restiform* and *olivary fasciculi*, with both of which it is said to be connected. Some of its fibres are derived from the pons. Phillipeaux and Vulpian affirm that the fibres arise from the outer wall of the fourth ventricle, and that many of them decussate in its floor.

Another point of diagnosis is to be recognised in the behaviour of the paralysed muscles under electric irritation. In facial paralysis of cerebral origin, the muscles respond *normally* to electric irritation; but if the paralysis is due to a lesion of the *portio dura*, their contractility is not at all, or scarcely at all, roused by an electric current (DUCHENNE, SANDERS, VULPIAN, TROUSSEAU). *The symptoms of cerebral facial paralysis* vary as the central lesion is more or less distant from the origin of the seventh pair. There is therefore much more variety in the expression of the face (according to the varying extent of the paralysis) than in cases of Bell's paralysis. The peculiar aspect of the face in *cerebral facial hemiplegia* is due to the want of symmetry between the two halves. The contraction of the muscles on each side is not co-ordinate. The sound cheek is wrinkled and shortened. The labial commissure on the sound side is drawn outwards and upwards, and is on a higher level than on the opposite side; the angle of the mouth on the paralysed side is lower than its fellow; and if the paralysis is extreme, the commissure on the paralysed side remains partially open, so that the saliva constantly escapes. The flaccid cheek is the result of paralysis of the buccinator. The nostril on the paralysed side is more closed than on the sound side; but "in the great majority of cases of cerebral hemiplegia of the face, the *orbicularis palpebraris* is not materially affected; the act of winking and of voluntary closure of the eyes continues on the paralysed as on the sound side, with the small exception that the voluntary closure is usually weaker on the palsied side.

"These phenomena furnish the best diagnostic marks between *centric* and *peripheral* paralysis of the face; the hanging cheek, with wide open, staring, unwinking eye, denotes lesion of the *portio dura;* the flaccid face, with the natural position and motion of the eyelids, is the sign of

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Connected with the *portio dura*, and intermediate between it and the *portio mollis*, is a smaller white funiculus. The roots of this accessory or intermediate portion are connected deeply with the lateral column of the cord (QUAIN'S *Anatomy*, Edited by Drs. SHARPEY, THOMSON, and CLELLAND, p. 587).

cerebral lesion, and indicates a more serious disease" (RECAMIER, TODD, SANDERS).

There is difficulty in the articulation of labial consonants and vowels. The tongue protrudes in the normal direction, and if its point seems to diverge from the median line to the paralysed side, it only seems to do so in consequence of the commissure of the lip being drawn in the opposite direction—namely, AWAY FROM the paralysed side. But sometimes the tongue, too, is paralysed and really deviates, and then *it is drawn to* the sound side, when the branches of the *portio dura,* going to the *stylo-glossus* and the *genio-glossus,* are implicated. Opinions differ greatly as to the implication of the tongue, the uvula, and the soft palate in cases of cerebral facial hemiplegia. Trousseau, Todd, Hasse, Longet, Romberg, Bidder, and Sanders, all refer to such cases. Dr. Sanders shows that the position of the uvula varies frequently, both in the natural and hemiplegic palate. The only reliable sign that the palate is involved in the lesion of the *portio dura* is the existence of a vertical relaxation, or lowering of the (paralysed) corresponding half of the *velum palati,* with diminished height and curvature of the posterior palatine arch on the paralysed side (SANDERS). Such cases indicate lesion before the geniculate ganglion in the Fallopian duct, and implicating the petrosal nerves.

In both forms of facial paralysis the action of spitting is difficult, so also is whistling, as the patient cannot close the mouth nor inflate the cheek. A coachman was very thankful to Sir Charles Bell for the successful extirpation of a tumor near his ear; but complained that ever after the operation he could not whistle to his horses. No doubt the *portio dura* was cut in the operation. Mastication is badly done, but only so far as the buccinator is concerned, because the food accumulates between the paralysed cheek and the teeth. The hand is often seen to be held almost instinctively against the cheek to give it support, and to press the food between the teeth for proper mastication, which is efficiently performed by the *temporals, masseters,* and *pterygoids.* In some cases of facial paralysis it has been noticed that hearing on the affected side becomes much more acute; and the taste is sometimes perverted, both indicating lesion high up within the bony canal traversed by the *portio dura* (TROUSSEAU, ROMBERG, BROWN-SÉQUARD, LANDOUZY, BAZIRE).

A peculiar and characteristic result of facial paralysis is the contracture and tonic spasms, or convulsions, of the muscles of the face, which have been paralysed—the "*spasmodic tic*" of Marshall Hall. Simple contraction of the muscles is very common after Bell's paralysis, when it has lasted a long time and been extreme. It comes on after the affection appears to be cured, and after the symmetry of the face has been apparently restored. At first, and most usually, the contraction is partial, and is mixed up with some degree of paralysis, the contraction of the muscles not being effected at will. This contraction is common to all muscles which have been paralysed and out of use for some time. The spasms are sometimes painful, and the contracted muscular masses are painful on pressure. Sometimes one muscle and sometimes another is thus affected. Sometimes it is the orbicular muscle of the eyelid, when the eye, instead of being more open, as it was when paralysed, now looks smaller, and is more closed. Such contraction is a most frequent termination of paralysis due to *rheumatism.* A rapid return of tonicity in a paralysed muscle, the electro-muscular contractility of which had been completely abolished, is always a sign of approaching contraction (DUCHENNE).

Prognosis.—Bell's form of peripheral facial paralysis generally gets well in time, and all the more rapidly that it sets in suddenly, and the patient young. From four to ten months is the ordinary duration of the affection. There are instances, however, in which the paralysis yields in twenty-four, fifteen, or even twelve hours, but such cases are exceptional (TROUSSEAU). The prognosis in *central facial hemiplegia* is less hopeful; so also in *double facial hemiplegia;* because the cause indicates—from the symmetrical nature of the exciting lesion (generally periostitic)—some constitutional source as the origin of the mischief—*e.g.*, scrofulosis or syphilis.

Treatment.—Paralysis due to lesions which are destructive of the nerve are beyond medical aid. In cases suitable for treatment, and within the first few days, the agents used with most benefit are,—Blood-letting by leeches behind the ear of the affected side, or over the mastoid region. They ought to be applied repeatedly, especially if any pain exists on pressure in that region. Blisters, with dressings to keep them open, medicated by veratria or strychnia ($\frac{1}{25}$ to $\frac{1}{5}$ of a grain *sulphate of strychnia* or of *veratria*), mixed with five or six times their weight of pounded sugar. Electrisation in the form of faradisation by a Stöhrer's two-celled volta-electric machine; or in the form of an *interrupted galvanic current*, from a *continuous current* battery (as from a Muirhead's battery of fifteen cells), the number of cells being very gradually increased, are agents of much service.

In using faradisation each facial muscle should be separately influenced, instead of attempting to pass the current through the facial nerve. The muscles are thus more powerfully influenced, and they are more apt to regain *voluntary* contractility at different periods, some muscles before others. At first the apparatus should be one capable of producing currents with very rapid intermissions; but when any muscles begin to *contract*, the intermissions should be few, and the sittings at long intervals (three times a week, for two or three minutes only at a time), otherwise contraction of the muscles is apt to increase and become incurable. The voltaic current, from a continuous current battery, induces contractions when the circuit is closed and when it is opened, and more powerfully at the moment of closure. It is said to restore to palsied muscles the power of contracting under the influence of faradisation. When improvement follows the use of the voltaic current from a continuous battery, the number of the cells should be gradually increased as the muscles get less susceptible to their influence, and then the battery may be used oftener. One of the electrodes should be gently moved along the surface of the muscle, just as the brush is used in faradisation. During the first weeks it may not be used oftener than once weekly, commencing with six to ten of Stöhrer's elements.

When contracture of muscles supervenes, they ought to be stretched or pulled out mechanically. To remedy contraction of the buccinator, Duchenne recommends the use of a small billiard ball worn for a long time inside the cheek, to be replaced after a time by a larger one.

The late Dr. Bazire gave favourable testimony to the value of this mechanical aid.

The daily action of the bowels ought to be carefully regulated, especially by such medicines as keep the colon in functional activity. *Bichloride of mercury* (*corrosive sublimate*) in small doses, continued till the gums are slightly tender, is sometimes of service. In cases where rheumatism prevails, or periostitic affections, small doses of *iodide of potassium*, persevered in for a lengthened period, are of service, with the frequent use of *phosphate of soda* as a daily morning aperient.

IV. *Paralysis of the muscles supplied by the nervus accessorius Willisii* (spinal accessory), especially the sterno-cleido mastoid and trapezius, may arise from rheumatism, or from disease of bones, from tumors, swellings of lymphatic glands and abscesses, or from neuritis, or wounds and injuries. In unilateral paralysis of the *sterno-cleido mastoid*, the head assumes a slightly oblique position. The chin is turned to the affected side and somewhat elevated. Persistent obliquity follows contracture of the healthy muscle. In paralysis of the *trapezius* the deformity is expressed by the scapula being drawn downwards and forwards. Its inner border is drawn from the vertebral column and placed obliquely, whilst its inferior angle remains relatively nearer to the vertebral column. The acromion process falls downwards and forwards. The clavicle projects, and the supra-clavicular fossæ appear deeper than natural. Voluntary elevation of the shoulder is limited.

V. *Paralysis of the muscles supplied by the cervical and dorsal nerves*:—*Paralysis of the serratus muscle* is one of most frequent occurrence. The muscle is supplied by the *long thoracic nerve*, and receives no other branches; the exposed position of which, and length of its course, renders it liable to many injuries, such as from carrying loads on the shoulder, blows and concussions, or from over-exertion, as in mowers, puddlers, shoemakers, and ropemakers. Exposure to cold is also a frequent cause. Its paralysis is often a symptom of progressive muscular atrophy; and is often preceded by neuralgic pains in the region of the supra-clavicular branches of the brachial plexus. At rest, with the arm hanging, the scapula is raised and approximated to the vertebral column, and so rotated that its inferior angle approaches the vertebral column; its anterior border is inclined downwards, while its inner border is directed obliquely upwards and outwards. The scapula thus seems to stand out like a wing at the inner border and inferior angle. Certain movements at the same time become difficult. The arm cannot be raised above the horizontal level. It is also difficult to cross the arm in front of the chest, and to bring the apex of the shoulder forward. Respiration is not affected. It only very gradually terminates in recovery, unless in some traumatic cases where destructive lesion renders recovery impossible; and in the most favourable cases the muscle long remains feeble.

VI. *Paralysis of muscles of inspiration* is of importance mainly in connection with *paralysis of the diaphragm*, which may result from inflammation of its serous investments, as in pleurisy and peritonitis, the inflammation extending from those coverings to the muscle. It may also attend progressive muscular atrophy, and has been noticed as a consequence of lead-poisoning (DUCHENNE). It occurs during adolescence without any apparent cause (OPPOLZER). *The symptoms* are characteristic. During inspiration, especially deep and long, the epigastrium and hypochondria are drawn inwards instead of being curved outwards; in expiration the epigastrium projects. Dyspnœa occurs on the smallest exertion, to the extent of forty or fifty respirations in a minute.

VII. *Paralysis of muscles supplied by the circumflex nerves.*—These are especially the deltoid and teres minor. Injuries, such as concussions and blows, are a frequent cause. Rheumatism and chronic inflammation of the shoulder-joint also cause paralysis of the deltoid, from a neuritic process extending along the circumflex nerve. There is then much pain in the deltoid muscle and in the joint. The arm cannot be raised; it lies flat against the side, from which it is difficult to separate it; and when the arm is lifted up the deltoid remains relaxed, and eventually becomes atrophied; and the joint becomes so relaxed and loose, that a deep groove can be felt through the atrophied deltoid, between the head of the humerus and glenoid surface of the scapula. Electrical excitability may be normal at first, but it afterwards undergoes diminution.

VIII. *Paralysis in the region of distribution of the radial or musculo-spiral nerve*, is of most frequent occurrence. One of its most common causes is pressure on the nerve, as when one falls asleep with the arm hanging over the back of a chair, and the nerve is compressed. The paralysis appears as soon as the patient awakes. Blows on the arm may also produce a similar result. Sleeping "on damp earth," or near a "moist wall," or exposure to a draught of air, are also generally combined with compression on the nerve in some part. There are numbness and tingling, extending to the tips of the fingers—"pins and needles." Lead-poisoning is one of the most frequent causes of this paralysis—appearing in the later stages of the poisoning. It commences almost without exception in the *extensor communis digitorum*, subsequently extending to the other muscles supplied by the musculo-spiral nerve, followed by "dropping" of the hand and incapability to extend the fingers. But the *supinator longus* is never implicated in lead-poisoning (DUCHENNE). In complete paralysis of the musculo-spiral nerve, the hand is kept in a state of flexion. It hangs flaccid, and cannot be raised or extended. The fingers are flexed, and cover the thumb, which is flexed and adducted. The fingers cannot be extended; and neither the thumb nor index finger can be abducted or extended. When the arm is extended (in order to exclude the action of the biceps), supination of the forearm cannot be effected on account of the paralysis of the *supinator brevis;* neither can it be bent and half supinated by the paralysed *supinator longus.* If the hand be laid flat on the table, no lateral movements can be made, nor can it be raised from the surface of the table. The hand is quite useless, so that the patient can neither hold nor grasp anything. There is generally also more or less intense anæsthesia in the region supplied by the musculo-spiral nerve.

IX. Other forms of palsy, such as those from the action of certain poisons, will be considered under the respective poisons which produce them.

Treatment of most of the forms of local paralyses is mainly by the continuous voltaic current to the muscles when the acute symptoms have passed away, and when no fever or local signs of inflammation exist. The use of cutaneous faradisation, on a dry surface with a feeble and slowly intermittent current, has also been strongly recommended by Duchenne. Friction, stimulant applications, and blisters are also of use.

The consideration of the subjects numbered 5, 6, 7, and 8, at page 99, will be reserved till the diseases of the brain, of the cord, and of their meninges have been described, in connection with which two important symptoms may be noticed here, namely:—

Delirium embraces wanderings of the mind, which cannot be fixed, and varies from mere confusion of thought to fixed delusion. It is shown by incoherence of expression especially. It is generally combined with some degree of unconsciousness. Its relation to coma must be carefully inquired into. There is generally restlessness, prompting the patient to get out of bed. It is generally most expressed at night. The conditions under which it occurs must be constantly kept in view. They are,—(1.) During the natural course of specific fevers; (2.) alcoholic poisoning, to which the name of *delirium tremens* is given; (3.) alterations of the blood during acute inflammations, as in *pneumonia* and *acute hepatitis, carditis, erysipelas;* (4.) in the course of acute constitutional diseases—*e.g.*, *gout* and *rheumatism;* (5.) in connection with cerebral conditions, as headache attending *tubercular meningitis,* especially in youth and adult age; and generally in inflammatory affections of the *meninges* of the brain, and in *acute mania.* It is always a sign of nervous disturbance. The slighter forms occur when the patient is not quite awake, so that he talks as if asleep, and in a dream. His ideas are "wandering," and in the confusion of the dream he introduces them into his conversation, and is conscious of his mistake. In severer forms of delirium he is quite unconscious of any confusion of thought or expression, and may become violent. *Sleeplessness* is of importance chiefly as associated or not with delirium, and if it has gone before it. The account given by a patient as to sleeplessness is often incorrect.

Coma, unconsciousness, insensibility, stupor, sopor, are names significant of that condition where consciousness is more or less entirely suspended, with the appearance of profound sleep, from which the patient may sometimes be partially aroused. The condition varies in intensity from profound coma or complete unconsciousness to partial coma or stupor. A history may be obtained as to whether the condition is of sudden onset, with or without convulsions, or has come on gradually with increasing stupor and somnolence; and whether there is any obvious cause. It is necessary to keep in view the causes or conditions under which coma may occur. They are,—(1.) *Injury to the brain*, such as by compression or concussion. Evidence of fracture of the skull is to be looked for; and the ears, orbits, nasal passages, and mouth cavity must be very carefully examined—

especially for evidence of any foreign body hidden in them by which injury to the brain or its membranes has been brought about. If *hemiplegia* is present, extravasation of blood exists, and coma will then be most profound. If such *coma* comes on suddenly, without any injury and without convulsion, or very slight convulsive movements, the condition is due to apoplexy. (2.) *Blood-poisoning*, such as from *alcohol*, *opium*, *codeia*, *conium*, or other narcotics; or from such morbid poisons as occur in certain diseases, such as *uræmia.* The odour of the breath and of the patient is of much importance; also the degree of consciousness, and its mode of coming on. It commences very insidiously in cases of general ill-health, when it generally points to albuminous urine, with urea, in the blood. The urine ought to be carefully tested in such cases; and the amount passed before coma came on should be ascertained if possible. (3.) *Effusion of serum into the ventricles* causes coma, which is generally *preceded* by convulsive spasmodic movements, and the coma is more or less incomplete. (4.) *Tumors, brain softening, embolism ;* and in all cerebral diseases, *coma* is of common occurrence with or without convulsions or paralysis.

INFLAMMATION OF THE CEREBRAL AND SPINAL DURA MATER; PACHY-MENINGITIS.

Pathology.—In describing the diseases of the parts contained within the cranium and spinal canal, however convenient it may be to consider the several structures separately, their pathology must be, nevertheless, studied together.

Inflammations of the cerebral and spinal *dura mater* comprehend the following pathological conditions:—(1.) *External cerebral pachy-meningitis*, or inflammation of the outer layer of the *dura mater* of the brain: (2.) *Internal hæmorrhagic pachy-meningitis*, or hæmatoma of the *dura mater:* (3.) *External spinal pachy-meningitis*, or inflammation of the external layer of the *dura mater* of the spinal cord: (4.) *Internal spinal pachy-meningitis* (5.) *Cerebral pia-arachnitis* (*lepto-meningitis*), inflammation of the *pia mater* with arachnoid of brain: (6.) *Spinal pia-arachnitis.*

(*a.*) *Causation.*—The *dura mater* may be regarded as the periosteum of the skull bones; and is mainly affected in connection with the diseases and injuries of these bones, rather than with diseases of the brain. It is a source of protection to the arachnoid and to the brain; and its external relations towards the bones may be diseased and injured without the brain suffering. Hence, inflammation of the *dura mater* is either of *idiopathic* origin, or it is the result of the extension of diseases from parts external to it. As a rule, it is not found diseased except in cases of fracture of the cranium or necrosis of the bones. In cases of disease of the ear and caries of the temporal bone (petrous or mastoid portion), the contiguous *dura mater* may be implicated, when it appears of a dark colour and sloughy; or pus may be infiltrated so as to separate the *dura mater* from the bone, thus inclosing the pus as in an abscess. The danger in such cases is due to implication of the sinuses and purulent infection. Similar lesion may be associated with disease of the ethmoid, sphenoid, or orbital parietes; or with syphilitic or scrofulous growths affecting the bones of the skull; or with caries of the vertebræ, or bed-sores on the sacrum, opening to the *thecæ vertebralis.* The inflammation may also spread, in a secondary way, by continuity with the surface of the head externally, when erysipelatous or other inflammation exists there.

Inflammation of the *dura mater* of the cord probably never occurs as an independent disease.

(*b.*) *Morbid Anatomy.*—The anatomical relations of the arachnoid and *dura mater* must be kept in view in all interpretations of the pathology of these parts (see p. 5, *ante*). A reflection of the arachnoid on to the *dura mater* does not exist, so that what has been known as the arachnoid sac is better named the *subdural space.* When the *dura mater* becomes perforated from inflammatory overgrowth or suppuration, the pus may find its way into the subdural space, and lie on the outer aspect of the arachnoid. If true arachnitis is thus set up, the lesion invariably originates from the *dura mater* and skull, in the ways already mentioned. It never springs up as a spontaneous disease; and when found on *post-mortem* examination, it suggests the necessity of finding out some such primary lesion external to it. Whenever, therefore, there is lymph or pus in the *subdural space,* its source or cause must be looked for in the *dura mater,* the skull bones, the pericranium or skin covering the head. There are, however, such exceptional cases, as when pus extends from the spinal canal, as in cerebro-spinal meningitis. In the *theca vertebralis* the products of inflammation accumulate between it and the bones; in the first instance between them and the vertebral ligament.

The rachidian *dura mater* may be inflamed, either at its free or at its adherent surface. On examining the spinal canal after caries of the vertebræ, the areolar tissue uniting the *dura mater* to the walls of this cavity is often found greatly loaded with venous blood, and in some instances is broken down, so that the *dura mater* is entirely detached—a state most probably due to inflammation. This inflammation may terminate by resolution, or it may advance, and serum be effused between the osseous structure and the *dura mater.* In this site the effusion has no communication with the cavity of the cranium, because the *dura mater* of the cord, while it is but loosely attached by areolar tissue to the vertebræ, is very firmly attached round the margin of the *foramen magnum,* and especially to the basilar portion of the occipital bone.

When the *theca vertebralis* becomes involved in the process of inflammation, it becomes thickened, excessive local germination takes place, which finally compresses the cord. Thus the inflammation occurs generally in more or less circumscribed patches. Injection, infiltration, and softening, are the usual results, which may go on to suppuration, or to permanent thickening of the membrane, which becomes adherent to the bone; or the pus may make its exit through the membrane, when *diffuse meningitis of the cord* supervenes.

Internal hæmorrhagic pachy-meningitis, or hæmatoma of the dura mater. These are cases of a chronic hæmorrhagic nature. The lesions are characterised by the presence of certain layers of false membranes, with more or less altered blood in them, attached to the *dura mater*—sometimes so closely as to thicken it. Hence the lesion is sometimes described under the name of "*pachy-meningitis.*" In the theca vertebralis it usually occurs towards the cervical enlargement. There are other forms of the lesion in which effusions of recent blood exist between layers of false membrane, so as to make a blood tumor—a *hæmatoma* of the *dura mater.* They thus form sanguineous encapsuled flattened masses, composed of fine layers of fibrine, spread to a greater or less extent over the under surface of the *dura mater,* accompanied by repeated small extravasations of blood, which are converted into pigment. By repetition of the process several layers come to be deposited one upon the other. Numerous and large blood-vessels form in these layers; and from these vessels renewals of the hæmorrhages occur. The condition is chronic, and terminates, after continued cephalic suffering, generally suddenly, with symptoms of apoplexy. Blood, therefore, either recent or old, or both, co-exists in these lesions *with* false membranes, such as are seen in serous inflammations. It is, in fact, an extensive encapsuled collection of blood on the under surface of the *dura mater* in the subdural space. As to its origin, it was believed to be simply an extravasation of blood, at the periphery of which the fibrine became precipitated and the fluid part encapsuled. But the question still remains, "as to whether the blood causes the false membrane by inflammation; or the false membrane, which is vascular, bleeds, and so accounts for the extravasated blood." One view as to their origin is maintained by Virchow and Weber, and is very generally accepted—namely, that the inner surface of the *dura mater* produces, under some irritation, a layer of inflammatory false membrane, which becomes highly vascular. New layers continue to go on forming, and becoming vascular—meanwhile hæmorrhagic exudations take place, the blood coming from the large thick-walled capillaries that form in the membranes—the old layers contract and obstruct the returning circulation through the vessels of the superficial layer. Hence these burst, and cause the effusion of blood between the layers. By repetition of the inflammatory process, numerous layers of fibrine thus become deposited one upon the other; and much more numerous and larger blood-vessels form in these layers than are to be met with in the *dura mater* itself. From these new-formed vessels the hæmorrhage proceeds which gives rise to the formation of the *hæmatoma*, and its cystic inclosure is formed by the extravasation taking place between the layers of the false membrane (VIRCHOW). The lesion is comparatively rare, and was at one time believed to have been the result simply of an apoplectic effusion of blood, at the periphery of which the fibrine had been precipitated, and the whole encapsuled. The lesion is now believed to be the remains of repeated chronic inflammations of the *dura mater*, with hæmorrhagic exudations. The blood comes from the large thick-walled capillaries which form in the *pseudo*-membrane deposited in the *dura mater* during the chronic inflammation, and the blood is effused between the layers of this exudation. Old age (above fifty), disorders of the intellect, especially general paralysis, chronic alcoholism, and forms of Bright's disease, are the kind of cases in which the lesion is found; and sometimes it seems traceable to external injury. In such cases years may intervene between the injury and the symptoms of hæmatoma (GRIESINGER). Of several specimens of the lesion preserved in the museum of the Army Medical Department at Netley Hospital, one occurred in a soldier thirty-three years of age, with tropical service, a history of epilepsy, fever, and lunacy, and finally death with apoplectic symptoms; a second occurred in a soldier twenty-seven years of age, with a history of epilepsy; a third in a soldier thirty-eight years of age, also with a history of epilepsy, tropical service, chronic alcoholism, injury by a fall into the trenches at St. Mary's Barracks, Chatham, and death from paralysis. Cases are also chiefly met with amongst the inmates of workhouses, lunatic asylums, and hospitals for the incurable.

When the effusions are large, the false membranes become distended into the form of a cyst. The smaller effusions are formed long after, as old and altered blood pigments. Such false membranes are also caused by effusions of blood quite apart from inflammation, the process being hæmorrhagic from the commencement (HUGUENIN), due to degeneration and rupture of veins, proceeding from the surface of the brain to the longitudinal sinus. Thus, when a considerable extent of *dura mater* is lined by a membrane, composed of an adherent but separable layer, of a brick-dust colour, it may be ascribed to old effusions of blood. In their microscopic structure they are characteristic of "*pachy-meningitis*"—a wide capillary mesh-work of long tortuous vessels, showing varicose dilatations in many parts. These meshes are supported by a delicate texture of spindle-cell connective tissue, in which are numerous crystals and glomerules of *hœmatoidin.* These give the red colour to the membrane. Hæmorrhage, therefore, must be regarded as generally the first step in the process. Such hæmatoma are usually found near the sagittal suture, with the long diameter parallel to the falciform process, having the form of an oval flat sac. It may attain the size of 4″ or 5″ × 2″ or 3″, and half an inch thick. The walls of the sac are of a dirty colour, from the altered colouring matter of the blood. The contents of the capsule are partly fresh fluid or coagulated blood, and partly dirty red-brown clots of an ancient date. The brain below such lesion is flattened, and may even show a depression. They may exist on both sides of the head.

In *syphilitic inflammation of the dura mater* the outer surface of the membrane and the bone are generally affected together. The tendency of these lesions is to extend inwards, causing adhesion of the *dura mater* to the brain by the arachnoid at the affected spot, and sometimes to excite suppurative arachnitis. A fleshy swelling, pink or red, may result from congestion and vascularity of the new products. This exudation very soon becomes caseous in its centre, and occasionally it very much resembles a sarcoma.

Morbid growths in the dura and subdural space.—(1.) Bone is the most frequent production, and the *falx major* the most common site. Microscopically the structure is that of true bone—the lacunæ and canaliculi being always demonstrable. These bony lesions of the *dura mater* are very different from the so-called ossifications of the heart and blood-vessels, which consist of earthy concretions simply, and are to be regarded as cretifications or petrifactions. (2.) *Cancer of dura-arachnoid* in many instances appears to commence in the *dura mater*, and then to penetrate the bone. Hence its existence may not be suspected till the calvarium is attempted to be removed, when the cancerous matter is opened into and exposed. It is then seen to be made up of round soft patches on the membrane, and on the corresponding surface of the skull—the bone is seen to be eaten away as to its internal table, its cavities being occupied by the same cancerous matter. In the greater number of cases, however, the cancerous tumor more properly is developed in the bone, and frequently co-exists with general cancer of the skeleton.

Symptoms.—(1.) In connection with diseases of the ear, inflammation of the *dura mater* is sometimes set up by exposure to cold, or a blow on the diseased ear; or it occurs insidiously without any obvious cause. The discharge from the ear ceases or diminishes. Intense pain on the ear, or in its vicinity, occurs, or severe headache. The pain is constant, with extreme exacerbations. Convulsions may be the earliest indications of cerebral irritation; or vomiting (page 89, *ante*), vertigo (page 142, *ante*), incoherence, rambling, delirium, or a rigor. In *pachy-meningitis of the cerebral dura mater* and hæmatoma, there is more or less weakening of the memory and of the intelligence, the occurrence of giddiness, and of continuously intermittent general or local pain in the head. At a later period an aggravation of all these phenomena occurs, with transitory losses of consciousness from the momentary arrest of cerebral circulation. Somnolence and apathy prevail, with weakness, and generally one-sided paralysis of the extremities, with occasional epileptic or apoplectic attacks, in one of which the patient may die. The symptoms of inflammation of the *dura mater* of the cord are often obscure at the commencement. The disease is characterised by pains in the back, with affection of the muscles, which are rigid, and sometimes retention of urine. The pain is liable to great aggravation from movement of the limbs or trunk, or of the head or neck. There may also be some loss of motor power, and also of sensation and loss of control over the bladder and rectum. The phenomena of the different forms and extent of hemiplegia which may result have been already considered. (See page 354, *ante.*)

Treatment.—Circumscribed suppurations, when recognised, must be dealt with by the surgeon, under rigid antiseptic precautions. Evaporating lotions or ice to the head or spine are also indicated. Free action of the bowels (especially of the lower bowel by aloetic laxatives) should be maintained. Circumstances may also indicate *iodide of potassium*, and pain is best relieved by large doses of *opium* or of *morphia*. The patient ought to be kept perfectly at rest.

CEREBRAL AND SPINAL MENINGITIS.

LATIN EQ., *Meningitis;* FRENCH EQ., *Méningite;* GERMAN EQ., *Gehirnhautentzündung*—Syn., *Meningitis;* ITALIAN EQ., *Meningitide.*

Definition.—*Inflammation of the immediate coverings of the brain and spinal cord, the pia mater, and arachnoid (pia-arachnitis).*

Pathology.—(*a.*) *Causation*, (1.) Of Cerebral Meningitis.—The inflammation may be simple and idiopathic, or it may supervene as the result of some specific or constitutional disease, such as the specific fevers, or of the *syphilitic, rheumatic, gouty, rachitic, or scrofulous* cachexiæ. It is also known to occur in convalescence from *pneumonia* and *pleurisy*, or from *acute* *exanthemata* and *erysipelas*, from protracted diarrhœas, and especially also as intercurrent with Bright's disease. Every age is liable to meningitis. Children are often attacked by it whilst teething, under the form described as *hydrocephalus acutus*, and also when labouring under *scarlatina*, *measles*, or other disease caused by a specific poison. Adult age, and the middle periods of life, are still more liable to this affection, both from the greater exposure to the action of the typhus, typhoid, and paludal poisons, to syphilis and to mechanical injuries, as well as to the greater intemperance and greater excitement incident to this age. In old people meningitis is likewise common; and two forms of the disease are met with in the aged—namely, the *acute* and the *chronic*, both of which present great varieties in their character and progress, and both sexes suffer in nearly equal proportions.

In an epidemic form *cerebro-spinal meningitis* is a disease which occurs from the action of some specific morbid poison, and it is then believed to be contagious (p. 577, Vol. I.). There are many instances also of persons suffering from *pia-arachnitis* after exposure to the heat of the sun, or to what is in common language called the "*coup de soleil*" (see under "Sunstroke," p. 314, *ante*). Intemperance, as well as great mental work and anxiety, are frequent causes of the chronic forms of the disease; but the abuse of alcoholic liquors is perhaps the most common predisposing cause; while fits of intemperance occasionally directly induce the disease. It is especially apt to be associated with or to follow *delirium tremens* in the aged. It is also especially connected with *insanity*, and with every structural disease of the brain. To these causes must be added mechanical injuries, diseases of the bones, and morbid growths, especially in cases of primary implication of the *dura mater*.

(*b*.) *Morbid Anatomy.*—The products of inflammation are formed in connection with the *pia-arachnoid* and subarachnoid space. The lesions are those which attend some of the most important acute diseases of the brain. The effusion appears beneath the arachnoid, and immediately in connection with the cerebral surface. The effusion, be it lymph or pus, comes from the *pia mater*, and follows all the inequalities of the convolutions, lying beneath the level surface of the arachnoid membrane. In such a morbid condition of the *pia mater* the brain-structure is always more or less affected, and the ventricles suffer, for into them the choroid plexus of the *pia mater* carries with it the inflammation, by simple continuity of texture.

Pia-arachnitis, or *acute meningitis*, as it is commonly called, is mainly of two kinds:—(1.) *Simple*, (2.) *tubercular*, sometimes called "*acute hydrocephalus*." It is of importance to distinguish between these two forms, the peculiarities of each being characteristic.

In simple pia-arachnitis (or simple meningitis) the surface of the brain n the space beneath the arachnoid is covered with lymph, or with purulent lymph of a green colour. This is seen to be most in the sulci between the convolutions, and may be so abundant as quite to hide the cineritious structure; so that when the *dura mater* is removed, both hemispheres are to be seen completely covered with a layer of green purulent lymph. This effusion is seen and felt to be underneath the arachnoid, so that it cannot be removed, or got at without cutting through this delicate membrane. On scraping the external surface of the arachnoid, a small quantity of matter may appear on the scalpel, which, on microscopic examination, is found to consist merely of epithelium and granular *débris*. The locality of the effusion is generally defined and limited to the vertex surface of the brain, disappearing towards the sides, which are covered to a less degree; while at the base there is generally very little effusion. The fluid in the ventricles is increased and somewhat turbid. If the *pia mater* is removed from the convolutions, much of the cineritious substance of the brain *sticks* to it.

A microscopic examination of the smaller vessels of the *pia mater* in *meningitis* shows that they are studded with the exudation-corpuscles described by Gluge and Bennett, and with oily-like vesicles; and these appearances are found both within and without the vessels (SIEVEKING). Lymph, varying in density and thickness, is the form which the exudation ultimately assumes, if life continues, dipping down between the convolutions. It is most abundant on the upper portions of the hemispheres. Foville has met with cases in which the effused lymph covered the whole of the brain, or nearly so, as far as the *tentorium;* deposited in two layers—one in the subdural space, and the other adherent to the cerebral arachnoid; while between them was a stratum of serum. He mentions six cases of this description under his care for several years, and that they were all in a state of the dullest stupidity, labouring under a paralysis of every sense. They were like statues, with this difference, that, placed upright, they preserved their balance; if pushed, they walked; and if food was placed in their mouths, they swallowed it. Were these cases of "paralysis of the insane," as now so-called? (Art. "Méningite," p. 406, *Dict. de Médicine*). Lymph also may be effused into the arachnoid cavity, but it is generally in small quantity.

Chronic thickenings of the pia-arachnoid are chiefly in the form of thick, white, nodulated bands along the course of the longitudinal fissure, with more or less white opacity over the vertex, extending for perhaps an inch on each side. Sometimes such thickening extends along the Sylvian fissure, with patches on the posterior lobes corresponding to the entrances of veins into the lateral sinuses. In the thickened membrane over the vertex, the Pacchionian bodies are more numerous than usual, forming white villous masses—most developed at the entrances of the veins which join the sinus most posteriorly.

A peculiarity is to be noticed in the anatomical connection of the arachnoid in its visceral portion—namely, that in the greater part of its extent it is thrown loosely round the parts which it covers, a few fine fibrous bands being the sole bond of connection; and a quantity of pellucid fluid is interposed, especially in the vertebral canal and base of the cranium, between the arachnoid and *pia mater*.

The *spinal arachnoid* and *pia mater* are liable to inflammations similar to the corresponding membranes of the brain. Effusion of serum, both into the cavity of the *spinal arachnoid*, and between the *dura mater* and *pia mater* of the cord, is not uncommon. Such effusion communicates freely with the cavity of the cranium, so that fluid may pass easily from the one to the other. Suppurative inflammation of the spinal membranes also occasionally takes place in the cavity of the *spinal arachnoid.* If the inflammation is acute, it may be associated with disease of the *cerebellum,* or of the intracranial membranes; and in the chronic form it rarely exists, except in connection with caries of the vertebræ.

Symptoms.—*Simple meningitis,* or *pia-arachnitis,* has usually been described in three stages. The symptoms of the first stage are those of excitement, resulting from diffuse inflammation; of the second stage, those of compression, marking that effusion has taken place; those of the third are associated with progressive recovery, or with the modes in which death may occur. But there are symptoms characteristic of several forms of this disease, in which the membranes covering the convexity of the hemispheres are the parts generally affected. The most important facts to be ascertained in the previous history of the case, before the development of cephalic symptoms, relate to the general health, and especially to any signs of cachexia or diathetic states, such as *tuberculosis, rheumatism, gout,* or of the specific action of implanted poisons, such as *syphilis.* It is important also to ascertain whether any blow on the head has been sustained, or if the patient has been much exposed to the sun; whether any disease of the ear or nose exists; whether application to study has been intense, or to the cares of business. Premonitory symptoms may be trifling, or absent altogether. The most common are, slight but increasing pains of the head, sensorial disturbance, irritability of temper, or restlessness, with some general *malaise.* Rigors quickly supervene, or simple chilliness, with *cutis anserina* and pallor of the surface, quickly followed by febrile reaction. An attack of convulsions may supplant the rigors, especially in children. Such, however, are by no means common in the adult, and are not necessarily indicative of any severe or advanced lesion. The fever is commonly high; the pulse sharp, hard, and frequent; the respirations irregular, performed with a sigh, and often with a moan. In scarcely any other disease of the brain is the fever of a similar character and equal in severity to that of meningitis; consequently, it is of great importance in diagnosis (NIEMEYER).

If the frequency of the pulse subside after the disease has lasted for some time—if it fall from 120 or 140 beats to 60 or 80, while the other symptoms of fever and the functional disturbance of the brain increase—the evidence is almost pathognomonic of *pia-arachnitis.* The skin is hot; the bowels obstinately constipated; and evacuations, when they occur, are dark and offensive. In this stage there is little or no prostration of strength. The headache of fever is supplanted by acute and intense pain; the face flushes and turns pale alternately; the eyeballs stare, and the conjunctivæ become injected. The functional disturbance of the brain is partly of the character of irritation, and partly of depression, or of complete paralysis. The temper is extremely irritable. There is marked somnolence, or wakefulness, and the two sometimes alternate for several days. The most marked feature is delirium, commencing early, and of a furious character, the patient screaming and gesticulating in the wildest manner; the expression of countenance is savage and malignant, or sometimes fierce. Marked and continuous headache (*cephalalgia*) prevails, with exacerbations of a darting, violent character, eliciting from the patient, and especially from children, a sharp piercing cry. Pain is increased by movement, and the patient holds the head with the hands; or, if a child, frequently carries them to the head. The headache is increased by sensorial impressions. Hence the eyes are obstinately closed, and the ears, if possible, kept covered with the bed-clothes. Double vision, *tinnitus aurium, formicatio,* and subjective sensations of various kinds, are present. The sensorial disturbances are highly marked. Restlessness is incessant, sometimes general or partial. The muscles of the face and limbs twitch involuntarily. There is strabismus, or the eyeball is unsteady, with a contracted or oscillating pupil. Vomiting is frequent, without epigastric pain or tenderness, and often without nausea.

This stage generally lasts from *one* to *four* days, and its characteristics may be shortly expressed as consisting of—*the combination of great nervous hyperaction, with marked fever, a peculiar cry, cephalalgia, vomiting, and constipation.*

The second stage is one of a transition from the first to the third. The fever diminishes. The pulse sinks in frequency and force, becoming variable in frequency between very wide limits, and in very short intervals of time. Respiration becomes peculiarly irregular. The bowels continue constipated. The tongue becomes furred and dry. The heat of the head persists, but the body generally is cool. The nervous phenomena present remarkable intermissions during the further progress of the affection, especially in the following respects: Delirium becomes quieter, or passes into coma; or the patient may appear collected and well. Excitement diminishes and disappears, and drowsiness is the most common feature. Muscular twitchings, generally, are increased on both sides of the body. Convulsions are common in the child, and spasms often alternate with paralysis. A violent general convulsion may throw the patient at once into *the third stage,* which may come on almost immediately, or a week of transition symptoms may intervene. The face becomes sunken, the extremities cold, and the abdomen retracted. *Sordes* form on the gums and teeth. The pulse flutters, becomes thready, feeble, and uncountable. Great prostration of strength supervenes. The nervous functions are gradually suspended. Perception, volition, and ideation, become lost, so far as can be ascertained by corporeal signs. Anæsthesia is complete. There is absolute paralysis to almost every form of stimulus, observed first in the eyelids and eyeballs, and then in the limbs. Muscular relaxation becomes complete, as evidenced by the dilated pupil, stertorous breathing, involuntary micturition, and defecation. Generally there is absence of nervous action, and organic life gradually dies out. For the most part each stage lasts a week; but one or more stages may be wanting.

The tongue in the first stage is white; in the second it becomes brown; in the third it again cleans, if the patient does not die. The pulse likewise in the first stage is from 90 to 100; in the second from 110 to 130; and in the last stage it either gradually returns to its natural standard, or runs on too rapidly and too feebly to be counted.

The symptoms which have been described are those which especially mark *pia-arachnitis* at the superior portions of the brain. When, however it occurs at the base, or in the ventricles, some differences are observable although the condition cannot always be distinguished; but in som cases such a location of the malady is rendered probable if the intellect is less impaired, the passions more excited, and the patient lies fretful, impatient, morose, and although somnolescent, he occasionally cries out and grinds his teeth very early in the disease; while the parallelism of the axis of the eye is frequently affected at an early period.

In the aged acute meningitis seldom occurs suddenly, nor with the intense symptoms which prevail in the meningitis of early manhood. It commences insidiously, without rigors, and very frequently some days elapse before it can be recognised, even by persons familiar with the diseases of old age. The pulse is natural, the tongue remains clean, the bowels regular, and there is little or no vascular excitement, local or general; but the symptoms at the outset are purely of a nervous character. The temper is peevish or irritable, with more or less confusion of thought, inattention, and forgetfulness. "If infirm, and already an inmate of an hospital, the patient commits strange mistakes—takes possession of another's bed, uses the spittoon instead of the chamber-pot, and is frequently found lying outside the clothes, or with his feet where his head ought to be. When addressed, his answers are rational, but still there is a peculiarity in his manner and expression of countenance, an apparent slowness of comprehension, and a vacancy of the eye, that warns the physician of the approach of some cerebral disease" (MACLACHLAN, *The Diseases and Infirmities of Advanced Life,* p. 85). The appetite is slightly if at all impaired; and the skin remains of normal temperature. Restlessness prevails, although prostration is apparent. In walking, the gait is unsteady, and in lifting anything to the mouth the hand trembles. In a few rare cases the disease commences with convulsions. While such symptoms are being developed, after twelve, forty-eight, or sixty-two hours, but generally within twenty-four hours, more or less febrile reaction is expressed by slight increase of temperature merely, and without any marked redness of the face or acceleration of the pulse. The nervous symptoms are now of the following kind:—Wandering, low muttering, delirium, and incessant talking become frequent and characteristic symptoms. Maniacal excitement is uncommon. For a day or two the patient may answer questions rationally, though slowly and hesitatingly, when roused from the reverie in which he is generally wrapped. At a still more advanced stage there is coma. Headache is not a prominent symptom. It is so slight or unfrequent that unless the patient is pointedly asked the question, there is never any allusion either to it or to giddiness, or to *tinnitus aurium.* This absence of headache is a remarkable characteristic; for, even in the most acute pus-forming or false-membrane-forming meningitis, headache may be entirely absent from the beginning. The eyes are suffused, the pupils either slightly contracted or natural. Knitting of the eyebrows, intolerance of light, acuteness of hearing, and vomiting,—common and characteristic phenomena of the disease in early life,—are comparatively rare in the aged. The scalp may be hotter than natural, and, combined with the suffusion and injection of the eyes, is the only physical indication present of vascular excitement within the cranium. The feet are frequently cold, and the surface generally moderately warm. There is usually great thirst, the patient drinks greedily, but seldom asks for liquid. He is apt to refuse food, or takes but a small quantity at a time when it is presented to him. Nervous twitchings and convulsions are observed in the worst cases; and if the patient be raised in bed, these symptoms are frequently induced when otherwise absent, and the head trembles, as well as the upper extremities. When coma prevails, there may be sometimes slight convulsions of the limbs. The general features of the disease resemble typhus fever. The tongue becomes very dry, and generally brown in the centre. The bowels cease to act without medicine, but are not obstinately confined. Towards evening there is generally increased febrile disturbance, denoted by flushing of the face, where it had been previously pale and dejected; dryness of the skin, greater heat of the scalp, and acceleration of the pulse. In numerous examples, while dulness of intellect and somnolence are the first symptoms observed, in others great and unusual loquacity, with redness of the face and heat of the scalp, ushers in the disease. The progress of the disease is equally inconstant. In not a few cases the symptoms of febrile excitement are wanting; but dulness of intellect and stupor, with more or less insensibility of the limbs and relaxation of the sphincters, are frequently prominent symptoms throughout the whole course of the disease, as if the medullary substance of the brain were congested or suffered compression from early exudation on its surface. Such examples of an apoplectic character are more frequently met with in old persons addicted to fermented liquors, in whom the cerebral vessels are enlarged from repeated engorgement. Excessive venous congestion of the brain prevails in such cases during life; and the symptoms of meningitis are marked, and apt to be confounded with effusion into the brain, or softening of its texture (MACLACHLAN, l. c., p. 87).

Chronic Meningitis in the Aged is extremely subdued in its symptoms, and insidious in its approach. It is not unfrequent as a result of albuminuria, or of repeated attacks of *delirium tremens;* or it follows *gout* or *rheumatism.* The characteristic phenomena are accompanied with great impairment of the mental faculties. There is very marked loss of memory, slowness of comprehension, periodical fits of passion. Vertigo prevails, with ringing in the ears; and occasional attacks of headache occur, with or without vascular excitement. Speech is thick. There is paralytic weakness of the lower limbs, and the gait becomes tottering and feeble. All movements, whether of the upper or the lower limbs, are performed slowly, awkwardly, and with more or less of uncertainty. The energies of the whole system are reduced. The functions of organic life are impaired. The appetite may be good, but digestion is slow, the bowels inactive, and the various secretions vitiated or diminished. "Sooner or later the aged invalid takes to bed reluctantly. There he lies uncomplaining, vegetating, the mere wreck of what he formerly was, both in mind and body, gradually sinking, and dying often in consequence of sloughing of the hips and nates" (MACLACHLAN, l. c., p. 93).

As to the *spinal membranes,* one marked symptom of their congestion (referred to in a case related by Dr. Meryon), is the difficulty experienced in walking on first arising after a night's rest. It is a constant indication of spinal congestion, and may be temporarily induced by *strychnine* or *nux vomica.* The affection of the muscles varies from simple stiffness of the part to *opisthotonos.* This latter symptom is often limited to the neck or trunk, without the limbs participating, as in a case given by Rayer, in which the trunk and neck were drawn backwards, while the patient walked freely till the time of his death. In the case of a waggoner thrown off his cart, and pitched on his neck and shoulders, the neck was stiff, the jaw was locked, the body convulsed, and the patient delirious. It was not till the twelfth day, however, that the lower extremities became affected and palsied, when the patient sank into a typhoid state and died. A large quantity of pus was found in the spinal arachnoid cavity. Neither the pulse, nor the tongue, nor temperature, is much affected at the commencement of *spinal meningitis;* but towards its close the pulse becomes rapid and feeble, the tongue brown and dry, and the teeth fuliginous. The patient is now said to be "*typhoid,*" and he dies delirious or comatose. Retention of urine generally persists from the beginning to the termination of the disease. Constipation often exists to a great degree at first, but afterwards the bowels act regularly, or even suffer from diarrhœa.

Severe irritation of parts supplied by spinal nerves, followed by paralysis, indicate acute inflammation of the *pia mater* of the cord. Fever follows a chill, and severe pain in the back and the extremities is complained of, aggravated on motion and on pressure over the spine. Opisthotonos and contraction of the limbs may ensue, which intermit, and are greatly aggravated by movements of the spinal column. Pain in the extremities is often a very prominent symptom at the outset, and may be mistaken for rheumatism; but the most important sequence of symptoms is paralysis commencing in the lower limbs, extending to the bladder, rectum, and, finally, the upper extremities. The paralysis is at first incomplete, but afterwards increases, and anæsthesia succeeds.

Diagnosis.—*Simple meningitis* is distinguished from *encephalitis* by the headache—always severe—the early delirium, and by the general absence of *hemiplegia.* It must be admitted, however, that disease of the brain and of its membranes is often conjoined, so that *meningitis* is not in all cases a simple affection, but is ultimately conjoined with *cerebritis,* the inflammation of the membranes usually extending to the cortical portion of the brain, as in general paralysis, already described. From *delirium tremens* meningitis is mainly distinguished by the character and mode of accession of the delirium and the tremor, and by the *mental, sensorial,* and *motorial* phenomena. From *typhus fever* it is mainly distinguished by the absence of the characteristic eruption or mulberry rash (see Vol. I., p. 555).

The symptoms which distinguish *spinal arachnitis* from inflammation of the substance of the cord, are *pain* and *contraction* or *convulsions* of the limbs. In pure *myelitis* there is seldom any severe or constant pain, while the limbs are generally palsied, and their sensations benumbed or lost. It is distinguished from *rheumatic lumbago,* or *psoas abscess,* by the affection of the limbs and of the bladder, and the gradual advance of paralysis from below upwards.

Prognosis.—Many authorities consider *spinal arachnitis* to be incurable; but numerous cases, marked by the characteristic symptoms in a mild form, do recover. On the other hand, more severe cases progress from bad to worse; and as paralysis extends to the medulla oblongata, death soon follows, or from catarrh of the bladder.

Treatment.—As a general principle, remedies have little influence over those forms of the disease which arise during the progress of any *specific* or *constitutional* disease, independent of the general treatment indicated for that disease. When *pia-arachnitis* arises from mechanical injuries, the treatment is generally by bleeding. If the first symptoms are characteristic of inflammation of the meninges of the brain, and we can only conjecture what it may turn out, *leeches* behind the ears ought to be applied in the first instance. If this local abstraction of blood proves beneficial, it may be repeated during subsequent relapses. At the outset of the disease laxatives and ice compresses may also be used (NIEMEYER). There is, perhaps, no class of cases in which the sanative powers of judicious blood-letting become so apparent as in children in whom the disease has been observed early and carefully watched. When the inflammatory symptoms are less decided, and the headache slight, and the disease drags on slowly, a single venesection is admissible.*

With reference to the sanative influence of general blood-letting in children for inflammatory diseases, if performed at the outset, Dr. West observes, that such depletion is as important a remedy as in the adult; nor will the most energetic employment of any other antiphlogistic measure enable us to dispense with blood-letting. In a healthy child two years old, a vein (if easily found) may be opened in the arm, and *four ounces* of blood allowed to flow, if faintness be not earlier produced, without our having any reason to apprehend that the plan adopted is too energetic. The immediate effect produced is greater than that which follows local depletion, and the quantity of blood abstracted is less (WEST, *Diseases of*

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* Dr. Alison relates the following highly illustrative case, confirmed now by the teaching of Niemeyer:—"A boy, aged eight years, of rather delicate habit, and who had complained occasionally for *some weeks* of headache and disordered bowels, temporarily relieved by laxatives, calomel, antacids, and a careful regimen, but recurring, and attended with gradually increasing febrile symptoms, and *shooting* pain of head; impatience both of light and sound; pulse gradually rising to 108, distinctly sharp, and beginning to intermit; and then nausea and vomiting, not referable to any medicine taken, but gradually increasing, until it recurred every time that he sat up in bed, for nearly twenty-four hours; and a slight but quite perceptible squint showing itself. The full action of laxatives and one application of leeches, as well as cold to the head, having failed to make any impression on this course of things, he was bled at the arm (which in such circumstances and at that age has often appeared to me distinctly preferable) to *twelve ounces*, and the blood was sizy. I do not know what further evidence we could have had of the existence of such inflammation within the cranium as would infallibly, if let alone, have gone on within a few days to delirium, stupor, dilated pupil, slow pulse, succeeded by very frequent pulse, convulsions, and death, and have left after it the usual appearances of the *acute hydrocephalus* (of WHYTT, CULLEN, and ABERCROMBIE, now described), dryness of the membranes on the surface of the brain, distension of the ventricles of the brain with serum, and some of the marks of inflammatory action either on the membranes or surface of the brain. Instead of this, however, I am quite certain that from the time of the bleeding at the arm, this boy *never once vomited*, that the intermission of the pulse was *never again observed*, nor did he again complain of lights or sounds. The pain of the head, although less violent, continued in the evening of the same day, and *twelve leeches were applied* within eight hours after the bleeding; and from that time he never once complained of this symptom, nor admitted that he felt it; and *from this moment he recovered perfectly*, and much more rapidly than he had done from much slighter febrile attacks previously; neither has he suffered from that time to this (now thirty years) any return of serious disease" (*Edin. Med. Journal*, p. 777, March, 1856). In short, all the symptoms vanished in twenty-four hours, most of them during the flow of blood, and never recurred,—a change, under the circumstances, so sudden and sanative as is rarely if ever seen after the use of any other remedy for the same combination of symptoms.

Children). Bleeding in children and in adults exercises an influence quite different—because under totally different circumstances—not yet inquired into; but certainly blood-letting at ages under adult life is soon repaired compared to its reparation after that period. Hasse recommends very small doses of *morphia* ($\frac{1}{24}$ of a grain) in the early stages.

The first thing to be done by way of medicine is to purge the patient. The purgative is not of great moment, provided it acts freely. Some prefer two to five grains of *gamboge,* others five grains of *calomel,* with thirty grains of *compound jalap powder.* Such doses are to be followed up by a *black draught,* or the *sulphate of magnesia.* So difficult is it to obtain the action of purgatives in this disease, that doses of three times the strength for adults have been in some instances required; but such large doses are never to be given until the inefficiency of smaller ones has been ascertained (Sir THOMAS WATSON'S *Lectures,* No. XXVI.). The stools are generally black, or extremely offensive; and, this state of the bowels corrected, the disease, if sympathetic, often ceases. If, however, the head be not relieved, some leeches should be applied to the temples, and the head should be shaved and surrounded with some cold evaporating lotion, such as a towel dipped in cold spring water, or in *vinegar and water,* or in solution of the *muriate of ammonia, with vinegar.*

In chronic cases, Foville strongly recommends the cold *douche*—pouring cold water over the head from a pitcher held some distance above it. This remedy must be used with caution, as it is a powerful depressant, yet producing less ultimate debility than bleeding. It acts, doubtless, by cooling down the general mass of the blood; and patients almost always recover consciousness under its influence. To secure a lasting result it must be repeated at intervals of a few hours.

In children I have certainly seen good results from inunction with mercurial ointment. (See remarks under the treatment of "Hydrocephalus.") In advanced life it rarely happens that the symptoms are so intense as to demand general blood-letting; but in vigorous constitutions this measure is sometimes necessary. The cases which demand it are those attended by high cerebral excitement and vascular action. But as a rule local blood-letting is not only infinitely more safe, but more beneficial, and it can be repeated from time to time, as the nature of the symptoms may indicate; while general blood-letting can very rarely be resorted to more than once, and that only at the commencement of the attack. The beneficial effects of local blood-letting may be greatly aided by keeping the head well raised, and by the constant application of cold water to the scalp; or the occasional use of bladders filled with crushed ice. The bowels should be opened as rapidly as possible, unless the patient is feeble, emaciated, or greatly exhausted. The most active and searching purge is obtained by *calomel* and *jalap;* or four or five grains of the *compound extract of colocynth* should be given, combined with two or three grains of *calomel,* in cases uncomplicated with *gout* or Bright's disease, and followed in a few hours with a dose of *salts* and *senna.* A fair proportion of nutriment must be given, in the form of *milk, strong beef-tea, sago, tapioca,* or *arrow-root;* and the patient should be kept in a quiet, cool, well-aired, and darkened room, and with cold compresses constantly applied to the head. The more active symptoms being subdued, but not till then, a blister should be placed on the nape of the neck. The bowels should be kept open regularly, and the strength supported by mild unstimulating nutriment. When nervous irritability continues during convalescence, *henbane*, or *muriate* of *morphia*, may be given. In the *chronic* form the periodical attacks of headache or of insanity may generally be relieved by a brisk purge; and the head should be kept cool by cloths dipped in vinegar and cold water. If vascular excitement prevails, leeches may be applied behind the ears. The bladder must be kept empty of urine by the catheter, and although wine must be withheld during the active stage of the disease, it is beneficial when the vital energies begin to fall (MACLACHLAN).

The dietetic treatment should be strictly antiphlogistic, the patient especially avoiding all mental excitement; and, if not secluded, he should at least be kept tranquil, not only in body, but also in mind.

Spinal *arachnitis*, seldom depending on a morbid poison, is perhaps in all cases best treated by bleeding and mild purgatives. General bleeding is sometimes necessary; but local bleeding, either by cupping or leeches, along the vertebral column, is most useful, and cannot be omitted with safety. The medicinal treatment consists in moderate purging by the neutral salts, as the *sulphate of soda* or the *sulphate of magnesia;* for, as these act on the bladder as well as on the bowels, they are probably the best remedies. But whatever purgative may be selected, it will be proper to combine it with the *tincture of hyoscyamus*, or other mild opiate, to procure the patient some relief from his sufferings. Ergot of rye (*secale cornutum*) has been very much used in France; and Dr. Meryon speaks favourably of its effects, combined with *iodide of potassium*, in a case which manifested no complication of spinal effusion (*Medical Times and Gazette*, Aug. 31, 1863). The warm bath is an excellent adjuvant in the earlier stages of the disease; whilst in the later stages *blisters, setons, moxæ, or the ointment of the tartrate of antimony*, are more beneficial, or at least are deserving of a trial. The paralysing effusion may disappear during their use, combined with the action of diuretics. The *external* application of *belladonna* and *chloroform* will be found of essential service in diminishing the violent pain which accompanies meningitis of the cord (MERYON). Cold, in the form of bladders filled with ice, applied along the spinal cord, may be of service where congestion prevails. Abstinence from all animal diet should be imperiously prescribed throughout the whole course of the disease.

TUBERCULAR MENINGITIS—Syn., ACUTE HYDROCEPHALUS.

LATIN EQ., *Meningitis Tuberculosa*—Idem valet, *Hydrocephalus Acutus;* FRENCH EQ., *Méningite Tuberculeuse;* GERMAN EQ., *Tuberculöse Hirnhautentzündung*—Syn., *Acuter Wasser-kopf;* ITALIAN EQ., *Meningitide Tubercolare*—Syn., *Idrocefalo Acuto.*

Definition.—*An effusion of serous fluid between the membranes of the brain, or into its ventricles, associated with miliary tuberculosis of the meninges or at the base.*

Pathology.—The *meningitis* which accompanies the cachexia associated with *tuberculosis* is of a very distinctive kind, and frequently terminates the lives of tuberculous children. Its essential morbid character consists in the growth of tubercle on the arachnoid, generally in the shape of small miliary granules. They are found most frequently and most abundantly within the fissure of Sylvius, between and upon the convolutions of the brain, and at the base of the brain, and very rarely on the cerebellum. They are generally of the nature of the grey granulations imbedded in the vascular network of the *pia mater.* The effusion into the ventricles and the softening of their walls are secondary, but not invariable results of the meningeal lesion. It is a disease quite peculiar and characteristic. It is chronic as to its course, compared with acute *pia-arachnitis*—a question of weeks rather than days, and the symptoms are less violent.

(*a*.) *Causation.*—The lesions are part of an acute attack of scrofula, *with* deposit of tubercles in the meninges. They occur generally in children who are of such a scrofulous disposition that in later life they would be likely to suffer from pulmonary consumption; or they are the offspring of consumptive or otherwise debilitated parents—badly nourished, physically ill-developed, but mentally often very sharp and bright. Regarding such children it is a common saying that they are "too wise and clever to live long." They generally have a fine skin, very perceptible veins, long eyelashes, and a clear bluish sclerotic. (See pp. 16, 17, *et seq.*, Vol. I.)

The remote causes of hydrocephalus are often extremely obscure; but exposure to cold or heat, errors in diet, falls or blows on the head, the retrocession of a cutaneous eruption, or the extension of an inflammation of the ear, are among the most common. Disordered functions of the liver and alimentary canal, dentition, and the presence of worms, are also frequent causes; and the circumstance of a child being seized in consequence of its feet having by accident been put into a bath of boiling water will show that any other extreme irritation will equally produce it. Many morbid poisons also will occasion it, as that of *scarlet fever*, *pertussis*, or *measles.* Of *constitutional* diseases, *tuberculosis* is the most common exciting cause.

In adults the development of acute hydrocephalus is generally subsequent to acute miliary tubercle of the lungs, or chronic pulmonary consumption, forming an acute complication to these diseases. The disease may also be a sequence to typhus, enteric fever, measles, or scarlet fever (NIEMEYER). The age of childhood exercises a most remarkable predisposing influence upon this disease. At that period the rapid growth of the brain, the irritation of dentition, and the great susceptibility of the nervous system generally, are all powerful causes of determination of blood to the head. The greatest number of attacks occur between the second and the fifth year; or, as a more general law, the disease occurs from infancy to twelve years of age. During the first year of life, however, the disease is rare. Children with large heads and precocious intellects, and more especially those of a scrofulous diathesis, are its most frequent victims. It is also said to be most common in the children of parents addicted to drunkenness, and from this cause it often runs in families.

But unless in the form of *tubercular meningitis, acute hydrocephalus* is undoubtedly a rare disease. A disorder also occasionally occurs, which is really an *acute hydrocephalus*, due to certain changes of nutrition of the nature of inflammation in the membranes or *ependyma* of the ventricles, followed by more or less effusion into their cavities. This form of *acute hydrocephalus* is rapid in its course, ending within two, three, or four to fourteen days (BEVERIDGE).

Premature or excessive mental exertion and blows on the head may be, and often are, alleged as the exciting cause of *acute hydrocephalus.* It does not always follow, however, that such is the case. The blow may only be a means of directing attention to the child, after which the discovery is made that symptoms of cerebral irritation exist, which had already commenced.

(*b.*) *Morbid Anatomy.*—On removing the *dura mater* the surface of the convolutions are visible; but they are flattened, owing to the pressure from fluid within the ventricles forcing the hemispheres against the skull. This condition may be so extreme, as to remove all appearance of sulci—from the pressure of the convolutions so closely together. The surface of the brain looks like a smooth and even mass, with blood-vessels ramifying over it. The arachnoid has lost its lustre. Scraping it yields little or nothing. Beneath it no lymph is seen. The surface of the brain in this form of pia-arachnitis presents no marked inflammatory products, thus markedly contrasting with the simple and acute form of the lesion. On removing the brain, however, very distinctive and characteristic appearances are seen :—In the presence of tenacious lymph at the base, in the diamond-shaped space about the pons Varolii and the cerebellum (sometimes called meningitis of the base, basilar meningitis). A yellowish opaque exudation is usually conspicuous over the base of the brain, and within the *subarachnoid spaces;* and generally plentiful in the meshes between the *pia mater* and *arachnoid.* It is especially abundant about the *chiasma* of the *optic nerves,* the larger fissure of Sylvius, and cerebral fissures towards the convexity of the brain. The *pia mater* is generally covered with whitish granulations, like particles of sand or a hemp seed, most abundant and distinct in the vicinity of the blood-vessels (BASTIAN, *Ed. Med. Journ.*, April, 1867). The anatomical characters of the lymph are peculiar. Tubercles are to be found in it, attached to the small blood-vessels, on microscopic examination. The lymph is most plentiful, especially about the optic chiasm and commissure; and those portions of the meninges which extend towards the pons and medulla oblongata; and along the larger cerebral fissures—the fissure of Sylvius and convex surfaces of brain, so that when the lobes are separated a quantity of the lymph may be seen gluing them together. The tubercles are best seen on searching the *pia mater* after its separation from the brain. They are not generally seen from the surface; but it is especially in those portions of the membrane which dip down between the convolutions that the tubercles are best seen. The membrane should be spread out in water, over a dark surface, so that the tubercles will appear as white dots.

The ventricles, especially the lateral and the third, are sometimes considerably distended with serous fluid, to such an extent as to cause bulging upwards of their roof, lifting up the corpus callosum and carrying it backwards. The fluid may be as much as 4 ounces; but generally less, and of increased specific gravity, as much as 1010 (normal being 1000), occasionally quite clear, but sometimes clouded with flocculi. The larger the amount of fluid, the more bloodless and pale is the brain, and the whiter the softened parts (NIEMEYER). The walls of ventricles, especially the fornix and commissural parts, are softened, so soft as to be diffluent, and more so in the central and commissural parts than at the sides. The fornix and septum lucidum are sometimes quite broken down. The cause of this softening has been the subject of much discussion, but the weight of evidence is in favour of its being of the nature of an "inflammatory *œdema;*" for a microscopic examination shows inflammatory products, the elements of granule masses, collected especially along the course of the capillary vessels. The ependyma of the ventricles are generally granular, as if sprinkled over with fine sand, a condition sometimes extending into the fourth ventricle. This is a condition apt to be overlooked, unless the surface of these ventricular cavities be held up to the light, so that it may come obliquely upon it.

That the cineritious surface of the brain is also implicated is shown, (1.) By shreds of it coming away with the *pia mater;* (2.) by microscopic examination. The degree and manner in which the substance of the brain is implicated, are best shown by a microscopic examination. Strip a little piece of the membrane from a convolution in the fissure of Sylvius. Examine it with a low power. If tubercles are present they will be seen following the course of the blood-vessels, the small arteries. If a favourable spot is chosen and examined with a higher power ($\frac{1}{4}$ inch), the tubercles will be seen inclosed between the vessel and its perivascular sheath, sometimes compressing the vessel and narrowing it. Another method is to harden a portion of the brain with the membrane attached, and cut sections through both brain and membrane; the tubercles will be seen within the sheaths of the vessels, following them as they enter the cortex or cineritious substance of the brain. Hence it is that the functions of the brain generally show severe derangement in tubercular meningitis.

The distinctive *post-mortem* characters of tubercular meningitis are—(1.) The absence of lymph on the surface of the brain (in this respect differing from simple meningitis); (2.) a flattening of the hemispheres; (3.) the existence of lymph at the base; (4.) tubercles in the *pia mater* following the course of the vessels; (5.) increased fluid in the ventricles, with softening of their walls. If tubercles were not visible to the naked eye, the rest of these characters would at once indicate the nature of the disease; and a microscopic examination would be sure to reveal tubercles along the vessels (WILKS, l. c., p. 212).

The brain affection is often apt to be regarded as the primary disease; but on *post-mortem* examination it will be found that there has been softening of bronchial, lymphatic, and mesenteric glands, after they had undergone caseous degeneration, or of yellow masses in the ovaries, with tubercles in the lungs or intestinal canal, all of which bespeak the general constitutional state of scrofula and the infective source of tubercle (see p. 1014, Vol. I.).

In the form of acute hydrocephalus due to changes in nutrition in the ependyma of the ventricles described by Dr. Beveridge of Aberdeen, the surface and substance of the brain appear natural, and the fluid in the ventricles may be clear, but a granular layer of lymphy exudation covers the floor of the ventricles. This layer of exudation appears thin, soft, and grey in colour, and is easily overlooked on account of its resemblance to the parts which it rests upon. It is composed of granules, exudation corpuscles, and imperfectly formed cells. It may be seen covering the optic thalamus and corpus striatum. It is most abundant towards the fore part of the ventricles, where, in the angle between the rounded anterior end of the *corpus striatum*, and the *crura* of *the fornix*, it accumulates to the greatest extent, so as sometimes almost to fill it. At or opposite the foramen of Monro it causes a very peculiar appearance. There the exudation, so far from closing the opening, prolongs it on either side into the ventricle, converting it into a sort of short canal, which opens by oval apertures with sharply defined borders, placed not vertically, but so obliquely as to be nearly horizontal, quite clear of the edge of the fornix, and visible at once on opening the ventricle from above; so that, instead of its being necessary to look underneath the apex of the fornix to see the foramen, the openings of it appear like two oval eyes between the fornix and corpus striatum, and are in full view, without disturbing or touching the parts in any way. This appearance gives the impression as if a soft granular layer had settled down on the floor of the ventricle, filling up its angles and interstices, but prevented from covering the foramen of Monro by the steady set of a current through that opening (*Med. Times and Gazette*, Vol. II., 1868).

Symptoms.—(*a.*) *In the Child.*—Acute hydrocephalus has generally been described in three stages; the first stage being that of increased irritability; the second, that of diminished sensibility; and the third, that of convulsions or palsy (CHEYNE). If the scrofulous cachexia exists, *tubercular meningitis* may supervene on the occurrence of any febrile disturbance, with slight thirst and anorexia; irregular and somewhat quick pulse; vomiting and constipation; clayey evacuations deficient in bile; red and moist tongue; dry and hot skin, and other phenomena of general derangement. The temperature, as measured by the thermometer, will be found to be persistently above the normal amount (RINGER). The first stage may thus be either sudden in its attack, or be preceded for several days by insidious and indefinite symptoms, such as giddiness, so that the child stumbles or falls at play; by a furred tongue, constipated bowels, and perhaps offensive breath. Or the child shows some change in manner, declines play, desires to sit apart and rest the head. At length the senses of sight and of hearing become morbidly acute; he starts at slight noises; complains intermittingly of headache; rests his head on his nurse's lap; occasionally complains momentarily of his head; and then, after a time, rises up and plays again. There is intolerance of light and sound. The special nervous phenomena are generally feebly marked. There may be irritability of temper and peevishness, with some slight delirium at night, rarely commencing early in the disease, disturbed sleep, and restless manner. Vertigo is also indicated by staggering, or clinging to objects for support. Grinding of the teeth prevails, occasional vomiting, unsteady restless movements, and dragging of the limbs. As this stage advances, the pulse rises, the skin is hot and dry, the urine scanty, the stomach irritable, the bowels constipated, perhaps painful, the stools black and offensive, while the brow is knit, and the pupil of the eye contracted or expanded. The most remarkable feature, however, is a great fretfulness of temper, so that the child is not merely pettish, but quarrelsome. If he sleeps, his sleep is short, uneasy, moaning; he also grinds his teeth, rolls his head, and when he wakes up it is with a scream. He is generally sleepy, and dreams a great deal. "We are led to suspect some deeply-seated evil from the frantic screams and complaints of the head and belly, alternating with stupor, or rather lowness, and unwillingness to be roused" (CHEYNE). These symptoms may extend over several weeks; and the child becomes emaciated by fever and impaired digestion. After three or four days of these premonitory symptoms, *the second stage* commences, with heat of head and flushings of the face, alternating with pallor. The pulse is irregular, and commonly, when the child is still, it is of little frequency, but it rises rapidly if the child is disturbed. The vomiting ceases, but the constipation persists, with retracted abdomen. The following special nervous phenomena become more marked:—The child inclines to lie quiet, and resists being moved, as if pain was increased by motion. There is delirium, which is sometimes fugitive and sometimes persistent. The cephalalgia increases. The expression of countenance bespeaks great suffering, and the face looks aged. A peculiar piercing cry is now and again given by the child. The eyes are closed, and there is a tendency to drowsiness. Strabismus and muscular twitchings occur. The pupils are variable, and often oscillate, and the eyeballs are unsteady.

The second stage may also be recognised by the occurrence of effusion. The pulse, instead of being rapid, is then as slow, perhaps slower, than sixty beats a minute; but this is chiefly when the patient is in a horizontal position, for if he attempts to sit up, it again becomes rapid. The sickness is also abated; nevertheless, the child lies in a state of stupor and of great unwillingness to be moved, with his eyes half-closed, dull, and heavy, or perhaps staring or squinting, the pupil being still contracted begins to expand—first one and then the other—so that he often suffers from double vision. Loud noises do not now disturb the child, nor light influence the eyes. The stupor, however, is still interrupted by exclamations or shrill piercing screams, which are reflex, while the tremulous hand of the little sufferer is incessantly engaged in picking his nose or mouth. As the disease thus advances, the capillaries of the brain are compressed by the effusion, loss of consciousness begins, epileptiform convulsions supervene, and finally paralysis of the extremities. For a time the inspirations are imperfect, and then the lungs are filled by a deep sighing inspiration; *coma* becomes deeper, the face assumes a dark colour, and appears pinched, with sunken eyes; and often tetanic contractions of the body cause a painful feeling to those unaccustomed to such a disease.

In the third stage the patient either sinks or recovers, and temporary appearances of improvement may excite deceitful hopes. If the event is about to be unfavourable, the pulse again rises, the eye becomes red and dim, and the child, delirious, is often attacked by partial or general convulsions, or one limb or one side may be palsied. From this point the powers of life gradually sink, till at last death closes the scene; but for days and days the child may linger on the very brink of the grave. If the patient should fortunately recover, the stupor subsides, the countenance becomes more natural, the bowels more regular, the secretion of urine perfectly restored, and at length his health, though long broken, is gradually re-established. The duration of this acute form of the disease is estimated at about three weeks, each stage averaging about a week. Thus, *the third stage* of tubercular meningitis may be ushered in by the general symptoms of approaching dissolution, such as coldness of the extremities, clammy perspiration, and an excessively rapid but feeble pulse. The special nervous phenomena are, first, those of exalted spinal action, and then those of general prostration. Drowsiness passes into stupor, with an idiotic expression of face. There is loss of perception and volition. All signs of activity give place to anæsthesia, and the eyes are half open. Death approaches by convulsions, with partial paralysis, *subsultus tendinum*, clenched hands, retracted head, and automatic movements, giving way to general relaxation; the skin perspires copiously, while the urine and fæces are passed involuntarily.

In much of this description some of the phenomena of *meningitis* may be recognised, and, doubtless, a great number of the acute cases are associated with this morbid stage already described (page 419, *ante*), and especially with meningitis at the base; for at the commencement the lesion is localised there, and the characteristic peculiarities in the sequence of its symptoms are so distinctly traceable to the functions associated with these parts as to give grounds for very certain diagnosis. The numerous nerves starting from the base of the brain and passing through the foramina, so that (1.) irritation, and (2.) paralysis in the parts supplied by the *nerves of the eye*, the *vagus*, and *medulla oblongata*, direct attention to the child. Among the first signs are *contraction*, and then later, *dilatation of the pupil, ptosis of the upper eyelids, vomiting, slowness, and subsequent frequency of the pulse, peculiar changes of respiration*, and depression of the abdomen (NIEMEYER, BUDGE).

(*b*.) *In the Adult.*—The symptoms occasionally assume an apoplectic, sometimes a convulsive form, at the commencement; and the febrile character is generally imperfectly marked. The premonitory symptoms are those associated with the scrofulous cachexia, and *meningitis* may occur at any stage of the lung disease. The following are its features:—After some remission of chest symptoms, special nervous phenomena, referable to the head, may supervene. The patient looks bewildered, with a dull, heavy, expressionless face, often highly characteristic. There also appears to be some *intellectual* incapacity to speak; the patient, seeming to understand what is said or asked, looks at the inquirer for a few seconds, and then turns the head away without a reply. There is often marked somnolence. Pain in the head, fixed to one spot (generally the forehead), is the most striking symptom, of considerable intensity, and is persistent for many days. An attack of convulsions may precede every other symptom, but paralysis is rare. *The second stage* is of variable duration, the pulse is highly irregular, while alternate flushings and pallor of the countenance are common, and all the symptoms already noticed become more intense, mild delirium prevails, and the face becomes increasingly stupid-looking. Paralysis of volition alternates or co-exists with clonic or tonic spasms, strabismus, or prolonged convulsive attacks. *The third stage* of tubercular meningitis in the adult is marked by increasing stupor, immobility, and involuntary defecation and micturition.

The general characteristics of *tubercular meningitis* thus consist in the occurrence of fixed pain, vomiting, dulness of intellect, and duskiness of the face, with partial paralysis or convulsions, slight fever, and diminution of the chest symptoms in a patient demonstrably tuberculous. The indications of inflammatory action are only feebly marked, not only during life, but after death; and very often the febrile state which had existed before becomes less noticeable at the onset of cerebral symptoms. Still heat of head, injected conjunctivæ, and flush of face, denote a condition of vascular excitement; while persistent headache, with febrile symptoms in a patient otherwise phthisical, when it can be traced to no other known cause, is strong presumptive evidence of impending *tuberculous meningitis.* The aid of the thermometer should invariably be sought in diagnosis, (see p. 271, *et seq.*, Vol. I.) But there are also cases in which the disease of the brain is marked by the absence of ordinary symptoms. The premonitory phenomena already noticed, for example, may be absent, especially in children; and then the steady persistence of any one symptom becomes of great importance in diagnosis—for example, sickness, headache, constipation, drowsiness, heat of head (WEST).

Diagnosis.—The vomiting in this affection is characteristic, and has been described as cerebral vomiting (p. 89, *ante*). It is not traceable to errors of diet, nor does it occur after eating, but when the child is raised up, or the head is elevated. When the child is also seen to bend the head backwards, or bore it into the pillows, with the muscles of the neck contracted, and perhaps the cervical glands swollen—these circumstances are very suggestive of the onset of acute hydrocephalus. It is to be distinguished from enteric fever by the screaming, rolling of the head, grinding of the teeth, and by the absence of the peculiar state of the tongue which marks the latter disease. There is always intense marasmus, although food may be taken.

There is a morbid state resembling this disease which is neither *acute* nor *chronic*, to which Sir Thomas Watson has given the name of *spurious hydrocephalus.* To three great men of the past we owe our knowledge of this condition—namely, to Gooch, Abercrombie, and Marshall Hall. In children, from a few months to two or three years of age, of small make and of delicate health, from exposure to debilitating causes, this morbid state not uncommonly supervenes. It is indicated by heaviness of the head and drowsiness. The child lies on its nurse's lap, unable or unwilling to raise the head. It seems half asleep, one moment opening its eyes and the next closing them again, with a remarkable expression of languor: the eyes are unattracted by any object put before them, and the pupils remain unmoved on the approach of light. The breathing is irregular, sighs are occasionally expressed, and the voice is husky. The tongue is slightly white, the skin is not hot, but sometimes colder than natural. In some cases there is now and then a slight and transient flush. Diarrhœa has often existed for some time, or the child has been severely purged by medicines, or, having been weaned, has ceased to thrive since its change of food. The peculiar green colour of the stools, so frequent in this disease, seems to be imparted to them in the lower portion of the intestine, the fæcal contents of the upper portion being of a pale drab colour, while the bile in the gall-bladder is of a yellow colour. When this condition of things occurs in a more elderly child, it has been generally brought about by depletion, by loss of blood, or by medicines. As the more marked symptoms are sometimes ushered in by extreme irritability and a feeble attempt at reaction, cases of this kind (which Dr. Marshall Hall named "the hydrocepha*loid* disease") have not unfrequently been mistaken for acute hydrocephalus, and treated accordingly, the patient being generally leeched out of its life. In very young children the diagnosis is sometimes very difficult between congestion and exhaustion, between fulness and emptiness. Sir Thomas Watson suggests the following test:—"As a guide to diagnosis, take notice of the state of the unclosed fontanelle. If the symptoms proceed from plethora, or inflammation, or an approach to inflammation, you will find the surface of the fontanelle convex and prominent, and you may safely employ and expect benefit from depletion. If, on the other hand, the symptoms originate in emptiness and want of support, the surface of the fontanelle will be concave and depressed; and in that case leeches or other evacuants will do harm, and you must prescribe better diet,—ammonia," brandy in arrow-root, milk from the mother's breast, if possible, and all such means as will tend to foster and nourish an infant.

Prognosis.—Most physicians are agreed that if acute hydrocephalus shall unequivocally declare itself, it will be rapid, and almost inevitably fatal. The signs of a fatal end have been already stated.

Treatment.—Tuberculous meningitis, in the form of *acute hydrocephalus*, can only be successfully combated in the first or earliest stage.

If the disease be advanced, no efficient treatment has as yet been determined. The symptoms do not yield to the lancet like those of simple inflammation. *Mercury* has been used to a great extent, but with little success. In severe cases *mercurial ointment* has been rubbed on the back and thighs, even in very young children, to the extent of half a drachm to a drachm three or four times in the twenty-four hours. *Calomel* also has been rubbed on the gums to the extent of three or four grains every four or five hours. It has likewise been given by the mouth in doses of two grains every third or fourth hour. *Mercury* given in these large doses seldom produces salivation in children under three years of age. But the remedy is not successful, and more generally produces *spinage-like* stools, and irritates the alimentary canal.

On the other hand, Professor Gölis of Vienna recommends, after much experience, *calomel* in quarter or half-grain doses twice a day; also at the same time inunction of an eighth or a fourth of an ounce of mild *mercurial ointment* into the shaven scalp once in twenty-four hours. The head is to be kept constantly covered with flannel, to prevent any check to perspiration. After a lapse of six or eight weeks, diuretics in the form of *acetate of potash*, or *tincture of squills*, or both, are to be given with the mercury. Counter-irritation by issues in the neck are also advocated by him to be kept open for months. Compression of the head by bandaging, or by well applied strips of adhesive plaster, are also to be applied over the whole cranium. Puncture of the skull, if the fluid is *arachnoidean*, may also prove of service,—not so if the fluid is *ventricular*. It is to be done with a small trocar and canula at the coronal suture, about an inch and a half from the anterior fontanelle, taking care to avoid the longitudinal sinus.

The fluid must be slowly evacuated, and compression of the skull carefully maintained for many weeks while the fluid is allowed to drain away. The operation has been successful in very young children.

Hygienic measures ought to be of the first importance in the rearing of delicate children, so as to prevent if possible the development of those conditions which lead to effusion of fluid within the cranium. During the course of the disease the diet should be slops and light puddings.

In congenital hydrocephalus the unassisted efforts of nature are incapable of effecting a cure, and it is extremely problematical if medicine is of any use. When, however, the case is deemed hopeless, the propriety of evacuating the water by means of an operation may be entertained. Gölis has given the names of twenty-seven writers who have expressed themselves in favour of it, especially if the fluid be slowly evacuated, and at several repetitions of the operation; yet he himself, along with seven or eight others, proscribe it altogether as cruel and useless. But it has been successful; and when the operation is performed, it seems an axiom that the fluid should be allowed to escape gradually, for otherwise extreme faintness and collapse may be expected. In such cases small doses of ammonia, or a few teaspoonfuls of brandy and water may revive the little patient. Should reaction take place, however, at a subsequent period, a few leeches and a cold lotion ought to be applied to the head. It seems also determined that the younger the child the greater are the chances of success; for if it lives a few years the sutures of the cranium, though open at the top, are united by bone towards the base of the skull, and thus present a mechanical obstacle to their closure; consequently, the operation is more apt to fail. If this disease, though existing at birth, should not develop till later in life, *blisters*, *mercury*, and *iodide of potassium* to salivation are the remedies mostly relied on.

CHRONIC HYDROCEPHALUS AND HYDRORACHIS.

LATIN EQ., *Hydrocephalus Longus;* FRENCH EQ., *Hydrocéphale Chronique;* GERMAN EQ., *Chronischer Hydrocephalus;* ITALIAN EQ., *Idrocefalo Cronico.*

Definition.—*Effusion of fluid in the subarachnoid space, or generally distending the ventricles of the brain, and differing from cerebro-spinal fluid in containing more albumen* (HOPPE); *occurring chiefly among children, and when discovered later in life, generally dating back to childhood. The tissue of the brain in contact with the fluid, especially the commissural parts, is apt to be broken down by œdema into a thin white pulp* (*hydrocephalic or white softening*).

In hydrorachis the effusion of fluid is within the cavity of the spinal cord.

Pathology.—(*a.*) *History.*—Chronic hydrocephalus was very little known till Dr. Whytt published his *Observations on Dropsy of the Brain* in 1768; but since that period Dr. Fothergill, Dr. Watson, Dr. Cheyne, and a number of other writers, have contributed to illustrate its nature.

(*b.*) *Morbid Anatomy.*—There are a few cases in which effusion of serum into the ventricles, or beneath the arachnoid, is unaccompanied by any morbid appearance of the brain or of its membranes whatever.

Thus there are many instances in which hydrocephalus is not demonstrably inflammatory. More commonly, however, some lesion of the brain or its membrane does exist. Thus, the substance of the brain is often marked with more bloody points than usual; the *septum lucidum*, the *fornix*, and other parts forming the walls of the ventricles, are often found in a state of softening—sometimes so soft that Gölis gives a case in which water could be expressed from it as from a sponge. The membranes also are sometimes congested, or opaque and thickened, with spots of lymph, evidently the effect of a low inflammation. The quantity of fluid effused varies from a few teaspoonfuls to seven or eight ounces, and of this the greater part is generally contained in the lateral ventricles, which from this cause are often so enlarged and distended that the finger placed on the brain, immediately over the ventricle, is sensible of a distinct fluctuation, while the anterior portion of the fornix is often so raised as to cause a free communication with the third ventricle, and perhaps with the fourth —at least, the effused fluid is found likewise distending those cavities. The quantity of fluid effused underneath the arachnoid is often very great. The choroid plexus, or ventricular membrane, although in general pale and healthy, yet sometimes has the intercellular tissue so infiltrated that it appears studded with small cysts. The bones of the skull are remarkably thin and transparent. The sutures, although generally closed towards the base, are commonly separated from each other by a wide extent of membrane at their superior portions. If, however, the patient should survive for several years, the membranous portion becomes ossified at a number of points, forming "ossa Wormiana," and the sutures are thus partially closed. In some few instances the sutures not only close, but the bones of the skull have a morbid thickness. The membranes of the brain are generally thickened, and fluid is found effused either into the subarachnoid space, or into a cyst, or into the ventricles of the brain. When the fluid is contained within the subarachnoid space, the brain is sometimes so compressed that there are instances in which hardly a vestige of brain remains. A singular and rare variety of this affection occurs when the arachnoid protrudes through the *fontanelle* or open suture, and the *dura mater* and integuments yielding, a pyramidal bag, with its apex downwards, forms externally, which hangs low down the back.

When the effused fluid is contained within the ventricles, those cavities are found exceedingly dilated. The convolutions have no depressions, but appear unfolded. The *corpus callosum* is much raised, the *septum lucidum* is torn, or the grey commissure destroyed, and the white commissure elongated to the extent of an inch, so that the ventricles communicate. The parts at the base of the brain also, as the *corpora striata* and *thalami optici*, have scarcely any existence. In fact, the brain seems expanded into a large sac, in which the medullary and cortical substances are so confounded as to be undistinguishable. In Dr. Bacon's case the brain and membranes, even the *dura mater*, had ruptured, and a probe passed easily through the ethmoid bone into the nose, by whose orifices a considerable dribbling of fluid took place during life. Gölis met with a case in which the water was contained in a cyst the size of a goose's egg, situated between the hemispheres of the brain of a child aged six years, and who died, the cyst being entire. The quantity of fluid contained in the cranium in cases of chronic hydrocephalus varies from a few ounces to a few pounds. In the case of Cardinal it was found to exceed ten pints (nine pints in the cavity of the arachnoid, and one pint in the ventricles). Other cases have been, however, recorded in which the quantity has amounted to twenty pints.

Authors have greatly differed as to the nature of this disease. Some, considering it a mere increase of fluid from functional activity, have named it *dropsy of the brain*, most often congenital, and others have as constantly referred it to an inflammatory origin; but they have generally concurred in describing an acute and chronic form of the disease. The symptoms during life are due to the mechanical action of a variable amount of fluid, causing enlargement of the head.

In the chronic form of hydrocephalus certain parts of the base of the skull are liable to deviations, which are quite as important to be recognised as are the numerous extensive changes in the vault of the cranium. These changes in the base have been especially pointed out by Mr. Prescott Hewett in the first volume of *St. George's Hospital Reports.* They occur especially in the lateral parts of the anterior and of the middle fossæ, especially the orbital plates of the former. These orbital plates are driven downwards, and either present a plain surface, oblique from before backwards, or they may be perpendicular, or even convex, bulging into the orbit so as to reduce the orbit to a mere chink. The orbital arch is more or less done away with; the frontal and orbital portions of the bone may even present one continuous line convex in its whole length. This deviation is usually associated with dropsy of the ventricles. If the orbital plates are natural in shape and direction, then the effusion into the ventricles has either occurred at a period when the bones were not easily acted on by the pressure of fluid, or when the accumulation of fluid is on the surface of the brain—subarachnoidean, and not ventricular. In the former—*the arachnoidean dropsy*—the fluid is limited to the upper and lateral parts of the surface of the brain; and, however great may be the quantity of fluid, it cannot press on the bones at the base of the skull. The deviation in the orbital plates characteristic of *ventricular dropsy* is recognised during life by the state of the eyeballs. In such cases the eyes are more or less driven out of their sockets, and have a marked direction downwards; a great part of the pupil is hidden beneath the lower lid, and the white of the eye is much more uncovered than usual. The deviations of the middle fossæ of the skull exist in the lateral portions, where, by the bulging out of the bones, the base, in some cases, undergoes extensive and strange alterations, leading to singular deformities about the face. The deviations are of the following kinds:—The fore part of the squamous portions of the temporal bones, as well as the great wings of the sphenoid, may be driven outwards and downwards; bulging into the zygomatic fossæ, these bones lie on a level with the alveolar margin of the superior maxillaries. The zygomatic arches, closely fitted to the expanded bones, are themselves flattened, especially the malar bones, to twice their natural width. The orbital plates of the sphenoid project into the orbits, and are quite convex. In some instances, extending from the orbits in front of the spinous process of the occipital bone behind, large pouches bulge out on either side, as if the cheeks were blown out. These pouches are partly osseous and partly membranous. The fluid in these pouches was ascertained, in a case during life, to communicate with that in the skull (CREUTZWIEZER, in *Rust's Mag.*, 1835, p. 463), and the whole contained within the cranial cavity.

The blood circulation and nutrition of the fundus of the eye also undergo great changes, which may to some extent be explained by the compression of the cerebral substance from accumulation of fluid. As the fluid collects and the pressure increases, there occur,—(1.) Greater vascularity of the papillæ and choroid, with dilatation of the veins; (2.) an increase in the number of the vessels of the choroid; (3.) partial or total serous infiltration of the papillæ; (4.) atrophy of the choroid and its vessels; (5.) atrophy of the optic nerve, which may be complete.

Effusion of fluid within the cavity of the spinal cord is analogous to fluid in the ventricles of the brain. It leads to compression of the cord, and paralysis as a symptom of such compression. It is usually a congenital state, associated with a defective development of the vertebral arches. The paraplegia it induces generally extends to the sphincters and the bladder, producing incontinence of fæces and urine. The condition is indicated by a tumor at the lower end of the canal, over the region of the sacrum; and the cavity of such tumors communicates with the cavity of the vertebral canal, and the paralysing influence is occasioned by the pressure of the fluid on the spinal cord. Congestion of the membranes may occur as a stage preliminary to all these conditions.

Symptoms.—The first thing that strikes us on examining those patients who suffer from chronic hydrocephalus, is the enormous *size* of the head. The adult head averages about twenty-two inches in circumference. Dr. Bacon gives the case of a child whose head at three months had attained the enormous size of twenty-nine inches in circumference (*Med.-Chir. Trans.*, Vol. III.). The head of Cardinal, a celebrated hydrocephalic man about London, long in St. Thomas's Hospital (and who afterwards died at Guy's), measured thirty-three inches and a half. There are instances, however, in which the cranium has been found unusually small, and of a conical shape, the sutures being closed before birth: in these cases the children are still-born, or die shortly after delivery. When the disease comes on at later periods of life, after the sutures are closed, the size of the skull is natural, the cavities within the brain distended, and its substance wasted and anæmic. The *form* of the hydrocephalic head is sometimes very irregular. One side may be much larger than the other, while the bases of the orbits are, for the most part, convex instead of concave, thrusting the eye unnaturally forwards.

There are two forms of chronic hydrocephalus, the internal and the external, or hydrocephalus in which the membranes protrude. In either case, whether congenital or subsequent to birth, the child is generally of the most feeble intellect, irascible, often epileptic, and of extreme muscular debility, so that, if not palsied, he is hardly able to walk. Dr. Baillie met with an instance of chronic hydrocephalus in a man aged fifty-six, whose ventricles contained six ounces of serous fluid. His chief symptoms were pain in the head, and a loss of memory so great that he could recollect only five words, which he continually reiterated to express all his wants. Cardinal, whose case has been mentioned, had more memory, and he prided himself, says Dr. Elliotson, in being able to say "The Belief," but he usually stumbled when he got to "Pontius Pilate." This man was epileptic, of very feeble intellect, and so irascible as to be always quarrelling with the patients, and would have been extremely difficult to manage but for his muscular debility. His case was one principally of *arachnoidean dropsy;* and seven pints of fluid were found within the arachnoid. The perpendicular portion of the frontal bone was widely expanded; and its orbital portion had become horizontal, while the arch of the orbit was complete. Heberden, however, mentions a case in which eight ounces of water were found in the ventricles of the brain, and yet no special symptoms of hydrocephalus existed during life.

Prognosis.—The immediate danger in the cases of chronic hydrocephalus is not great, but few patients survive the age of puberty; Cardinal, however, lived to the age of thirty-two. Aurival speaks of another who reached forty-five; and Gall of a third, who survived till fifty-four.

Treatment.—For *chronic* cases of hydrocephalus there can be no relief. In cases of *hydrorachis,* where a tumor is associated with *spina bifida,* iodine injections have been proposed (VELPEAU, DEBOUT, BRAINARD, GROSS, MERYON). In operating, the puncture should be very small, by a small flat curved needle, directed subcutaneously into the sac. A drain of the fluid may then be allowed to escape through a canula, and the injection used must be very weak at first, the object being to excite a slow process of inflammation in the cyst. One-eighth of a grain of *iodine,* and a quarter of a grain of the *iodide of potassium* in solution, is the quantity prescribed for injection by Dr. Gross. Dr. Brainard uses a solution composed of five-eighths of a grain of *iodine* and half a grain of *iodide of potassium* to one drachm of water. The puncture is to be closed with a twisted suture, and coated over with collodion. An anodyne should be administered, and the child kept lying on its face. Mr. W. M. Coates, of Salisbury, has been also successful by a similar method of treatment. If the life of the child be saved, paraplegia is still apt to remain, and perhaps involuntary defecation and micturition (MERYON, in *Brit. Med. Journal,* July 11, 1863, p. 28; also, *Practical and Pathological Researches on the Various Forms of Paralysis,* p. 25).

ENCEPHALITIS AND MYELITIS: INFLAMMATION AND SUPPURATION OF THE BRAIN AND CORD.

LATIN EQ., *Inflammatio Cerebri;* FRENCH EQ., *Cérébrite;* GERMAN EQ., *Entzündung der Hirnsubstanz;* ITALIAN EQ., *Inflammazione del Cervello.*

Definition.—*Inflammation of the brain and spinal cord substance, with or without implication of the membranes, usually partial.*

Pathology.—(*a.*) *Causation.*—Inflammation of the substance of the brain is a rare disease, and is rarely uncomplicated and primary; myelitis is more common. As a rule, both result as a consequence of previous diseases, especially *specific fevers* (*exanthemata*), *diseases of the ears, extravasations of blood, tumors or tubercles of the brain, alcoholic poisoning, or external injury.* The most intense in degree, and at the same time often the most limited *cerebritis*, is from injury (*traumatic encephalitis*), not only from direct injuries affecting the brain after the skull has been penetrated, but numerous cases occur where the cranium has remained uninjured, and where there has only been an apparently slight contusion. In such cases concussion has been so great that the brain-substance has sustained a general bruise, small vessels being ruptured, so that blood escapes. Such a lesion may not at first induce any signs of injury, but afterwards encephalitis ensues. Other cases are the accompaniments of insanity. In some instances encephalitis has followed the suppression of a cutaneous eruption, or the accidental inclusion of a nerve by a ligature applied to a blood-vessel (LALLEMAND, quoted by SIEVEKING); in others, caries of the bones of the cranium, and especially of the petrous portion of the temporal bone, caused by *otitis*. Intemperance in alcoholic fluids is also a frequent cause of encephalitis, as well as of every other disease of the brain. Inflammation of the brain is sometimes brought about idiopathically, by exposure to the sun's rays in very hot summer days, especially in tropical climates. As a secondary disease, encephalitis is produced by cancer, tubercle, clots, or immature parasites, and by every other structural lesion incidental to the brain textures; such as, softening of the nervous substance, or any implication of it from without. It is sometimes a result of pyæmia.

When suppuration occurs, and an abscess forms, its most frequent cause is internal *otitis*—a lesion which often results from *scarlet fever*, *typhus fever*, *small-pox*, *measles*, or *scrofula*. The ear affection is usually either disease of the *tympanic* cavity or of the *dura mater* investing the temporal bone. The inflammation, which terminates in abscess of the cerebral substance, is usually the effect of inflammation of the membranes. The internal inflammation is generally observed to succeed quickly the suppression or disappearance of the external discharge. It is generally observed in cases of scrofula (CRAIGIE). Abscesses also occur as the sequelæ of inflammations of distant parts, as of *pneumonia*, *pericarditis*, *enteritis*. They may also occur as so-called metastatic abscesses, *pyæmia*, and as a result of traumatic injury.

Encephalitis occurs at any age; in childhood during the tendency to hydrocephalus; in adult age, from the action of morbid poisons, and from mechanical and other accidents; and in old age from causes incidental to the natural decay of the frame. Men are supposed to suffer in a larger proportion than women, probably from their greater exposure to the exciting causes.

The more common causes of myelitis are accidental violence, as blows or falls. Affections of the cord, however, sometimes occur idiopathically, and the constitutional causes producing them are undetermined. They have been referred to a suppression of the menses in the female, and to the suppression of a hæmorrhoidal flux in the male; while others attribute them to sitting in damp or wet clothes, to onanism, or to venereal excesses, and prolonged exertion in the erect posture without active movement, inordinate muscular exertion, the action of cold, and the development of tubercle. No age is exempt from *myelitis*, but it occurs more frequently from ten years old and upwards. It is most common, however, in adult age, and more frequently attacks the male than the female sex.

(*b.*) *Morbid Anatomy.*—1. *General.*—The essential elements of nervous tissues are in a large extent exempt from the more pronounced and active forms of disease. Acute inflammation, in particular, rarely attacks the true nervous elements, except in a secondary way. But as the proper nervous substance is everywhere surrounded and supported by connective tissue, it is in this connective tissue that the majority of the acute and chronic forms of inflammation commence. It is especially so in regard to the spinal cord, the *myelitis* of which is, for the most part, a *spinal* meningitis. But in cases of congenital encephalitis, it would appear that nerve elements do undergo active changes of multiplication under the influence of inflammatory irritation (VIRCHOW, 1865). Softened patches are to be found in the brains of such infants, where not only the usual fattily degenerate neuroglia cells are to be seen, but other cell-like structures, spindle-shaped and clear, and without nucleus or nucleolus, made by the swelling and severance of axes cylinders and nerve tubes. But such would seem to be a passive breaking up, rather than evidence of active vital multiplication. Under irritation the tubercular, or nerve elements never multiply, but waste away, or rapidly break down. The early stage of a pyæmic abscess shows itself as a patch of deep red softening, different from the so-called red softening (which results from embolism of cerebral arteries) in this respect, that in the red pyæmic patch the nerve-cells and fibres are seen breaking up into granules and dispersing. Muscular fibres behave in a similar manner. There is no attempt to multiply in either (MOXON, *Med. Times and Gazette*, Dec., 1870). The inflammation-process, alike in the brain and spinal cord, is of an interstitial character, leading to degenerations and disintegrations of the nerve structures; and not usually to the production of cell-elements by germination, nor to pus formation (MOXON, l. c.; KESTEVEN, *St. Barth. Hosp. Reports*, 1872). Interstitial cerebritis thus appears to be as definite a process as interstitial hepatitis or nephritis. It is, however, more difficult to demonstrate, and arises less easily. The encephalon is not very susceptible to common sources of irritation. Its highly specialised tissues do not reproduce themselves, nor multiply their elements under irritation or inflammation. Such processes tend only to their degeneration (CLIFFORD ALLBUTT).

2. *Encephalitis.*—The affected part acquires first increased vascularity, which increases to a bright red tint, deepening as the disease advances, until it assumes a reddish-brown, and occasionally even a brownish or green shade. In keeping with the inflammatory process, the parts affected lose cohesion, and become softer than natural, and may proceed to the formation of pus—a result more common in chronic or subacute forms of cerebritis, rather than in the acute, and most apt to occur in persons of scrofulous and unhealthy constitution, or as the result of injury or local disease, especially of bone (CRAIGIE). There is little interstitial exudation, connective tissue is scanty, and the most important changes take place in the nerve-filaments and nerve-ganglionic cells. They swell, in the first instance, and gradually break up into a detritus, with more or less abundance of the compound inflammatory corpuscle of Gluge. Vascular turgescence and extremely red congestion of the brain, as seen in *typhus*, the *exanthemata* generally, *epilepsy, delirium tremens, tetanus, sunstroke, hydrophobia,* bring the lesion very nearly, if not altogether, to the condition of inflam-

mation. Vessels greatly distended with blood are accompanied by a decrease of arachnoid fluid. It is only in cases where the finest vessels of the brain are distinctly seen injected with red blood, out of proportion to the congestion of central vessels in other parts, that hyperæmia can be said to exist. Distension of the large blood-vessels (which are veins), always seen on the surface of the brain when the calvarium is removed, is a normal appearance. The first fine ramifications of vessels are those supplying the *pia mater*, passing thence into the substance of the brain. It is in such vessels that hyperæmia is to be recognised, mainly by their number visible and their distension with red blood. In the normal condition they can scarcely be recognised by the naked eye. But the congestion of acute cerebritis goes a stage farther. The colouration is general, effusion of blood actually takes place in spots or foci, and the consistence of the texture is changed. A green shade in the colour of the morbid part of the brain is suggestive of the formation of pus; and in proportion as the colour passes from red to yellow or grey, before greenness sets in, associated with pulpiness or a semi-fluid condition, the lesion of red or of yellow softening is to be recognised. It is rare to find perfect purulent matter in a distinct cavity. Effusion of serous fluid generally attends the softening (MORGAGNI, ROSTAN, LALLEMAND, BOUILLAUD, BRIGHT, CRAIGIE). There is, at the same time, good grounds for believing that many recorded instances of brain abscess have been really examples of disintegration and loss of substance from intercepted blood supply, or perverted nutrition ending in local patches of white softening (DURAND FARDEL, BOUCHARD, CHARCOT).

The lesion may be either on the convoluted surface, to the extent of two or three square inches, or at the *septum lucidum* or *central ganglia*, the *cerebellum* or *cruces cerebri*; or the whole brain may be softened to a pulp. But the lesion is generally confined to certain parts; the entire brain is rarely if ever involved throughout. The spots of inflammation vary from the size of a pea to that of a turkey's egg, and are usually of an irregular spherical shape. There may be only one spot or several. Usually there is only one; but if there are many, they are generally small, and located in the cerebrum or cerebellum, in the grey substance or the white, or in both. The brain-substance affected is at first swollen by infiltration and hypertrophy of growth, then it softens, and becomes dotted over with red spots of extravasated blood. On section of the morbid part it rises above the level of the cut surface. The softening resolves itself into a red pulp, of a rusty-brown or yellow colour. It is so diffluent that it may be washed away by a light stream of water, leaving remains of nerve-tubes, blood corpuscles, compound granule-cells, and fine granular detritus, in single particles, or in masses. Cyst-like cavities are sometimes the result of such changes, filled with a sero-gelatinous fluid. Sometimes a characteristic cicatrix takes the place of all, which is at first pale, red, and vascular, but afterwards it becomes white and callous.

Various results follow the inflammatory process, just as in inflammation of other textures; and in the brain there are chiefly three to be recognised, namely—(1.) Red softening; (2.) yellow softening; and, (3.) abscess.

Pus is not always present in what appears to be abscess of the brain, but molecular granules and inflammatory exudation-corpuscles. Exudation in the softened state of the brain-substance, resembling pus, may be thus—(1.) Collected into the form of an abscess; (2.) it may be infiltrated into the substance of the brain; or, (3.) it may be detected on the convolutions, in the shape of a ragged ulcer, varying in size from a fourpenny-piece, or less, to that of half-a-crown. It is in this latter form and site of the suppurative process that well-formed pus-corpuscles can most frequently be detected. *In abscess of the brain* the surrounding substance is generally of the natural colour, except in a few cases in which it succeeds to apoplectic effusion, when the walls of the cavities are dyed by the previously extravasated blood. When the abscess is of a large size, the weight of the pus breaks down the neighbouring parts, and they look as if they had been destroyed by the pressure; and when the abscesses are small, there is an ulcerated appearance of the cavity in which the pus is contained (BAILLIE). In other cases the usual membrane of an abscess forms. This membrane is at first extremely delicate and easily torn; but as the patient continues to live, the membrane lining the abscess becomes of greater consistency, and even of considerable density, so that in some cases it is fibrous, fibro-cartilaginous, and even ossified, thus laying the basis for the formation of bony tumors of the brain. Recent abscesses of the brain form irregular round cavities, filled with a fluid more or less thick, glairy, yellowish, greyish, or greenish, and sometimes offensive. The walls of the abscess consist of ragged masses infiltrated with pus. In the immediate vicinity of the abscess there is usually inflammatory softening, and beyond that an area of *œdema.* Such abscesses increase till they open into a ventricle, or, reaching the meninges, extensive meningitis may occur. In other instances ulceration may take place through the cerebral membranes as far as the calvarium or skull bones. The ulcer may perforate the bone, or open into the cavity of the tympanum. In about a quarter of the cases the abscesses were multiple, and scattered in various parts of the brain. These are almost invariably pyæmic. The remaining three-fourths were cases in which solitary abscesses were found distributed as follows, in the order of their greatest number:—Left hemisphere, right hemisphere, cerebellum, *corpora striata,* pituitary body, *medulla oblongata* (LEBERT). On the other hand, the statistics of Sir William Gull and Dr. Sutton show that abscesses occur equally on both sides of the brain, and are most common in the middle cerebral lobes. The abscesses generally occupy the white substance, and for the most part only affect the grey matter by extension. In form these abscesses are generally oval, and vary in size from a pea to the size of a hen's egg or larger; so that one entire hemisphere has been found converted into a pouch filled with pus. The lateral ventricles are the parts into which the abscesses most frequently burst. Thickening of the *ependyma* follows, and scattered spots of inflammatory softening may be found in the neighbouring tissue. In some cases the abscess makes its way outwards through the petrous portion of the temporal bone or the aural passages.

3. *Myelitis.*—As the spinal cord is a continuation of the brain, and similarly composed of white and grey matter, it is reasonable to expect that its diseases will be similar. Such is observed to be the case. Inflammation of the cord may be diffuse. It is characterised, *post-mortem,* by a few more bloody points than usual, or by a slight red or rose-colour suffusion

throughout its substance. There is a much more marked tendency to blood extravasation than in cerebral softening. There is reason to believe that inflammation of the substance of the cord, like the corresponding affection of the brain, may terminate fatally, either—(1.) In the *acute inflammatory stage;* (2.) by *ramollissement;* (3.) by *undefined suppuration;* or, (4.) by *abscess.* The most common lesion is a ramollissement or serous inflammation, in which the substance of the cord is greatly broken down and softened to a mere pulp, or so diffluent as to give the sensation of fluctuation under the finger. This disorganisation may embrace the whole thickness of the cord, or sometimes only one of its columns. But, as a rule, it tends to implicate the whole thickness of the cord, and to involve extensive tracts, or short lengths of its substance. Idiopathic inflammation affects primarily and chiefly the central grey matter. The softening is constant, and the centre or *grey* substance of the cord is more softened than that of the circumference or white substance. The ramollissement may exist in the cervical, dorsal, or lumbar portions; but it is most common in the lumbar, and after that in the cervical portions, or in those parts which contain the greatest quantity of *grey* substance and the greatest number of blood-vessels. The part affected is generally swollen —more so than in similar lesions of the brain, because the spinal canal is large in proportion to its contents compared with the cranium. The softened part is generally ash-coloured or white.

When myelitis is secondary to disease of the membranes, the white substance of the cord is first implicated, whence, at a later period, it extends to the grey matter. Induration in patches of the spinal substance is another result of *myelitis,* and probably depends upon a form of inflammation in which fibrinous exudation becomes consolidated. Portal states he has found the cord of a cartilaginous hardness, while the membranes were red and inflamed; and Abercrombie gives a similar case (sclerosis or chronic inflammation). When myelitis is confined to circumscribed spots, it usually commences in the grey substance, and affects the whole thickness of the cord; or, commencing as "*central softening,*" it extends widely through the grey substance. The affected part is swollen, and on section is of a red pulpy appearance, rising above the level of the surface. By blood-extravasation and changes, the various coloured softenings are brought about, such as red, white, brown, or yellow. Abscess is rare.

Symptoms.—1. *Encephalitis.*—These are partly the result of irritation or destruction of the part of the brain affected, and partly the result of disturbance of the cerebral circulation, especially in the vicinity of the part, and therefore greatly influencing the functions of the brain. Large portions of the brain may be destroyed without causing perceptible functional disturbance, and the symptoms vary with the locality of the lesion. Many of the symptoms of cerebral meningitis are due to implication of the cerebral grey matter.

In idiopathic inflammation the case may be acute or chronic. The first stage of the acute form is generally of short duration, and in so far the attack may be said to be sudden. If there have been preliminary symptoms of ill-health, they have generally been headache or long-continued derangement of the digestive organs. *The general symptoms* of the first stage of encephalitis may be but feebly marked (unless *meningitis* predominates). There is usually, in the first instance, some heat of head and of surface generally. The face is pale, and the pulse low and irregular. The breathing is variable and sighing. There is slight feverish oppression, with headache, slow pulse, vomiting, and constipation. *The nervous symptoms* are generally well marked, and so are those of diminished functional activity in cases where the cerebral affection predominates as distinct from the meningeal. The patient is sullen, and his faculties become obscured. There is confusion of thought, rather than delirium, which occurs only in a mild form when the patient doses. Unless meningitis is present, there is no hyperæsthesia; but there is deep-seated, violent, oppressive pain, frequently described as shooting from the centre to the vertex, the temples, eyes, or ears. It is out of all proportion to the intensity of the febrile phenomena, and while the febrile phenomena subside (as they very generally do in the course of twelve or twenty-four hours) the local pain does not diminish.

The *second stage* may be ushered in by convulsions; or, after the first stage has lasted two or three days, the pulse usually becomes rapid and weak. The cerebral nervous functions become inactive: anæsthesia, paralysis, and coma commonly follow two or three convulsive paroxysms, and death follows after the first stage has lasted two or three days. When the signs of *meningitis* co-exist and are unusually severe, the pain is deep-seated, and followed, after twelve or twenty hours, by convulsions and coma, and there is commonly *meningo-cerebritis* of considerable extent. When the *cerebritis* is local or partial, the symptoms are those which are usually regarded as indicating "inflammatory softening"—"red softening," as it is commonly called, or "acute ramollissement." Premonitory symptoms are common—namely, some loss of intellectual vigour, failure of memory, confusion of ideas, irritability of temper, and a consciousness of weakness—these symptoms becoming gradually persistent. There is pain of the head, dull, deep-seated, fixed, and protracted; tingling or numbness in one limb or side; imperfection of the special senses; dimness of sight; dulness of hearing. There may be loss of power of one limb or side—the most important of all the premonitory symptoms. There is more or less fever in proportion to meningitis: there is heat of head, vomiting, and general malaise. The signs of meningitis, on the other hand, may be feebly marked; but there may be convulsions, followed by coma, partial paralysis, with rigidity returning more or less rapidly, and ending fatally in a day or two, or from two to three weeks. Thus there may be the symptoms of meningitis, or of cerebritis, of both together, or alternately; and, occurring with such premonitory symptoms as have been indicated, they leave little doubt of the existence of a partial cerebritis. In general cerebritis the symptoms may be almost negative, or a little more than a gradual failure of all bodily and mental power, as in cases of *typhus fever.* If one part of the brain should be affected more than another, there will be a special paralysis; otherwise the symptoms are little more than weakness of the limbs, strange feelings, and interference with the intellectual faculties, with perhaps giddiness, sickness, headache, and other symptoms common to all febrile disorders (WILKS).

Usually the symptoms of *cerebral irritation* precede those of depression. *Cerebral irritation* expresses itself more prominently, at one time in the *sensory*, at another time in the *motory*, or at another time in the *mental* functions. *Sensory symptoms* are manifested by *headache*, which is a frequent but not essential symptom of all cerebral diseases. Light causes inconvenience; a slight sound, or irritation of nerves by touch, causes unpleasant sensations. The excitation is morbid *ab initio*. Expression of cerebral irritation by *motorial symptoms* consists of restlessness, sudden starting, gnashing the teeth, crying out, twitching of single muscles, and general convulsions. The expression of cerebral irritation by *mental states* mainly consists in a rapid change and loose connection between the thoughts, so that clear thinking is impossible. Ideas are thus confused, so that the patient has false notions about himself and the world generally. He is, in a word, "delirious." So real and intense is the delirium, that it cannot be distinguished from true perceptions. Hence he has *hallucinations* and *illusions*. *Dizziness* is a form of hallucination. It is a vivid representation of the movement of bodies surrounding the patient, or of the body of the patient himself—all being really at rest at the time. Conditions directly opposed to these are indications of cerebral depression.

The extreme local lesion of actual *cerebritis* is indicated by *partial anæsthesia, partial paralysis,* and *loss of certain mental functions.*

2. *Myelitis.*—The symptoms are, in general, expressed by paraplegia—*i. e.*, by paralysis of the parts below the lesion. In a few cases, however, the effects of the accidents are reflected from below upwards. In general, both upper or both lower limbs are affected; but in a few instances only one limb. The earliest symptoms are recognised in the fingers and toes, in the feeling of numbness, with a sensation of coldness extending up the limb. Shortly afterwards the patient complains of pain in the back, corresponding to the seat of greatest intensity of the inflammation. This is not constant; but when we make pressure with the finger over the spinous processes of the affected part, it may be augmented or only then felt. These symptoms are succeeded by impaired motion, and often likewise by diminished sensation of one or more limbs, followed by paraplegia or other form of palsy. If only one side of the cord be affected, the paralysis which results is confined to one side of the body. When the anterior columns chiefly are the seat of the inflammation, the paralysis which follows is that of muscular motion, but of sensation if the lesion exist in the posterior columns; and if a careful analysis be made of the several cases in which the grey substance of the cord has been implicated, it will be found that the function of reflex action has been deranged (MERYON). In the early stage, when congestion prevails, there is exaltation of tactile sense and of muscular contraction. When myelitis proceeds from meningitis, motion is lost in greater proportion than sensation. Another marked symptom is a difficulty experienced in walking on first rising after a night's rest—a feature more or less constant in cases of spinal congestion. The extensive involvement of grey matter soon leads to peripheral nutritive lesions, especially, first, to wasting of the muscles. The palsied limbs may be either relaxed or permanently contracted: thus, the hand may be bent on the upper arm, or a leg be flexed upon the thigh, or the affected limb may be attacked with convulsive twitchings, or may beat incessantly. As the

disease advances, the urine becomes ammoniacal, the bladder and kidney become inflamed, and the patient is incapable of retaining his urine. The action of the bowels is slow in the first instance; but towards the close of the disease the patient may be purged, and the stools pass involuntarily. If the disease be the result of an accident, the pulse is at first rapid and full; but if the disease be spontaneous, the pulse is generally natural, until the powers of life are broken down by the continuance of the affection. The nates and the prominent parts of the pelvic region, on which the body rests, ulcerate extensively, so that deep sloughs form, sometimes within four or five days of the commencement of paraplegia. Priapism is occasionally present.

Diagnosis.—If symptoms of local brain disease commence after an injury, or if there be disease of the bones of the skull, such as the petrous bone, followed by "*head symptoms,*" it is probable that encephalitis has commenced. Diseases of the spinal cord and diseases of the brain are often followed by nearly similar symptoms; and, consequently, the one may be confounded with the other. But the history of the case, whether it has or has not been preceded by a fit of apoplexy or of epilepsy, will often enable us to determine the particular seat of the disorder. The antecedents and concomitant circumstances of every case must be carefully inquired into, and judged of upon their own merits. Myelitis is distinguished from lumbago, psoas abscess, and hip disease, by the absence of pain, and by the existence of palsy.

The characteristic symptoms of paralysis, as induced by destructive myelitis, are as follow:—(1.) Pain over that portion of the back which corresponds to the seat of inflammation. (2.) Lesions of sensation, giving rise to feelings of formication, creeping, prickling, tingling, heat or cold, to numbness or complete anæsthesia. (3.) A gradual and progressive diminution of muscular power, distinguishing it from the paralysis which the French have denominated "*ataxie locomotrice progressive.*" (4.) An equable degree of paralysis in all the muscles which are implicated; for as in health the nerve-force is distributed to whole groups of muscles in an equal degree, so likewise is it annulled when the nervous centre is disorganised. (5.) Convulsive and reflex movements of the paralysed muscles. (6.) Spasm or paralysis of the rectum and bladder. (7.) Alkaline urine. And, finally, (8.) The loss of electro-muscular contractility (MERYON, l. c., p. 35).

The latent character of brain abscess is important in diagnosis. Sudden headache is the symptom which most frequently excites attention; and it is generally accompanied by febrile symptoms and vomiting. The patients become heavy, morose, and may be delirious, with contraction of the pupils and photophobia. Difficult articulation, numbness, formication, and convulsive attacks may supervene. While the intellect may suffer comparatively little, sensibility suffers more frequently; and headache, generally at first diffused, is more or less intense, and subsequently becomes unilateral. Coma occurs frequently, is often temporary, and paralysis occurs in about one-half the cases. The paralysis is generally local; but it may assume the form of general muscular debility. The duration of such cases fluctuates from two or three weeks to two months (LEBERT, SIEVEKING, in *Med.-Chir. Review*, 1857, p. 526).

Prognosis.—There seems no reason to doubt that, as many perfectly recover from superficial inflammatory lesions of the brain after fever, so also many slight inflammatory affections of the substance of the cord may subside, and the patient do well. Many cases, indeed, even when the bladder is slightly affected, recover. If, however, the disease be of more than a few weeks' continuance, the prognosis is always grave. Still, some few cases recover, the palsied limb becoming withered. But more commonly the disease runs on, and the patient at length dies after a long illness. When paralysis has once supervened, there is great reason to fear that the inflamed portion of the cord has passed into a state of disorganisation, and that the disease is incurable; but the prognosis also eventually depends in some degree on the precise seat of the lesion. If it be in the cervical region, the immediate danger is greater than when the lesion is in the dorsal region; in this latter, again, the prognosis is more unfavourable than when lower portions of the spinal marrow are affected; and when the patient retains the command over the motions of the rectum and bladder, and the acid character of the urine is persistent, the case is still more hopeful (MERYON).

Treatment.—1. *Encephalitis.*—So soon as there are any circumstances which may lead to "head symptoms" after injury, it is well to commence treatment at once with local antiphlogistics, leeches, and evaporating lotions, such as *muriate of ammonia* in solution with *vinegar*, applied by cloths, which are to be frequently changed. The patient must be made to observe a rigidly abstemious regimen, and rest with the head elevated above the shoulders. Purgation is to be instituted by means of watery evacuants, and the same general treatment is to be adopted as in meningitis.

2. *Myelitis.*—In classing ramollissement of the cord with inflammation, it might appear to infer that the treatment should be strictly antiphlogistic. It may be laid down as a general rule, however, that bleeding ought not to be had recourse to after palsy has occurred. Previous to that symptom it may be admissible; and it may be stated generally, that so long as the affected muscles are convulsed, rigid, and irritable, the use of antiphlogistics and counter-irritants may be indicated; but when the means calculated to subdue excitation have failed to arrest the further progress of the disease, and paralysis supervenes, stimulants are the only remedies which have the power of restoring to functional activity those nerve-cells and conducting fibres which are not irretrievably destroyed (MERYON). The chances of saving the patient by other antiphlogistic remedies mainly rest on acting on the alimentary canal so as to produce three or four motions in the twenty-four hours, and thus create such a derivation as in some degree to relieve the parts. The greater number of patients that recover are restored by these means. The particular purgative is not perhaps important; but as the neutral salts act not only on the intestines, but also on the bladder, that class of remedies is generally preferred.

With respect to local counter-irritants, as blisters, moxas, or setons, little favourable can be said, unless they are employed previous to paralysis, as the tendency to gangrene renders their application of doubtful utility. When had recourse to, however, it will be found better to apply them above the seat of the disease than immediately over it, the greater vitality of the superior parts giving more assurance of the disposition of the wounds to heal. Of all stimulant remedies, *electricity* and *strychnine* are the most potent and the best. *Secale cornutum* has been recommended as a remedy possessing the same power as *strychnine* (BARBIER, PAYEN, MERYON). When there is no great pressure beyond that which simple congestion produces, nor actual disorganisation of the spinal cord, the remedial power of *secale cornutum* is said to be very great. It seems especially to resuscitate the muscular contractility of the rectum and bladder, and pelvic viscera generally (GUERSANT, TROUSSEAU, BROWN-SÉQUARD, MERYON). The *ergot of rye* may be given in the forms and doses as follow :—(1.) *Ergot*, 5 to 10 grains, infused in boiling water, three times a day; (2.) *Ethereal tincture* (not now in the pharmacopœia), *in doses of from ten to twenty drops, twice or three times a day;* (3.) *Extractum ergotæ liquidum*, 15 to 30 minims; (4.) *Infusum ergotæ*, made fresh on each occasion, 1 to 2 ounces; (5.) *Tincturæ ergotæ*, 15 to 60 minims. It does not relieve the reflex convulsions, which are sometimes alleviated by *Prussic acid, digitalis*, or *belladonna* (MERYON, l. c., p. 40), *chlorodyne* or *chloro-morphine.**

After the local pain in the back has been subdued (by the regular and repeated application of two or three leeches to the painful part, followed by a large warm poultice over the whole length of the spine, and a belladonna plaster of equal length to follow it, or an occasional blister on each side of the spine, together with mild, warm purgatives, if necessary), Dr. Meryon has found no remedy so effectual as *strychnia, in the dose of one-twentieth of a grain, repeated more or less frequently* (*twice or three times a day*), *according to the evidence of its action.* It may be combined advantageously with *ipecacuanha* in cases where the intestinal mucus seems deficient. The absence of pain and of spasmodic muscular contraction necessitates great caution in determining the precise moment when the spinal cord is likely to be benefited by the energetic excitement of *strychnia.* The internal administration of this remedy ought, therefore, to be always preceded by its external use, together with other stimulants, in the form of embrocations over the spine, when the stage of excitation has been subdued. In cases with a history of syphilis, and where there may be some reason to believe that hardening or induration of the cord or its membranes has taken place, the *iodide of potassium* may relieve the early phenomena, and by the aid of *setons* the progress of the disease may be held in abeyance, so long as the discharge is maintained from the *seton.*

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* Conflicting statements exist in text-books on Materia Medica as to the best form of ergot for internal administration. 1. Ergot contains one-third of its original weight of oil, got out by percolation with ether (SQUIRE). 2. It contains a large quantity of fixed oil, about 75 per cent. (GARROD). 3. It contains 25 to 28 per cent. of fixed oil (STILLÉ and MAISH). Ergot preserves its virtues not much over a year (STILLÉ and MAISH). The active principle is a body somewhat resembling gelatine, formed from the gluten of rye ovary, and readily changed by the influence of chemical agents (BUCHEIM). A number of non-crystallisable compounds, possessing more or less activity, have been isolated from *ergot*, the most important being *sclerotic acid* (to the amount of 4 and 4½ per cent.), the other, *scleromucin*, to the extent of 2 to 3 per cent. This multiplicity of uncrystallisable principles may explain why preparations possessing some activity may be obtained from very different processes—*e.g.*, Wiggers' *Ergotine*, obtained in 1830, is soluble in alcohol, but insoluble in water and ether, and is said to have poisonous properties. Bonjean's *Ergotine* (*Germ. pharmacop.*, as "Extr. Secalis Cornuti") is a concentrated aqueous infusion, precipitated by half its weight of alcohol, and the clear filtrate evaporated. Wenzell (1864) proved the presence of two alkaloids (*ecbolina* and *ergotina*). They exist in combination with *ergotic acid.* These have been since shown to be identical, and to possess no decided physiological action (DRAGENDORFF and BLUMBERG). In 1875 Tannet isolated another alkaloid, *ergotinina, from the fixed oil of ergot prepared by ether*, by agitating it with acidulated water. Fresh ergot yields ·12 per cent. of *ergotinina*, which is crystalline, but soon becomes resinous. As regards the properties of the oil extracted from ergot by maceration with ether, Stillé and Maish write that "it displays even greater energy in producing the same symptoms, and in addition other symptoms (which are specified), than does ergot itself. The

When disorganisation of the spinal cord has become an accomplished fact, the disease is incurable; but yet the exigencies of the patient are not the less pressing on the careful attention of the physician, and in nothing more so than in the protection which is called for against bed-sores, which will sometimes occur in spite of the greatest care (MERYON, *op. cit.*, p. 40).

Electricity, after the activity of inflammation has been subdued, is a therapeutic agent of great value; and the continuous current of galvanic electricity seems to be just as efficacious as the induction or intermittent current. But whether galvanism or electro-magnetism be employed, no high degree of tension is required for the restoration of muscular power; on the contrary, Dr. Meryon justly believes that the favourable course of many a case has been retarded by the employment of strong currents. Dr. Althaus, also, is in favour of weak currents.

CEREBRAL AND SPINAL HÆMORRHAGE: APOPLEXY (CEREBRAL AND SPINAL).

LATIN EQ., *Apoplexia;* FRENCH EQ., *Apoplexie;* GERMAN EQ., *Apoplexie* —Syn., *Hirnschlag;* ITALIAN EQ., *Apoplessia.*

Definition.—*Hæmorrhages within the skull or spinal canal, consequent on the rupture of blood-vessels in the membranes or substance of the cerebro-spinal nervous system.*

Pathology.—(*a.*) *History.*—It has been usual hitherto to describe cerebral

effects continue for several days;" but they add that "the oil exhibits no influence upon the gravid uterus, but the residual ergot from which it had been extracted seems to possess the ecbolic power of the drug unimpaired" (*The National Dispensatory,* p. 545). It does not appear, therefore, that we are warranted in rejecting the *ethereal tincture* of ergot as an inert preparation, as Mr. Squire has represented it to be (*Compan. to Pharg.*, 1871). It may still be the best form of remedy for some cases, although it has become obsolete as a pharmaceutical preparation in our pharmacopœia; while the residual ergot, possessing ecbolic powers, may be also useful for other purposes. Besides, Mr. Squire states that in the directions for preparing the *Extractum ergotæ liquidum* (doubtless a valuable and energetic preparation when properly made from fresh ergot), the ether ordered is not sufficient to extract all the oil. The quantity, he states, ought to be doubled. So that this energetic preparation, if prepared as directed in our British Pharmacopœia, still contains half the oil left by the insufficient quantity of ether which is used, so that the whole of the oil is not now rejected in preparing the *Extractum ergotæ liquidum.* A writer in *Medical Times and Gazette* (Oct. 16, 1875), under the signature of "Eyes Right," has described how "teachers differ" on matters of fact, and gives the teachings of Squire, Aitken, and Garrod, on "Ergot" as an example. But from what I have quoted from the most recent teachings of Stillé and Maish, I do not find we are justified in excluding the *Ethereal tincture of ergot* as an inert preparation.

hæmorrhage by one of its clinical symptoms, namely—"Apoplexy," of which it is by far the most frequent cause. Apoplexy is thus one of the symptoms of cerebral hæmorrhage, and is a condition essentially characterised by the sudden loss, more or less complete, of volition, perception, sensation, and motion, depending on sudden pressure upon the brain (the tissue of which may be morbid), the source of pressure originating within the cranium. Not fewer than 12,000 persons die annually in England and Wales of cerebral hæmorrhage: and we may assume that at least twice as many people living in these parts suffer from the effects of it. It constitutes, after convulsions, the most fatal of all diseases of the nervous system, carrying off year by year, with unerring certainty, more victims than either paralysis, epilepsy, or insanity. It must be ranked with the most important diseases which the physician is called upon to treat; and as it threatens the life chiefly of the aged and the middle-aged—*i.e.*, the heads of families—its importance becomes thereby considerably enhanced; for the social position of entire families may be, and often is, suddenly and completely altered by a death from apoplexy (ALTHAUS).

(*b.*) *Causation.*—(1.) Cerebral.—Among the most frequent causes of cerebral hæmorrhage, is an intemperate use of fermented liquors, which powerfully excite mainly by inducing paralysis of function, thus allowing the evidences of excitement to appear as the prominent phenomena (ANSTIE). Alcohol also acts specifically on the heart and arteries, causing not only temporary energetic actions of those parts, but also organic alterations in their structure. In the latter case the powers of the heart are permanently augmented, while the coats of the arteries, thickened, thinned, or ulcerated, have their elasticity thereby destroyed. The tendency to hæmorrhage in the brain is increased. The excessive use of narcotics, as opium or tobacco, is also supposed to predispose to congestion of the brain, and consequently to cerebral hæmorrhage. The following conclusions have been arrived at from a comprehensive view of numerous cases:—(1.) That in by far the greater number of cases, cerebral hæmorrhage is due to degenerations or aneurismal dilatations of the cerebral arteries as its predisposing cause. In the smaller arteries these degenerations consist of fatty metamorphosis, or simple atrophy, with the various forms of consecutive aneurismal dilatation; while in the larger arteries of the base there is *arteritis*, issuing in ossification, or fatty degeneration, or *atheroma*, or passive calcification. (2.) A not entirely rare cause are true aneurisms of the large cerebral arteries, such as the middle cerebral or basilar. (3.) Hypertrophy of the left ventricle will only favour cerebral hæmorrhage when it permanently increases the normal tension of the aortal system; but this is not the case in compensating hypertrophy of vavular disease of the heart. (4.) In about one-seventh of all cases of apoplexy, neither predisposing diseases of the heart nor of the vessels could be demonstrated (EULENBERG in VIRCHOW'S *Archiv.*, Vol. XXIV., p. 329; and *Syden. Soc. Year-Book* for 1862, p. 81). The predisposing causes of cerebral hæmorrhage may, therefore, be classified under two sets of phenomena, namely—(1.) Increase of blood tension, especially in the vessels of the arterial system; (2.) disease of the walls of the vessels themselves (NOTHNAGEL). The diseased condition of the arteries is mainly a sclerosis of the arterial walls; and (as in other parts of the body such sclerosis) gives rise to aneurisms—minute or miliary aneurisms—of the blood-vessels of the brain, as Charcot and Bouchard have demonstrated—the sacular dilatations of Virchow. These "miliary aneurisms occur regularly or constantly in cases of so-called spontaneous hæmorrhage, the hæmorrhage taking place in consequence of the rupture of these minute formations, which constitute a disease by themselves. These observations have been confirmed by Vulpian, Barth, Béhier, Lionville, Zenker, and Roth. Such aneurisms are of a reddish colour, visible to the naked eye as dark red granulations, varying in size from that of scarcely visible bodies, or from the size of a millet seed to that of a pin's head. They are situated on the arterioles, the middle coat of which is atrophied, while the connective tissue of the adventitia is hypertrophied. Few may be found; but sometimes they are scattered through the entire brain, to the number of one hundred and upwards. Their relative frequency of occurrence in certain regions of the brain is constant; most are to be seen in the fissures between the convolutions, the thalamus opticus, and corpus striatum; next in order of frequency, in the pons Varolii, centrum ovale, middle cerebellar peduncles, cerebral peduncles, and medulla oblongata. These sites also correspond with what is known to be the relative frequency with which hæmorrhagic lesions occur in those different regions of the brain. They are found in those who die from other diseases, but who with such a tendency have nevertheless escaped from cerebral hæmorrhage. They rarely occur before forty years of age; but subsequently they increase more rapidly with advancing years, and at a more rapid rate than the age of the patient. The nature of the process which leads to these miliary aneurisms is of the nature of a chronic periarteritis. Zenker has shown that the inner coat of these miliary aneurisms, and of the arterial twigs bearing them, is found to be the seat of circumscribed thickenings, associated with other changes peculiar to arterio-sclerosis. Gowers has seen them *during life* on small arteries of the retina in Bright's disease (*Medical Ophthalmology*, p. 16).

The connection of cerebral hæmorrhage with advanced disease of the kidney has occasionally attracted the attention of pathologists; and the late Dr. S. Kirkes brought forward facts in proof of the frequency with which the kidneys are found diseased in fatal cases of cerebral hæmorrhage; and which, to a certain extent, explained the connection between the renal and cerebral affections (*Med. Times and Gazette*, 1835, p. 515). In all the cases except one the heart was found enlarged—an enlargement due to the prolonged disease of the kidney—and generally in the form of hypertrophy of the left ventricle, without vavular disease. The primary disease is that of the contracted granular kidney; then the heart and the blood-vessels become diseased in consequence of the primary affection. The disease of the blood-vessels is chiefly in the form of the well-known yellowish-white thickening and deposit within the coats of the blood vessels,—diffused arterio-sclerosis. Dr. Kirkes believed that the disease o the vessels is in a great measure the result of the continual over-distension and straining to which the arteries are subjected by the unwonted energy with which a hypertrophied heart propels the blood along them—th full force of the hypertrophied ventricle, independent of vavular disease being exercised upon the arterial current. When local changes in th vascular walls of the blood-vessels of the brain exist, then it is that the increase of pressure is more active in bringing about rupture and hæmorrhage than any other influence (TRAUBE, EULENBERG, NOTHNAGEL). Ordinary hypertrophy of the left ventricle, from aortic valve disease, does not lead to cerebral hæmorrhage.

Extremes of temperature are likewise powerful predisponents to apoplexy. In summer the fluids of the body tend to produce turgidity of the vessels in some constitutions, and the tone of the capillaries is impaired; while in winter the cold drives the blood from the periphery of the body to its central organs, and consequently to the brain, increasing the tension in the cerebral arteries. But cerebral hæmorrhages are least frequent in summer, and occur most frequently in winter.

The powerful effects of *moral causes* in producing this fatal disorder are well known. *Mechanical obstruction* is also a frequent occasion of cerebral hæmorrhage. If an obstacle, for example, be opposed to the course of the blood, as when the valves of the heart are diseased, the blood accumulates in the capillary system generally, and consequently in the brain; or errant clots of fibrine choke up minute cerebral vessels or sinuses, leading to venous stasis consequent on thrombosis. The bursting of aneurisms attached to the larger arteries at the base of the brain may also give rise to profuse bleeding. Rupture of such large aneurisms of the basilar and middle cerebral arteries gives rise generally to hæmorrhage into the meninges. *Mechanical violence*, also, may produce apoplectic effusion. Thus, concussion of the brain, however slight, always produces more or less congestion of that organ; and, if severe, effusion may take place in the subdural space (*hæmatoma, pachy-meningitis hæmorrhagica*), or between the membranes, or into the substance of the brain, which may be extensively ruptured, or the *septum lucidum* torn.

Cerebral hæmorrhage producing apoplexy has been known to occur even in childhood. Billard gives the case of a child three days old that died apoplectic from effusion into the left hemisphere and about the lateral parts of the *corpora striata*. Serres records a similar case in a child three months old. Nothnagel has observed several such cases. A large number of infants die in the first year, mostly from meningeal hæmorrhage. Of cerebral hæmorrhage there are hardly any instances between the first and fifteenth year of life. After fifteen they are few; but at thirty-five there is a perceptible increase (ALTHAUS). Cerebral hæmorrhage producing apoplexy, however, is extremely rare before the fortieth year, and only a few cases are met with before twenty. It is not unfrequent between thirty and fifty, while between fifty and seventy it is one of the most frequent causes of death. According to Dr. Burrows, the relative frequency of apoplexy increases constantly from twenty to eighty years of age; and the absolute number of cases increases up to seventy years, in spite of the relative smallness of the population at that age.

Both sexes are liable to this affection, and in nearly equal proportion. Those most liable to attack are the florid in complexion, of short-necked conformation, with prominent eyes, broad chests, with protuberant bellies, sometimes enormously fat, especially if high livers, sedentary, and indolent. But many thin persons, with spare long necks, also frequently die from apoplexy; for they, too, are similarly liable to the diseases of the arteries. The act of digestion, or rather fulness of the stomach, appears to predispose to cerebral hæmorrhage. Numbers are attacked after dinner. Sleep, also, associated with a temporary congestion of the vessels of the brain, is another predisposing condition. Thus, of 176 cases examined by Genderin, 97 had been attacked during sleep. In the sleep of a healthy person the brain is anæmic (DURHAM). Many diseases, or conditions of the body, predispose to apoplexy, as mania, epilepsy, suppressed hæmorrhoids, amenorrhœa, and especially the "*turn or change of life.*"

2. *Spinal hæmorrhage* occurs usually as small ecchymoses, accompanying excessive hyperæmia, and generally traceable to injuries of the spinal meninges. In some cases such hæmorrhages are the result of chronic degeneration of the cord. Extravasations of blood from meningeal hæmorrhage of the cord are found chiefly in the lower part of the spinal canal, and often fill the subarachnoid space. The changes undergone, alike by the cord substance and the blood clot, are similar to those described as going on in the encephalon, under similar circumstances.

(*c.*) *Morbid Anatomy.*—The essential phenomena of the *apoplectic state* are due to a variety of local lesions, and not to any constant lesion. One or more of the following may induce the apoplectic condition, namely,—(1.) *Congestion of the brain,* or what is commonly called *determination of blood to the head—congestive apoplexy.* (2.) *Hæmorrhage,* or *extravasation,* (a.) *into the substance of the hemispheres* or *cerebellum,* (b.) *into the ventricles,* or (c.) *into the arachnoid cavity. One or other of these lesions constitutes apoplexy* in the common acceptation of the term—*sanguineous apoplexy* or *cerebral hæmorrhage.* (3.) *Sudden serous effusion in large quantity* is equally efficient in bringing about the apoplectic state, commonly called *serous apoplexy.* (4.) *Local cerebritis,* or *softening of the brain,* produces, as already shown, symptoms, in the first instance, of an apoplectic kind. So also do, (5.) *Tumors of the brain,* or *meningitis.* (6.) *Tuberculous meningitis.* (7.) *The progress of various specific and constitutional diseases from blood poisoning.* (8.) *Anæmia,* as in the *hydrocephaloid disease of children, disease of the heart, and vascular obstructions.*

To the phenomena produced by the first three of these conditions only has it been common or usual to apply the term *apoplexy.* Although much stress is laid upon the fact that "a sudden pressure upon the brain is necessary to produce the apoplectic state," yet it is difficult in all cases to account for the proximate cause of the disease by such an explanation; for when the quantity of blood extravasated has been not larger, for instance, than a barley-corn, it is difficult to account for all the phenomena by mere pressure. The ideas or theories which have thus been formed to account for the apoplectic state may be shortly stated as—(1.) The result of sudden pressure, effected by causes within the cranium. (2.) From a peculiar morbid state of the nervous matter of the brain, which predisposes to the extravasation of blood (Drs. ROBERT WILLIAMS, WOOD, SIEVEKING)—an apoplectic orgasm, as it has been called by some, but which is probably of the nature of softening (ROCHOUX). (3.) A morbid condition of nerve-matter and minute blood-vessels, also probably of the blood itself (BOUILLAUD, PAGET), and in connection with chronic renal disease (Bright, Burrows, Christison, Kirkes). The cerebral hæmorrhage generally takes place from the rupture of a small or miliary aneurism or of capillaries, and there is generally structural disease of the arterial walls, as well as a morbid condition of the brain-substance surrounding the diseased vessels, combined with increased pressure of blood from some temporary cause—temporary plethora—such as may occur during prolonged and luxurious meals. The cases of cerebral hæmorrhage are thus practically divisible into two sets:—(1.) Those where the extravasated blood is collected into a mass or focus (apoplectic focus). (2.) The capillary or aneurismal *apoplectic foci* vary in size from a large pea to a mass so large as almost to destroy completely an entire hemisphere; and in examining apoplectic cases after death, it is not unusual to find a cavity scarcely bigger than a barley-corn in the substance of the brain (the evidence of the primary attack), containing a clot of blood variously changed. In severe cases the quantity of blood effused will sometimes fill the whole cavity of the arachnoid, or extensively rupture the substance of the brain, forming a cavity as large as a nut or an egg, or even lay the ventricles into one cavity. The number of apoplectic foci may be two, four, or more, occurring simultaneously, and in symmetrical parts of the brain.

The appearance of the blood effused into the substance of the brain varies according to the time which elapses before the patient dies. If that event takes place in a few hours after the attack, so that the blood is still fresh, the *focus* is seen as a dark red clot, generally uniform throughout. The substance of the brain, likewise, has no other appearance of disease than that of being flattened, from the presence of the extravasated blood. If the patient, however, survives a few days, the membranes show marks of inflammatory action. They are injected, thickened, and although dry and pitchy-like in the immediate neighbourhood of the clot, have yet some serum effused in other parts of the space they inclose. The convolutions of the affected part of the brain are likewise now not only flattened but softened. "The injured brain-substance forms with the extravasated blood a sort of gummous mass. The wall of the focus consists, at the part nearest the clot, of torn shreds of cerebral tissue; then follows a zone of varying thickness, averaging a few lines, made up of softened tissue, saturated with blood serum, and often the seat of capillary hæmorrhage. Within the central mass itself, close search will generally discover the remains of ruptured vessels. If the plan is adopted of floating out the mass under water (which should be repeatedly poured off, and again renewed), it will be found possible to pick out the very miliary aneurisms from which the primary extravasation took place" (Bouchard, quoted by Nothnagel). Thus various processes immediately commence in the blood after it is extravasated, as well as an interchange of processes between the blood and the surrounding brain-substance. The most obvious of these phenomena are, the solution of the clot and the formation of blood-crystals; the gradual absorption of the more fluid constituents; the formation of an organised membrane round the clot; continuous absorption of the exudation; induration of the surrounding cerebral parts; contraction of the cavity, and ultimately the formation of a cyst or of a cicatrix. These changes are effected with various degrees of rapidity, depending on the site and extent of the extravasations, the healthy state of the nervous texture, and of the patient constitutionally.

When hæmorrhage has taken place into the substance of the brain, if the patient has died apoplectic, the hæmorrhagic cavity is found filled with half-coagulated blood, its walls irregularly softened, and dyed to the extent of some lines deep with the colouring matter of the blood. A small stream of water directed upon this part at once removes the extravasated blood, and also a layer of softened cerebral matter. If the patient has survived a week, the blood is found coagulated, and the serum set free; but the presence of the clot has caused inflammation, so that the walls of the cavity are not only discoloured, but more decidedly softened, and are softer in proportion as they are nearer the clot. If life be prolonged till about the fifteenth day, the serosity is absorbed, but the walls of the cavity are still of a deep red. About the seventeenth day Virchow discovered blood-crystals, or hæmatoid crystals, in the cavity. Such blood-crystals were first discovered by Sir Everard Home, and have been more recently described, and their nature explained, by Funke, Kunde, Lehmann, Beale, Parkes, and Sieveking. The crystals do not form from clotted blood until the blood-corpuscles have become ruptured by endosmosis. The contents of the corpuscles then escape and crystallise as the solution gradually becomes concentrated (Beale). Thus another guide to the age of the clot exists in the presence of these crystals. About the thirtieth day, if the patient lives so long, the clot is isolated, and a membrane forms by the new formation of connective tissue growing from the neuroglia. At first this membrane is muciform, fragile, intermixed with particles of cerebral matter, and also with some of the colouring matter of the clot; but by degrees it becomes more consistent, thick, and hard, and incloses the clot, which diminishes, and some serum is probably secreted by the new membrane surrounding it. Eventually a new formation of delicate connective tissue and serous infiltration, coloured yellow by the pigment of the clot, covers the walls of the clot-cyst, and breaks up the clot into a fine network. The cyst has also been found fully formed, organised, and nearly empty, by the thirteenth day (Macintyre); and by the seventeenth day after extravasation it has been found to contain sanguineous fluid (Moulin, Sieveking). The cerebral walls surrounding the cyst, previously softened, now become indurated, and are stained yellow, from the usual changes which the extravasated blood undergoes—a colour, however, which they ultimately lose. The cavity thus formed may be filled at length with serum only; or, the serum being absorbed, the membranous cyst may ossify, and be thus converted into a bony tumor. At other times the opposite sides of the cavity unite by a kind of areolar membrane, inclosing rust-coloured pigment, which forms a species of apoplectic cicatrix, made up of dense connective tissue. Sometimes we find but one, sometimes two, and in a very few instances three or more cavities. When many of these cavities exist in the brain, it is rare to find them all in the same state. Some are old and almost obliterated, others are fresher, and others again quite recent, their different stages marking a distinct and different period of attack, thus furnishing evidence that the same patient may be the victim of repeated seizures. An account of an interesting case of this nature is given by Dr. Fuller, in which there were *six* clots, each of a different date, and in different stages of discoloration, and corresponding to each of six well-marked apoplectic seizures in the course of nine months (*Diseases of the Chest*, p. 602). Some of the most exact data we possess regarding the pathology of apoplectic seizures are to be found in the *Transactions of the Pathological Society of London*. In *fourteen* cases in which lesions of the brain connected with apoplectic seizures were shown at the society, the position of the blood effused was as follows:—(1.) Superficial meningeal effusions; (2.) Effusions within the substance of the hemispheres or the *central ganglia;* (3.) Effusions in the *pons Varolii;* (4.) Effusions in the *pons Varolii* and *crura;* (5.) In the *pons Varolii* and *cerebellum;* (6.) In the *pons*, *crura*, and *cerebellum.*

(1.) In *superficial* or *ventricular* extravasation the blood lay in a more or less coagulated mass under the arachnoid over the hemispheres, and was most abundant at the base and about the roots of the nerves. In two of the cases there was evidence of previous *meningeal* disturbance—namely, adhesion of the cerebral arachnoid and *pia mater* to the convolutions along the anterior margin of the middle line of the hemisphere; the history of another case recorded the occurrence of two previous apoplectic seizures. In it *post-mortem* evidence of previous irritation also existed in the adhesion of the *dura mater* to the calvarium. All the cases appear to have been the result of morbid states of the vessels of the *pia mater.*

(2.) In *extravasation into the substance of the hemispheres or central ganglia* some part of the cerebral substance contained the extravasated blood more or less free, or surrounded by a cyst. In two of the cases the clot was surrounded by a cyst, in one of which, death occurring thirteen days after the seizure, the cyst was found nearly empty, disintegration and absorption having thus early taken place (MACINTYRE). In another case, where death occurred twelve weeks after the fit, blood-crystals were found in abundance in the coloured parts of the wall of the cyst, and most abundantly on the yellow parts. In this case, at the end of twelve weeks, the cyst was filled with slightly turbid yellow fluid, a small portion of coagulum only remaining (BRISTOWE). In a case where the extravasation occurred into the *corpus striatum*, it protruded into the lateral ventricle of the right side, and nearly filled it, and the nervous substance around was broken down and soft. Death occurred in half an hour (GIBB).

(3.) *Extravasation into the pons Varolii and other parts.*—Experience shows that the hæmorrhages occur much oftener in certain parts of the brain than in others. Their favourite seats are the corpus striatum, and the nucleus lenticularis, with the neighbouring parts of the hemispheres, and the thalami optici. To such an extent is this the case, that out of 444 cases recorded by Andral, Tacheron, Lerminier, and the Pathological Society of London, the hæmorrhage was situated 202 times at the level of the corpora striata, and into them; 66 times into the corpora striata; 44 times into portions of hemisphere above centrum ovale; 39 times into thalami optici; 16 times into the lateral lobes of the cerebellum; and 10 times into the pons Varolii.

It is now well established that the brain-substance is liable to compression from vascular hyperæmia generally, and increased turgidity of its vessels. There are also parts of the brain so constituted anatomically with regard to vascular supply (see page 53, *ante*), that hæmorrhage more readily occurs in them than in other parts. The anatomical constitution which favours this consists in the increased provision for the transit of blood-vessels. For instance, through the white perforated spot at the commencement of the fissure of Sylvius, the anterior and Sylvian or middle artery of the cerebrum send their numerous branches of various size into the substance of the brain. These, in the first instance, supply the large anterior end and middle portion of the *corpus striatum*, together with the *nucleus lenticularis* and the *capsula externa* and *interna*, which lie immediately over this *anterior perforated spot.* The *corpus striatum*, and the parts at its level, from their anatomical position and relations, are thus the most vascular parts of the whole brain. These arteries are also the direct prolongation of the trunk of the carotid; and so transmit directly the effects of any sudden increase of the heart's action. Hence it is a very ancient belief that the parts supplied by these vessels are those most liable to hæmorrhage, more especially when the vascular system of the brain is overloaded and the vessels morbid (BONETUS, MORGAGNI, ROCHOUX, ANDRAL, CRAIGIE. Opposed to this belief were—HOWSHIP, LERMINIER, SERRES, TACHERON). The result of these observations shows that the comparative liability of parts of the brain to extravasations of blood may be stated in the order already mentioned.

The observations of Paget, in 1850, have also been confirmed—namely, that sudden death from apoplexy is often associated with fatty degeneration of the minute cerebral blood-vessels; while, at the same time, the associated morbid states of other organs are such as to lead to the belief of a more general morbid state, and perhaps more especially of the nervous matter—an apoplectic orgasm which predisposes to the extravasation of blood from diseased blood-vessels, perhaps in the form of miliary aneurisms, a belief now most generally entertained regarding the nature of the apoplectic lesion. Atheromatous degeneration of medium-sized vessels has also been prominent as a lesion. It leads to the formation of aneurisms, met with generally in the blood-vessels at the base of the brain.

The term *capillary apoplexy* was instituted to designate those cases where a number of minute isolated blood points, of the size of a pin's head or smaller, are found in parts which are the seat of softening, or in the neighbourhood of the ordinary large apoplectic foci. They are small extravasations into the perivascular sheaths of the blood-vessels. They are met with in the cortex cerebri, especially as a consequence of thrombosis of the venous sinuses. They play an important part in cerebral hæmorrhages with apoplexy (NOTHNAGEL).

Symptoms.—The literal meaning of the term *apoplexy* conveys the idea of a *sudden stroke;* and it has been usual to confine the term to the results produced by extravasations of blood into the nervous tissue of the brain, a portion of which is thus destroyed. More comprehensive pathological doctrines teach us to give a wider clinical signification to the term. It is now used to characterise a group of symptoms irrespectively of the anatomical conditions upon which they may depend. These symptoms consist of—(1.) Premonitory warnings, extending over variable periods (seconds, weeks, months, or years), marked by sundry derangements of the nervous functions, such as frequently recurring dizziness, headache, ringing in the ears, muscæ volitantes; alteration of disposition (irritability, pettishness, drowsiness, lethargy), loss of memory, dulness of sensation, or temporary diminished power; or loss of speech. Phenomena, temporary and intermittent, suggestive of lesions of irritation, set in in those parts of the body which later on may be attacked with paralysis—*e.g.*, weight, awkwardness, or paresis of an arm or leg, or both. In the same parts abnormal sensations occur, such as "pins and needles," a sense of stiffness, formication, sensations of heat or of cold, or of pain of an indescribable character. Lesions also may be seen in the retina, especially if chronic nephritis exists. Epistaxis is not an uncommon premonitory warning. These symptoms are those usually ascribed to, or associated with, cerebral hyperæmia, more or less locally distributed. (2.) The individual is more or less suddenly deprived of volition and perception in their relation to sensation and motion. Consciousness is thus more or less lost, and paralysis is more or less complete. The patient may fall to the ground completely insensible, or he may only stagger, and cling to some object for support. The respiration and circulation may be unaffected, or the former may be stertorous and the latter laboured. Some group of muscles, a side of the body, or the whole body, is paralysed, flaccid, motionless; or it may be rigid with tonic, or convulsed with clonic spasm. From these states the patient may never recover. Life becomes gradually extinguished; or the sufferer may recover partially or entirely, in the former case leaving some mental, motorial, or sensorial faculty impaired for weeks, or for the whole of after-life.

The essential phenomena of an apoplectic seizure consist in the severance of the brain-functions—namely, volition and perception from motion and sensation: the other symptoms that occur are additional phenomena, depending on secondary changes subsequently induced in the part, or its vicinity, which has been the primary seat of lesion. Whatever may be the pathological doctrines taught regarding the morbid state of the cerebral parts in apoplectic states, we are able practically during life to do little more than merely recognise the apoplectic state itself. The diagnosis between the *congestive*, the *hæmorrhagic*, and the *serous*, or morbid nervous orgasm, can only be arrived at approximatively by a careful comparison of symptoms very closely observed.

I. *Apoplectic symptoms from congestion merely, or determination of blood to the head.*—The face, scalp, and conjunctivæ are increased in vascularity; the skin generally is of a dusky venous hue, and the surface warm. There is fulness of the jugular veins, with increased pulsation in the carotids. The tongue is foul, and nausea prevails, with constipated bowels. Respiration and the pulse are both laboured, and the extremities are cold. The symptoms peculiar to the brain itself are,—(1.) A diminished activity and power of intellect; general confusion of thought, with deficient memory. Any attempt at mental exercise increases the expression of these signs; so does the recumbent position and emotional disturbance. Sleepiness, with laboured respiration, is common, especially after meals; and there is a general tendency to inaction of body as well as of mind—a "not-to-be-disturbed" desire is experienced. Such mental phenomena, however, are not permanent; and while there is a readily induced state of general confusion, there is no persistent, special, or permanent loss of power of intellect. (2.) The senses generally are obtuse. The hearing is dull;

and heavy rumbling noises are constant auditory illusions. The sight is dim, or *amaurosis* is complete, and often black or variously-coloured spots are seen floating in the field of vision; flashes before the eyes, or other spectral illusions, are more or less constant. The patient may see only half of an object, or halves of objects of different colours. Attacks of dizziness occur, with a sense of fulness and oppression in the head; numbness and weight of the limbs; dull and heavy *cephalalgia*. There may be occasional feelings of formication and numbness in certain limbs, momentary loss of memory for some words and figures, or temporary paralysis, confined to certain groups of muscles. These symptoms, however, are only of occasional occurrence, and change their localities. While the absence of pain is not now considered of much diagnostic value, yet the occurrence of severe acute pain is generally indicative of something more than congestion. (3.) Little jerkings of the muscles, and irregular or sluggish movements of the eyeballs, are occasional.

These precursory symptoms having generally been more intense for a few minutes or hours, an attack distinctly apoplectic takes place. These "warnings" which precede cerebral hæmorrhage may, in some cases, depend on *thrombosis* of small vessels, or on small capillary hæmorrhages. Thrombosis and embolism (lesions to be afterwards described) now take a prominent place in cerebral pathology, both as regards cerebral softening and hæmorrhages. By occlusion of a cerebral artery, or of capillaries, clots in blood-vessels are capable of producing apoplectic symptoms, even when the brain-substance is otherwise sound. There is always paralysis of motion in the side opposite to the obstructed vessel, general sensibility, as a rule, being unimpaired; sometimes it may be lessened, but is never altogether absent. Loss of consciousness is also less complete, and the mind is less affected after cerebral embolism than after hæmorrhage. Aphasia sometimes happens. The middle artery of the brain is the most frequent seat of embolism, also the anterior, basilar, and vertebral. Apoplectic symptoms, hemiplegia, and death have also followed plugging of the carotid (HASSE); and of the innominata, right common carotid, and left internal carotid, and middle cerebral (MARKHAM). The seizure commonly occurs during some muscular exertion, such as lifting a heavy weight, pulling on a pair of boots, blowing the nose, straining at defecation, or even upon a simple change of posture, such as stooping, or suddenly assuming the erect attitude.

The special nervous symptoms of an apoplectic attack from congestion merely are—1. That some evidence of the existence of perception may generally be obtained by loud noises, speaking to the person by name, or pinching him. If, however, perception is quite extinct for a few seconds or minutes, it soon again partially returns, and there is confusion of thought, with little volition as to the direction either of thought or movement. 2. Except during the first few moments of the attack, when sensation generally is gone, the changes are slight. Sensation,—indicated at least by reflex action (the limbs being withdrawn if pinched),—is generally present. 3. There is more or less paralysis of all the limbs to a slight degree, and for a short time. It is very rare to have either hemiplegia or paraplegia. Short or involuntary evacuations do not occur unless there have been some convulsions. There is no rigidity of the limbs, but clonic spasms are not unfrequent. Generally, and in the course of a few minutes, the symptoms begin to abate rapidly, and they rarely last even for an hour. With the return of consciousness paralysis disappears, and sensibility rarely remains deficient. The manner of appearance and proportion between the three groups of nervous symptoms is of great diagnostic value; and, as indicative of *congestion*, rather than of *hæmorrhage* or *softening*, any one of the following combinations of symptoms are of importance to be noticed:—1. *The simultaneous development of the three groups of nervous symptoms—mental, sensorial, motorial. There being either*—2. *Distinct loss of perception, profound coma, and general paralysis, without rigidity or convulsion.* 3. *Imperfect loss of perception, with general paralysis.* 4. *General paralysis, incomplete in degree, and sensation unimpaired, or but little affected;* or—5. *Paralysis complete in degree, but without stertor or rigidity* (Dr. RUSSELL REYNOLDS).

II. *Symptoms of Apoplexy from Hæmorrhage,*—A. *Into the cerebral substance of the hemispheres.* Very different statements are made as to whether or not premonitory symptoms are present; and the practical point in diagnosis which such discrepancy of statements has taught is, "*that the non-existence of precursory symptoms in a given case is in favour of the belief that hæmorrhage rather than congestion is the cause of the lesion or softening.* The attack is generally sudden, and rapid in its development. Sometimes after a few hours there may be sudden aggravation of the symptoms, due to a renewed extravasation. The patient, if standing, falls instantaneously, often with a cry, as if knocked down, which constitutes the "stroke of apoplexy," commonly so called. It is the nervous symptoms, however, which are of the most importance. These are—1. Loss of consciousness (of volition and perception), which is commonly complete at the outset. For a few seconds, at least, the patient is utterly deprived of intellectual power, which in slight cases partially returns in a few minutes. In severe cases, however, perception does not return till after some hours, and with vague ideas of things; expressions of thought are confused, amounting to delirium; *and after the first few minutes or half hour has passed, the degree of intellectual obscuration may be taken as an approximate measure of the amount of extravasation,* although it is to be remembered that there are some exceptional and rare cases on record of hæmorrhagic apoplexy in which the mental faculties were very slightly, if at all, impaired. After some days the intellectual powers are often entirely restored; but in many cases confusion of thought and partial loss of memory remain. After recovery of consciousness the power to *think* and to *will* is generally unimpaired, so that if the patient is asked to give the affected hand, the desire to fulfil the request is shown by taking the paralysed hand in the sound one, whose nerves and muscles are under the control of the will, in order to accomplish the act (NIEMEYER). If the case does not terminate fatally, the well-marked character of the recovery is a strong presumption that the symptoms resulted from hæmorrhage, and were not due to softening of the cerebral substance. 2. Sensibility is usually less commonly affected, and less intensely, than mobility. In most cases with hemiplegia there is *anæsthesia* of half the body; but after a time this usually passes off partially or entirely. When cutaneous anæsthesia is complete, although the surface so affected may be limited in extent, the occurrence indicates severity of lesion. In slight cases there is generally only numbness and tingling of the tips of the fingers. Evidence of sensation may be obtained when there is no proof of distinct volition. During the profound coma of the attack at the commencement, the dilated pupil and the half-opened eye indicate that the retina has lost its impressibility; and if hearing and smell are similarly affected, the persistence of such symptoms are signs of evil omen. *Paralysis* of motion is present in the immense majority of cases of apoplexy, its characteristic form being *hemiplegia;* but sometimes it is general, the proportion of cases being as ·84 to ·16 per cent.; or, a bilateral paralysis—*i.e.*, a paraplegia, may occur; and these differences depend entirely on the varying seat of the extravasation. When the paralysis is general, the hæmorrhage is rarely limited to the substance of the hemispheres. Paralysis of half the body, characterised by its limitation to the muscles of the extremities, to the muscles of the face, mouth, and nose, and to the muscles that protrude the tongue on the side of the lesion, is due to destruction of the *corpus striatum*, lenticular nucleus, and *optic thalamus* of *one hemisphere.* Hence, the distribution and characteristics of the paralysis vary in different cases. With few exceptions, the paralysis is unilateral, the type of a cerebral hemiplegia—*i.e.*, it affects the side of the body opposite to that in which the brain lesion exists—*i.e.*, the arm and the leg, and the muscles of the face, on the *side opposite* to that lesion, and not unfrequently those of the trunk (as may be seen by observation of the scaleni), are paralytic or paretic (see p. 333, *ante*). With regard to the face, those branches of the facial nerve which supply the *frontalis, corrugator supercilii, and orbicularis palpebrarum* muscles almost always escape intact (Todd). Hence, such patients can wrinkle the forehead, and open and close the eyelids. But the mouth droops on the affected side, the nostril rests undilated, the naso-labial furrow becomes obliterated, and there is inability to whistle or to purse the lips. The tongue deviates when thrust out, with its point *towards* the paralysed side. Occasionally the cheek of the affected side flaps on expiration, and the uvula may hang obliquely, pointing sometimes to one side and sometimes to the other. In sudden attacks during the first few days, there may be deviation of both eyes towards the non-paralysed side, with rotation of the head in the same direction (Prevost)—a phenomenon which is not to be confounded with unilateral strabismus. On the sound side the angle of the mouth is drawn up and the nostril dilated. During profound stupor the deviation of the face alone indicates paralysis on one side. In less severe cases the condition of the limbs as to volition is the guide. Any extreme movement of the face, such as crying or laughing, renders the inequality of action more apparent. Amongst other nerves to be affected, the *hypoglossus* is most frequently implicated; next come impairment of the pupil, and of the muscles of the eyeball. The power of swallowing is rarely impaired, provided the fluid or morsel is brought within the influence of the pharyngeal muscles.

The loss of motion is commonly absolute at first, especially in the arm, which is generally more profoundly affected than the leg, the one being more completely paralysed than the other. Stertor, with involuntary defecation and micturition, are common. *Involuntary contractions* of a tonic or clonic kind are extremely rare from hæmorrhage limited to the cerebral substance. *Reflex* movements continue, and the respiratory acts are duly performed.

The more common combinations of symptoms by which the existence of cerebral hæmorrhage limited to the medullary substance might be inferred are—(1.) *Profound coma, with hemiplegia on one side, of marked intensity, and without rigidity.* (2.) *Paralysis of both sides, but one more profoundly affected than the other, is a rare occurrence in limited hæmorrhage. It is due to hæmorrhages occurring simultaneously in both hemispheres, especially in the central ganglia, or to extensive lesions in the pons or medulla oblongata.* (3.) *Slight coma, but paralysis hemiplegic and complete.* A large apoplectic clot, which destroys the *corpus striatum* or *thalamus*, leaves a *hemiplegia* that never disappears; but small clots in those parts, by which nerve filaments and unbroken and ganglionic nerve-cells are unimpaired, and only temporarily pressed apart, leave a paralysis which is generally only temporary. Extensive apoplectic clots at other parts of the cerebrum not unfrequently leave paralysis, which sooner or later disappears again, the capillaries of the motor centres being relieved of pressure by the partial re-absorption of the extravasation, and so become again permeable to blood; or that the collateral œdema in the vicinity of the broken-down part of the brain, which extended to the motor centres, has disappeared, with cicatrisation and contraction of the apoplectic clot (NIEMEYER).

Cases of *alternate paralysis* (see p. 334, *ante*), as when the paralysis of the face is on the opposite side to that of the paralysed limbs—*i.e.*, on the same side as the intracranial lesion (which is sometimes also called "crossed" paralysis, GUBLER), occur when the lesion implicates the lower part of the *pons* near the medulla oblongata. A second form is where the lesion implicates the *crus cerebri*, when the nerves distributed to the *motor oculi on the same side* of the lesion are injured in their course. Cases of paralysis from cerebral hæmorrhage may also be confined to the area of distribution of individual cranial nerves. In those cases where the extremities and muscles of the face are affected simultaneously on the side opposite the lesion, that lesion is to be sought for in the upper part of the *pons* near the *crus cerebri* (GUBLER, BROWN-SÉQUARD). The possibility of further localising hæmorrhagic lesion requires great care in drawing conclusions from the character of the symptoms. The first principle to be observed in attempting to indicate the localisation of cerebral disease is, "*that the lesion from the occurrence of which diagnostic conclusions are to be drawn, must be one which is definite and limited in extent, and one which must not affect important neighbouring structures, either by pressure or otherwise. The lesion also must have ceased to be progressive. It must have come to a stand-still* (ALTHAUS, NOTHNAGEL). With regard to the *cortex cerebri*, the observations of Hitzig, Fritsch, Nothnagel, and Ferrier show that destructive lesions at certain definite parts may give rise to paralysis; and it appears proven that motor centres, through which the will excites the nerves of the extremities, lie in the *gyrus præcentralis*, or *gyrus centralis anterior* (of HUSCHE and ECKER; p. 19, *ante*). Other affections of the cortical substance of this convolution may produce motor paralysis—an observation which is in harmony with certain clinical facts recorded by Hitzig and

Bernhardt; but the latter observer would include also all the parts bordering on the fissure of Rolando. Hæmorrhages into the third frontal convolution, on the left side generally (and exceptionally on the right side), even extremely small, cause *aphasia*—a condition to be described subsequently. Extensive hæmorrhages into the cortex usually occur on the convexity, and at several different points at the same time; and although they do not generally prove fatal, they cause greater impairment of the mental functions than any lesions in any other parts of the brain. Destruction also of certain portions of the brain in the *medulla* of the *cerebrum* does not cause any perceptible disturbance of function; small hæmorrhages there may escape recognition during life. Cysts are often found after death in the medullary substance of the hemispheres in cases which—(1.) never showed signs of cerebral disease, (2.) had a history of apoplexy from which there was complete recovery; (3.) after clinical evidences of hæmorrhage there followed paralysis of parts on the side opposite the side of the lesion, similar to what follows the usual lesion in *corpus striatum* and its vicinity; (4.) cases which show signs of hemiplegia which afterwards disappear—a most characteristic sign of hæmorrhage into medullary substance of the hemisphere (NOTHNAGEL).

B. *Hæmorrhage into the ventricles* cannot in some cases be distinguished from arachnoid extravasation, or in others from effusion into the cerebral substance only, especially when in the vicinity of the ventricles. The cases, however, which are less doubtful are marked by the following characteristics, in addition to apoplexy:—(1.) Coma is very profoundly marked at the commencement, and continues of equal intensity; or the patient, after partially recovering from a slight seizure, is again suddenly plunged into profound coma, from which there is no recovery. This second attack is presumed to indicate hæmorrhage either into the ventricles or the arachnoid cavity, from its original site of extravasation in the medullary substance of the brain near the ventricles or near the surface. Stertorous breathing is then strongly marked. (2.) *Paralysis* is complete in degree, and is developed simultaneously on both sides; or after having been hemiplegic for a short time, it becomes general. Involuntary evacuations follow. The pupils remain dilated. Deglutition is dangerous and difficult. When paralysis is general and the coma profound, it is almost a sure sign that hæmorrhage has taken place to a considerable extent into the ventricles. *Rigidity* or *tonic contraction* of the muscles is present in many cases of hæmorrhage; in nineteen out of twenty-six cases it occurs in the paralysed limbs; and in about four out of twenty-six cases it may be seen in those of the healthy side (Dr. RUSSELL REYNOLDS). Its presence is a sign of extensive hæmorrhage, with laceration of the brain. The most frequent combination of symptoms indicative of hæmorrhage into the ventricles may be shortly stated to be *profound coma, with general paralysis and rigidity.*

C. *Arachnoid or subdural hæmorrhage* occurs when the extravasation bursts through the *pia mater* and arachnoid into the space between the arachnoid and *dura mater*, and such cannot be distinguished from *ventricular extravasation.* If, however, the extravasation remains immediately subarachnoid at first, and of limited extent, it may be approximately diagnosed,—*First,* by the nature of the premonitory symptoms having partaken of meningeal inflammation, such as by severe pain in the head, with impaired intelligence and power of movement. *Second*, the attack is less sudden than in cases of congestion or of central hæmorrhage, and the symptoms are progressively developed. The following are the combinations of symptoms which indicate the occurrence of subarachnoid hæmorrhage:—(1.) *Complete and profound coma without paralysis, or with general paralysis very slightly developed in intensity.* (2.) *Complete loss of consciousness without paralysis, but combined with rigidity or clonic contractions of the limbs.* (3.) *Paralysis of hemiplegic distribution as regards the limbs, but without deviation of the features, the muscles of the face not being implicated.* (4.) *An apoplectic attack without anæsthesia.* (5.) *Imperfectly developed coma with general paralysis.* (6.) *An apoplectic attack, of which the symptoms are somewhat interchangeable or remittent.*

2. *Spinal Hæmorrhage.*—The *symptoms* are, effusions between the meninges, giving rise to severe irritation, pains in the back, and spasms, especially in the parts supplied by the nerves going off below the seat of injury, and ending in paralysis. Perfect anæsthesia accompanies large extravasations and complete paralysis; so that it depends upon the position of the hæmorrhage—high up or low down—whether the paralysis will be sooner or later fatal. If the symptoms of interrupted nerve-conduction from the brain are sudden—anæsthesia and loss of voluntary motion in the lower half of the body, combined with paralysis of the bladder—then it is most probable that the cause is rather hæmorrhage than inflammatory softening; and in other respects, what has been written regarding *spinal meningitis* and *myelitis* can only be repeated here.

Diagnosis.—The great difficulty in the diagnosis of cerebral hæmorrhage with its clinical symptom—apoplexy, is to distinguish it from acute softening. In most cases it is not possible to make the distinction. On the other hand, there are numerous cases of acute softening which can scarcely be mistaken for cerebral hæmorrhage. The further diagnosis will be considered under the next topic.

Prognosis.—In general terms cerebral hæmorrhage is of slight significance up to thirty years of age; its fatality increases *pari passu* with years; and, the greater the age, the less is the probability of recovery (ALTHAUS.) Apoplexy is always a grave symptom, and the more grave in proportion, generally, as the respiration is stertorous and the deglutition difficult. Each succeeding attack of hæmorrhage is more dangerous than the former. The practitioner should also be guarded in his prognosis till after the first week or ten days. It is rare that a patient expires without having egained his consciousness. It is very rare, indeed, for death to supervene mmediately; in the large majority of fatal cases from six to twelve hours lapse between the beginning of the illness and its end. The sopor may st from two hours to three days before ending in death. Death then kes place usually as a result of pressure on the vagus nerve, and its aproach is indicated by faltering respiration and by intermission and weakness the pulse, or as a consequence of œdema of the lung or pneumonia. If mplete coma lasts over 48 hours recovery rarely takes place. In favourle cases consciousness remains absent for from half an hour to three urs, when it begins to return. The first sign of improvement is a return reflex excitability. Consciousness returns slowly, while headache and general discomfort take the place of unconsciousness. It may be days before the symptoms due to the localised lesion become fairly developed, the patient meanwhile continuing drowsy, and his thoughts confused and movements feeble. The disturbances which remain are now due to the permanent destruction of brain-substance. If a patient does not die in the fit of apoplexy, a more or less severe *encephalitis* will set in after a few days, as a result of the injury done to the texture of the brain. When the quantity of blood extravasated is small, the disease is seldom fatal from the first attack. When the extravasation is extensive, although superficial, the result is rapidly fatal by coma, resulting from compression over a large surface. When effusion is sudden and extensive in *certain parts* of the brain, such as in the *corpora striata,* or *optic thalami,* or the *pons Varolii,* the result is rapid death; and, generally, the more nearly the extravasations approach the *medulla oblongata,* the more rapidly fatal is the result.

The temperature of the body is lower (96·5° Fahr.) in the severe forms of hæmorrhage into the pons or medulla, and so continues till death. It begins to fall in from ten to fifteen minutes after seizure, and if it rapidly falls from 95° to 97° Fahr., the prognosis is bad; but if the fall is only slight—say 1° to 1·5° Fahr., and protracted in its production, the prognosis is generally favourable (ALTHAUS). If life continues from ten to twenty hours, the temperature sustains a marked and rapid rise. If life is maintained for several days, the temperature vacillates between 99·8° and 100·4° Fahr.; and finally, just before death a period of great elevation occurs (103°, 104°, or 105° Fahr.) more or less rapidly. Where the rise is rapid and extensive, it has the same unfavourable meaning as the fall in the commencement. The rapid rise is a sure sign of collapse (ALTHAUS, NOTHNAGEL). Increased frequency of pulse, with increase of temperature, headache, sparks before the eyes, delirium, twitchings, pains and contractions of the paralysed parts, indicate "symptoms of reaction," which may be moderate and finally disappear, leaving the patient more or less permanently paralytic. These phenomena begin between the second and fourth day after the attack, with exacerbations at intervals of two, four, or eight days; after which a chronic stage begins, when for the first time it is possible to recognise how much mischief has been done (HASSE). In a very few instances the patient may be restored in a few days, or in a few weeks, or in a few months; but commonly, certain symptoms are permanent—*e.g.*, paralysis of motion. In general, however, some slight improvement takes place, even in the worst cases, so that the patient recovers some use, first of his leg, and then, perhaps, of his arm, so that he is able to walk with a straight leg and a dragging foot. The use of his arm returns more slowly and more imperfectly. This recovery is often preceded and accompanied by very severe pains, especially of the upper extremity, marking the still irritate state of the brain surrounding the destructive lesion. After this n further improvement takes place. The limb uniformly wastes, and th parts remain in a paralysed state for the rest of life—ten, twenty, or mor years. The vital powers of the paralysed parts are so impaired that, inflamed, the inflammation seldom terminates by resolution, but has great tendency to gangrene, while cicatrisation is slow and difficult. I on the other hand, after a severe expression of hemiplegia, the patient begins to recover the use of his arm more rapidly and completely than he does the use of his leg, it is an unfavourable sign, one of early mental decay (TROUSSEAU, TODD, BAZINE). Also, when the fingers of the paralysed hand continue flexed into the palm, through contraction of the flexors after cerebral hæmorrhage, the use of the hand will remain permanently lost, and no hope of improvement can be held out (TROUSSEAU). Where cerebral hæmorrhage occurs associated with granular kidney, gout or syphilis, the prognosis is unfavourable. Paralysis of motion sometimes persists in groups of muscles, such as those of the tongue (to the affection of speech), the forearm, or hand. Sensibility is generally first restored, then the motion of the lower extremity, then the arm, and lastly, that of the forearm and hand.

In paralysis due to cerebral hæmorrhage, and in intra-cerebral paralysis generally, the reaction of the muscles under both the galvanic and the faradic current remains unchanged. Although the paralysis may have existed for years, contractions of the paralysed muscles follow the application of either kind of electricity, just as in health. Changes of sensibility are less common than those of motor functions. Sensibility of the hand and foot remains longer impaired than does that of the arm and thigh.

Trophic changes may take place on the paralysed side, such that acute *bedsores* occur, generally in the middle of one of the nates, less often on the heel. They begin with an erythema of the part, two to four days after the apoplectic attack. On the next day *bullæ* appear; and sloughing of the skin follows quickly. These are highly unfavourable symptoms, and are of the same nature as ulceration of the cornea after section of the fifth nerve (CHARCOT). Rapid progress and a superacute course are characteristic of the progress of this affection (ALTHAUS). In all cases impairment of general nutrition is the result of injury to the brain from hæmorrhage into its substance; and when hæmorrhagic foci are of long standing, certain secondary changes of the nature of degeneration occur in certain tracts of conducting fibres, which have been severed at some point in their course by the extravasated blood (TÜRCK, BOUCHARD, CHARCOT, VULPIAN).

No doubt the principal adverse circumstances attending recovery from apoplexy are, that although the patient appears to be doing well the first few days after the attack, yet towards the close of the first week the brain, irritated by the presence of the clot, inflames and softens, and thus may induce another and a fatal attack of apoplexy. Should the patient, however, survive this dangerous period, he may continue to live many months, or years, according to his age; but he is generally at length cut off by a fresh attack of apoplexy, or his brain inflames and softens, and he dies in a typhoid state. Although it is rare that the patient, on recovering from cerebral hæmorrhage, has the good fortune to recover all the faculties of his mind, his memory is not unfrequently impaired, often to such a degree that he has forgotten all dates, the names of his friends, or even the names of things—the remembrance of all substantive nouns—the recollection of all adjectives. In some instances the power of association is also so destroyed, that although many remember both names and things, they are unable to connect the thing with the proper word, so that they call that which is cold hot, or speak of night when they mean day, or call a coffee-pot a wash-hand basin (see *Aphasia*). Others, again, have forgotten how to read, and the power thus lost either returns suddenly, or they are obliged to learn *de novo* (Dr. SHARPEY, in "*Brain*," April, 1879). The memory and attention generally are very greatly impaired, and the patient is no longer able to transact business; or if he begins a sentence, he is unable to finish it, or he repeats the same idea over and over again. The power of application is less enduring; and the sense of touch and co-ordination of movements are less quick. The emotions and passions, also, are little under control. Some weep like children, others laugh immoderately, and all are easily terrified, or otherwise easily influenced through the emotions. A state of profound dementia may finally supervene, with secondary cerebral atrophy.

All these circumstances must be taken into account in giving a prognosis regarding an apoplectic patient.

Treatment.—It may "incline the balance to recovery or death." Bleeding from the veins of the arm, the jugular vein, or the temporal artery, has been the most conspicuous method of treatment from the earliest times. It is now employed only under the following circumstances:—(1.) When there is an obvious increase of intracranial pressure, it is practised so as to bring about indirectly a diminution of arterial tension. With such intracranial pressure there is cerebral hyperæmia; and when a paralysis of the respiratory or vagus centre is threatened, then it is that *venesection* by diminishing this pressure may have the effect of prolonging life. Patients have thus been roused from a state of coma. Clinically, the indications for *venesection* under such conditions in cerebral hæmorrhage are:—A turgid face with distended veins, and increased pulsation of the carotids; a powerfully acting heart; the radial artery of normal tension; the pulse of normal frequency, or slow and regular; when the respiration is uniform, but of snorting character; when the patient is strong and not too old. Slow and deep respiratory movements, with stertor (indicating paralysis of the respiratory centre), and combined with rapid pulse (indicating paralysis of the vagus), add greatly to the necessity of immediate venesection. The beneficial action of the remedy is shown by the pulse becoming softer, more subdued, and more regular. Venesection is out of place in all cases which do not correspond to this description (NOTHNAGEL). Large bleedings are to be avoided. The blood ought to be permitted to flow from a large opening, in order more rapidly to relieve the congestion, to check, if possible, further effusion of blood, and to divert its active flow from the head. The head and shoulders should be raised while the blood is flowing.

On the other hand, in some cases, bleeding during an apoplectic fit hastens a fatal result—collapse occurring during or immediately after venesection, from which the patient never recovers. It is to be avoided in the old and decrepit, where marked arterio-sclerosis is present; also if the pulse be small and slow, feeble, or almost imperceptible, the skin cold and clammy, with a tendency to death by syncope; if the heart's action be feeble or weak, and the pulse irregular; if the patient has been of intemperate habits, or is suffering from organic disease of the heart and arteries;

or if there is a gouty or rheumatic history, no advantage is to be gained by the abstraction of blood at this time and in this way. Blood-letting is therefore absolutely contra-indicated under the following circumstances:—(*a.*) anæmia, (*b.*) aortic valvular disease, (*c.*) in cases commencing with syncope. In such cases the use of stimulants must be had recourse to in order to prevent paralysis of the heart. The clinical indications for stimulants are:—A pale collapsed expression of face, absence of venous turgidity, slight arterial tension, heart's impulse weak, respiration hesitating and intermittent. It may, in some exceptional cases, be necessary to follow or combine venesection with stimulants. The stimulant agents may be:—(1.) Such as will rouse the depressed activity of the respiratory centre,—*e.g.*, sudden sprinkling of the skin with cold water; the application of ammonia to the nostrils; (2.) substances which will stimulate directly the heart's action—*e.g.*, musk, strong coffee, wine, and preparations of ammonia. If the patient cannot swallow, enemata of musk may be used (NOTHNAGEL). Many still believe with Trousseau, that in *all* cases, grave or slight, patients do better without either blood-letting or purgatives. He considers the part played by congestion of the brain to have been much exaggerated. Dr. Althaus, also, is of opinion (after an experience of more than 400 cases of hemiplegia from cerebral hæmorrhage) that the former universal treatment by venesection was thoroughly irrational: and he considers that the condition of the brain during cerebral hæmorrhage is not one of congestion (as was formerly believed), but is one of anæmia; that the organ not only loses blood largely, but is also, through compression from the clot, unable to receive a fresh supply; that death takes place chiefly from anæmia; and that by venesection we hasten the fatal result. Instead of doing nothing, however, he recommends *ergotine* to be at once injected hypodermically (a grain of Bonjean's ergotine every hour, or even every half hour), into the subcutaneous areolar tissue. Additional chances of recovery may be given by applying to the head cold cloths, or crushed ice in a bladder, and mustard cataplasms to the feet; also, by placing a drop or two of croton-oil on the tongue, and by throwing up a cathartic enema of castor-oil and turpentine.

After the patient has in some degree revived, some time should be allowed for the absorption of the blood effused before deciding upon the future treatment. A few hours having elapsed, the conduct of the practitioner should be guided by the occurrence or not in the patient of *pain of the head*, which may be taken as a measure of the fulness of the brain, and its tendency to inflammation; but the less that is done during the first three or four days the better. If pain in the head continue, ten to twelve leeches should be applied from time to time, till that symptom s entirely relieved; or, supposing the pulse to be full and strong, and the patient free from headache, yet, under these circumstances, leeches should be applied to the head, to subdue that reaction which so generally takes place from the fourth to the seventh day. The further treatment of the ase is by moderately purging the patient, both as a means of relieving he head and of improving the secretions of the alimentary canal, which re often black and fetid. Active and searching purgatives generally do ood at this stage. Five grains of *calomel*, with a drachm of *compound jalap owder*, given as soon as the patient can swallow, and followed up by *black* *draught*, or by an ounce of *sulphate of magnesia* with *camphor mixture* every four or six hours, and continued, according to its effects, for a greater or less length of time, are the best means we have for promoting recovery, and for preventing a relapse. In cases of hypertrophy of the heart, without valvular disease, eight to ten minims of *digitalis* may be added to each dose of the purgative medicine. If the power of swallowing is in abeyance, then three or four drops of *croton oil*, with five grains of *calomel*, rubbed up with fresh butter, should be put on the back part of the tongue, and stimulating enemata thrown up the rectum. The following are recommended by the late Dr. Tanner :—

1. *Enema of Turpentine and Castor-Oil.*—℞. Olei Ricini, Olei Terebinthine, ā ā ℥iss.; Tincturæ Asafœtidæ, ℥ii.; Decocti Avenæ, ℥xii.; *misce*, fiat enema. To be thrown up the rectum by means of a long stomach-pump tube.
2. *Croton Oil Enema.*—℞. Olei Ricini, Olei Terebinthinæ, ā ā ℥i.; Olei Crotonis, ♏ vi.; Decocti Avenæ, ℥iv.; *misce*, fiat enema.

Although turpentine is objected to by some on account of the intoxicating effects which it is sometimes apt to produce, it is nevertheless an efficient remedy where torpor and insensibility exist. It is of great importance to empty the rectum and lower bowel.

If the intestines are distended by gases, an enema of *castor-oil* and *rue* may be given :—

3. *Castor-Oil and Rue Enema.*—℞. Confectionis Rutæ, ʒi.; Olei Ricini, ℥i.; Tincturæ Asafœtidæ, ʒii.; Decocti Avenæ, ℥vii.; *misce* (*Practice of Medicine*, 4th edition, p. 653).

Apprehension of a relapse being at an end, the patient is in general most willing to believe that the palsy which may remain is a mere local disease, and to submit to any treatment for its removal; but every attempt to act locally on the muscular system is prejudicial so long as any central irritation exists. Such remedies are neither theoretically nor practically useful. (See "Paralysis.") Active or passive exercise of the muscles are remedies highly beneficial. The *induced current of electricity* is also beneficial. It seems to improve the nutrition of the paralysed muscles, which tend to atrophy from long disuse, and paralysis tends also to get worse from diminished excitability of the nerves. Local faradisation by induced currents of electricity gives artificial exercise to these muscles, and thereby improves their functional and nutritive properties.

Dietetic and Prophylactic Treatment.—The diet of the patient should be limited to milk, boiled vegetables, light puddings, and fish. At no subsequent period ought he to indulge in a full animal diet, or to drink undiluted wines. At the same time, too lowering a regimen is to be avoided, as thereby the irritability of the system and the heart's action generally is increased. All the causes of the disease already fully referred to should be avoided, counteracted, or overcome. The diet and the bowels should be carefully regulated, and the patient placed under the best possible hygienic influences. A sojourn at the moderately warm waters of

Wildbad, Pfäffers, Ragatz, and Laudeck (Teplitz with great caution), and Rehme and Nauheim, are recommended by Niemeyer and Nothnagel, with careful regulation of diet and exercise.

SOFTENING OF BRAIN (WHITE AND YELLOW); ATROPHIC SOFTENING; OBSTRUCTION OF CEREBRAL ARTERIES; THROMBOSIS; EMBOLISM.

LATIN EQ., *Cerebrum Fluidum Albens* vel *Flavens*—Idem valet, *Mollities Atrophica;* FRENCH EQ., *Ramollissement Blanc* vel *Jaune;* GERMAN EQ., *Weisse Gehirnerweichung;* ITALIAN EQ., *Rammollimento Bianco* vel *Grallo*—Syn., *Atrofico.*

Definition.—*Softening may be the result of imperfect nutrition, or death of brain tissue, owing to deficient supply of blood, in most instances dependent on mechanical obstruction, embolism, thrombosis, or degeneration of some of the cerebral arteries, oftenest the middle cerebral in cases of embolism, and in a large majority of cases on the left side.*

Pathology.—(*a.*) *History.*—Whatever may be the circumstances under which softening is developed during life, or the form it assumes after death, the lesion is not so much a substantive disease as the effects of a morbid process taking place under several different conditions of the circulation within the brain. Much confusion has existed regarding the nature of the several softenings of the brain and spinal cord; and it has always been difficult practically to distinguish one kind of softening from another. As seen *post-mortem* it has been considered to occur under the following conditions:—(1.) *Exudation, or inflammatory* (*red*) *softening,* from exudation which has been infiltrated among the elementary nerve structures and neuroglia. (2.) *Hæmorrhagic softening,* from mechanical breaking up of the nerve texture by hæmorrhagic extravasations, either in large masses or by infiltration in small isolated points. (3.) *True fatty softening,* from fatty degeneration of the nerve-cells, independent of exudations, and as a result of deprivation of nutrient material (PAGET). (4.) *Serous or dropsical softening,* from the inhibition of serum, which loosens the connection between the nerve-tubes and cells. (5.) The consequence of the blood-stroke (*coup de sang*) (Dr. CRAIGIE). (6.) As a consequence of the process which accompanies or follows hydrocephalus. (7.) It may take place in the cerebral substance surrounding tumors.

Clinically, the term has been constantly used in rather a vague sense, generally denoting a condition of mere mental imbecility, or weakness of mind. Practically, as a lesion of the brain, it signifies a softening of its texture (of a white colour, as a true primary softening), quite different from the red softening of encephalitis, already described (page 442, *ante*), and different from mere embolic congestion. Another kind of softening of the brain—namely, yellow softening—is either, (1.) this true white softening tinted yellow or ochreous, from changed blood; or, (2.) it is a different and always a secondary condition, in the form of a yellow gelatinous-like œdema around tumors, tubercles, or syphilitic gummata of the brain. Thus it is always limited to the immediate neighbourhood of the growths to which it stands in the relation of a peripheral œdema (WILKS and MOXON). The true white softening has also been distinguished from that white sodden softening of the central cerebral tissue around the ventricles in acute hydrocephalus, which is little else than a mere œdema, devoid of the special characters of true white softening.

(*b.*) *Causation.*—The subjects of white softening are usually of advanced age, suffering from renal or cardiac disease, and in whom the arterial coats of the vessels within the brain are diseased, with the local formation of clots (*thrombosis*). When the clot originates in the blood-vessel itself, it is usually in the vertebrals, the basilar, the internal carotids, and their primary branches, or the artery of the corpus callosum. In connection with old age thrombosis is connected with the arterial degeneration of age. In early adult life and middle age, it is often a consequence of syphilis (see p. 923, Vol. I.). Or, some of these minute vessels may be obliterated by arrest of small blood-clots within them, carried from a distance, as from diseased cardiac valves (*embolism*), as a consequence of rheumatic inflammation of the valves of the heart. The primary obstruction may happen in the arteries at the base of the brain in old people, or in those prematurely old from laborious or debauched lives. Embolism usually occurs in one of the arteries of the circle of Willis, or in one of the trunk vessels, before the circle is commenced to be formed.

(*c.*) *Morbid Anatomy.*—The softening is limited to the district which the obstructed vessel supplies; and the softening which results is generally from obliteration of one or other of the middle cerebral arteries, consequent on coagulation of the blood in them, or on obstruction from clots of fibrine (CARSWELL, HASSE, KIRKES, SANKEY). In a large majority of cases the obstruction from embolism is in the middle cerebral artery of the left side, on account of the anatomical peculiarity of the *left* as compared with the *right carotid artery.* The softening is then to be recognised by the peculiar loss of cohesion of the brain-substance, which may be described as "pastiness" of the brain matter; and the fall of a gentle stream of water upon the softened part will indicate the change.

Microscopically the brain tissues are broken up, and with them is mixed a number of bodies, known as "compound granule masses" or cells —the originally described inflammatory globules of Gluge. They are large bodies appearing black under the microscope from their opacity—somewhat resembling mulberries, from their being composed of a congeries of round granules. Their principal composition is fat. Similar bodies are to be found in all degenerating parts—caseous strumous glands; cancer tumors, and phthisical lungs. They are the constant attendants of degenerated and softened textures. In this lesion (white softening) the microscope also shows decaying tubes and escaped myelin, a few amyloid corpuscles from the neuroglia, and the fatty and calcareous remains of blood-vessels, with accumulations of numerous fatty granules in their walls and perivascular sheaths.

The difference of white from red softening seems mainly to consist in the difference in degree and rate of the obstruction of the vessels. Hence the softening is "white" when the obstruction is sufficiently slow as to starve the tissue, without inducing absolute stasis of the blood and subsequent hyperæmia. Slow syphilitic inflammatory stoppage of the arteries has led to extensive "white" softening of the pons and posterior lobes of the cerebrum. *Red* embolic softening (on the other hand) occurs when the vascular tension is high and the obstruction sudden and complete; *White* softening when vascular tension is low, and the obstruction gradual and less complete (WILKS and MOXON).

Symptoms.—The clinical history of softening seems closely to resemble that of cerebral hæmorrhage, and it is very often impossible to tell whether the physician has to deal with a case of *apoplexy* from cerebral hæmorrhage, as commonly understood, or a case of *cerebral softening.* The premonitory symptoms peculiar to softening appear to be absent in a half, or more than a half, of the cases (ROSTAN, DURAND FARDEL). In some instances, however, the premonitory symptoms afford strong probability of softening, and are of much value: the absence of them, however, cannot be regarded as equivalent to the absence of softening (REYNOLDS). The attack itself may be gradual or sudden; it is much more sudden from embolism than from thrombosis. When the detached embolus settles, there is a sudden shock to the brain with anæmia, and for a short time there is headache, vertigo, and apoplexy, as in corpus striatum hæmorrhage. These symptoms pass away, so that in many cases there is only hemiplegia without loss of consciousness. In other cases (as of thrombosis), after the progressive development, during some hours or days of such premonitory symptoms as have been mentioned in the previous affections of the nervous system, the patient gradually becomes apoplectic, or he may at once appear to become so suddenly and instantaneously, without premonitory symptoms. Transient excitement or mild delirium may precede abolition of perception. Coma is frequently developed abruptly. The patient lies still, as if in a profound sleep; but immediately gives the hand or puts out the tongue, if told to do so, intelligence remaining intact. Perception and volition once lost, however, is not recovered from. Dulness and obscuration of thought and perception prevail often to a marked degree. Hyperæsthesia has erroneously been considered pathognomonic of ramollissement, perhaps because it is more common in softening than in any other apoplectic disease. Numbness and a sensation of cold are not unfrequent. Paralysis and spasmodic contractions of muscles occur. The face-muscles act unequally, producing deviation of the features, sometimes very slight, at other times highly marked. Speech is almost constantly impaired, and after slight recovery it remains so. Paralysis is commonly limited to one side, sometimes to one limb, but in rare cases it is general. The spasmodic contractions are either of a tonic or of a clonic kind, rigidity or occasional spasm being found in either the paralysed or non-paralysed limbs,—most commonly in the former. The physician, however, will not derive much information from the mere recognition of the presence of single symptoms; it is by a close observance of their combinations that exact diagnosis will be insured.

The following combination of symptoms are those which may with most probability be referred to softening:—(1.) Imperfect coma, partial loss of perception and volition, with rigidity of the limbs; (2.) Perfect coma without rigidity; (3.) Paralysis without loss of consciousness; (4.) Paralysis with hyperæsthesia; (5.) Rigidity, coming on after the return of perception and volition; (6.) Marked aphasia may be present; or total inability to utter articulate sounds if the softening occupy the left side of the brain.

In thirteen cases of ramollissement of the *cerebellum*, the impairment of intellect was trifling, while motion was greatly affected. In the greater number of cases palsy existed with or without contraction of the muscles of the opposite side of the body; in others, convulsive actions of both sides of the body; and in one case observed by Rostan, the palsy was on the same side. In no instance is it said that any sexual desire troubled the patient.

Diagnosis.—Whenever *hemiplegia*, complete and absolute, occurs *suddenly*, *without* loss of consciousness, softening of the brain may be diagnosed; and also that the softening is of the *colourless kind*, from arterial plugging (RÉCAMIER, TROUSSEAU, TODD). Whenever, on the contrary, complete loss of motor power is attended by loss of consciousness, especially when coma is sudden, *hæmorrhage* may be diagnosed, and that to a considerable amount. When the intellect is effaced to some extent, but not entirely,—when there is obtuseness, but not complete loss of sensibility,—whilst there is absolute loss of motor power, *capillary hæmorrhage* co-exists with softening (RÉCAMIER, TROUSSEAU). If there be heart disease present, or a history of rheumatism or of former cardiac mischief, the case is likely to be one of embolism. A history of syphilis would suggest arterial degeneration and thrombosis, with consequent softening. Hæmorrhage would be almost certain in cases of renal disease with albuminuria and retinal hæmorrhage. The age of the patient must also be considered. Embolism may occur at any age: cerebral hæmorrhage occurs in advanced life.

Prognosis.—The after-symptoms of softening are also strikingly different from those of apoplexy. The morbid phenomena do not suddenly disappear, nor is there the gradual improvement which takes place after apoplexy. Enfeeblement of the mental powers most commonly persists, and the motorial phenomena remain. Slight apoplectic-like seizures occur, convulsive movements and rigidity increase, and some little febrile excitement becomes developed, which in severe cases generally assumes a typhoid type, with brown tongue and rapid pulse. From such a condition recovery is rare, and death may occur from coma, bedsores, or pneumonia. But the progress of cases depends upon the extent of the softening and its site. If the patch be small, recovery is in proportion more complete; and may take place up to a certain point. Softening from thrombosis or embolism is rarely recovered from completely. Aphasia persists; hemiplegia persists, and contractions may occur from secondary lateral sclerosis of the cord. The intellect becomes more and more impaired, the patient becoming bedridden and childish. Death may occur also from apoplexy, by recurrences of cerebral hæmorrhage, or from inflammation of brain round the lesion, or simply from asthenia.

Treatment.—The patient must be kept quiet and clean, the bowels being attended to daily. Food should be nutritious, easy of digestion, and not bulky, nor too abundant. Stimulants are to be withheld (see under "Cerebral Hæmorrhage" and "Hemiplegia").

TUMORS OF THE BRAIN AND ITS MEMBRANES; MORBID GROWTHS; ANEURISMS; ENTOZOA.

Definition.—*New growths, parasites, and aneurisms, implicating the cerebral substance, the spinal cord or their membranes, or both.*

Pathology.—(*a.*) *History.*—Different forms of tumor occurring in the brain have been described under the following names:—(*a.*) *Simple cerebral tumor* (scleroma), or indurated portions of the brain; (*b.*) *Gliomata;* (*c.*) *Adenoid, sarcomata,* or fleshy tumors; (*d.*) *Strumous tumor,* tubercular deposits (*tyroma*); (*e.*) *Gelatiniform* tumors (*myxomata, colloma*); (*f.*) *Adipose* and lardaceous tumors (*lipoma*); (*g.*) *Cholesteatoma,* or pearl-like tumors; (*h.*) *Encysted tumors:* (1.) *Cysticerci and echinococci;* (2.) *Blood-cyst* (*hæmatoma*); (*i.*) *Fungoid tumor;* (*k.*) *Melanotic cyst;* (*l.*) *Cancers;* (*m.*) *Syphilomata;* (*n.*) *Aneurisms* (CRAIGIE, ROKITANSKY, VIRCHOW, NIEMEYER). Hence a list of cerebral tumors is a list of tumors generally, for every named kind of tumor has been found in the brain (see page 976, Vol. I.)

(*b.*) *Causation.*—The origin and progress of these tumors are very obscure; but tuberculosis and syphilis are sources of some of them, especially as scrofulous deposits in the meninges, or as gummata commencing in the *pia* or *dura mater,* or in the substance of the brain.

(*c.*) *Morbid Anatomy.*—Some tumors have special relations to the brain, either anatomically or pathologically. Of these are the following:—(1.) *The simple cerebral tumor* takes the form of an induration, or *scleroma,* of particular regions. There is an absence of red colour, and the part acquires the consistence of the white of egg boiled hard. Isolated portions of brain in this condition have all the characters of a cerebral tumor, and have been described as such by Meckel, Abercrombie, Greding, Bouillaud, and others. It is more correct to regard them, however, as portions of brain indurated to an unusual degree, and changed in structure (sclerosis). On section the part has generally a pale yellow or orange-grey tint, surrounded by some cerebral softening. The ventricles contain fluid; and if the indurated mass approaches the convolutions, the membranes become opaque, thickened, and adherent (CRAIGIE). It may be regarded as the hard form of the next kind of tumor—namely, (2.) *Gliomata,* which result from germinations of the *neuroglia,* or connective tissue of the brain; and as they grow up into tumors, the surrounding or implicated nervous elements become destroyed. Glioma is the true or proper tumor of brain tissue, almost special to the nervous centres. Microscopically they consist of roundish nuclei, distributed throughout a finely reticulated basement substance. They do not appear to form circumscribed tumors, but gradually to pass into healthy brain tissue; and are never seen to pass from the brain to the membranes of the brain. They generally originate amongst the medullary substance of the cerebrum. They are liable to hæmorrhagic exudations and fatty degeneration. In consistence they are between medullary cancer and healthy brain; firm and tough, and otherwise very like brain tissue, only more pellucid. On section they are seen to vary in colour from a whitish-yellow to a bright-grey red, translucent and highly vascular. Such tumors have often been taken for cancers; and they usually occur under the fortieth year of life (NIEMEYER). (3.) *Adenoid, sarcomata,* or *fleshy tumors,* are usually attached to, and seem to proceed from the meninges, generally from the *dura mater* at the base, in the vicinity of the sella tursica or petrous bones, although they appear to be for the most part imbedded in the cerebrum. Originating centrally in the neuroglia, their usual seats are in the optic thalami, corpora striata and quadrigemina, pons, cerebral peduncles, and cerebellum. It is occasionally met with in the cord substance. Sometimes they contain cavities filled with fluid. Microscopically they are seen to consist of spindle-shaped cells, arranged in fibrous-like striæ. They differ from cancer in being sharply bounded, and often inclosed in a vascular membrane, a continuation of *pia mater,* from which they can be turned out. They are liable to calcareous degeneration (CRAIGIE, NIEMEYER, VIRCHOW). (4.) *Strumous tumors, as tubercles of the brain,* or *tyroma,* are the most frequent form of cerebral tumors. They are composed of matter of a white or pale surface, yellow colour, firm, like soft cheese, but less tough, granular, and but little disposed to break down. The growth occurs in various forms :—(*a.*) As one to four, five, or six homogeneous masses of considerable size, from that of a pea to a walnut, always without blood-vessels or trace of organic structure. (*b.*) Numerous (30, 40, to 200) minute spherical masses disseminated through the substance of the brain (REIL, CHOMEL). They are located most frequently in the *cerebellum* or *cerebrum,* at its base; more rarely in the *central ganglia.* They are generally surrounded by loose connective tissue, and on section may disclose softening at the centre, as tubercular pus; or the centre is marked by dark discoloration. Sometimes the main portion of the tumor passes gradually into the brain structure by a slightly translucent exudation, consisting of young tubercle elements (see page 1000, Vol. I.). Such exudation indicates that growth is still going on, that scrofula is in the act of tubercle formation (CRAIGIE, NIEMEYER). They produce ventricular effusions and symptoms similar to chronic hydrocephalus. (5.) *Gelatiniform tumors, colloids,* or *myxomata,* are growths of a peculiar soft jelly-like, translucent material, like thin glue. Microscopically, they are composed of variously formed cells, imbedded in a mucous hyaline substance. They are found most frequently in the medullary substance of the cerebrum, or spinal cord, or in the membranes. They are liable to undergo blood extravasation, and to break down in their central part into cyst-like cavities, full of slimy fluid. Sometimes they appear to be congenital, and tend to grow large. The earliest indications of the lesion were epileptic seizures and loss of memory. (6.) *Adipose, lardaceous tumors,* or *lipoma,* are usually small, nodulated, and solid, attached to the dura mater; or they are cysts inclosing hairs, fat, or oily fluid, with excrescences from the inner wall of the cyst. (7.) *Cholesteatoma, pearl-like* or *margaroid tumors,* consist of white glistening globular masses like pearls, each mass varying in magnitude from the size of a vetch to a pea. The colour is of a dead silvery, pearly, or waxy grey, and the aggregation of those form tumors varying in size from a nut or walnut to a small pippin apple. The mass is generally irregular and elongated. They are situated mostly on the base of the brain, crura, or lower surface of the cerebellum, in the subarachnoid areolar tissue, between the *arachnoid* and the *pia mater.* They sometimes start from the cranial bones or meninges. They show no trace of organic structure, and chemically consist of cholestrine, with concrete layers of epidermic cells, inclosed in a delicate membrane (CRUVEILHIER, CRAIGIE, NIEMEYER). (8.) *Encysted tumors* are chiefly of two kinds, the one kind owing a parasitic source (see "Parasites," Vol. I., p. 144). The parasites contained in them are always immature, and transition stages of tapeworm parasites occur, such as the *cysticerci* or *echinococci.* The *cysticerci* are usually found in large numbers, and generally in the grey substance. Occasionally they have commenced to undergo degeneration (calcareous), but the hooks can generally be recognised. *Echinococci, hydatid tumors,* or watery bags, have been long known as existing in the brain. They form large vesicles, inclosed by their delicate germinal membrane, already fully described at p. 199, Vol. I. The blood cyst or hæmatoma has also been described (see p. 413, Vol. II.).

(9.) *Cancers,* in the form of medullary fungus hæmatodes, or scirrhus, are similar in the brain to their structure elsewhere, as described at p. 955, Vol. I. They sometimes start from the brain itself, from the *dura mater,* or the cranial bones; or, commencing in the external soft parts of the skull and neighbouring cavities, especially the orbit, they infiltrate by the vessels and nerve-sheaths, and so press into the skull. Although it is rare for cancers commencing inside to penetrate the membranes and skull bones, yet such growths frequently make their way outwards, and protrude from the orbit, when they at once commence to soften and ulcerate. Such tumors often commence symmetrically (CRAIGIE, NIEMEYER). Cerebral carcinoma has been found in a most typical form as secondary to scirrhus of the breast. Its structure is more hyaline than elsewhere, often of the nature of *myeloid* growths, a semifluid or almost fluid pulp, with very large giant cells. (10.) *Syphilomata* occur in the brain as in other parts, as *gummata* or nodes, or as diffuse infiltrations. They have been already described at p. 922, Vol. I. (11.) *Aneurisms* of the cerebral arteries form tumors generally at the base of the brain, in connection with the blood-vessels there, especially the *basilar arteries,* those of the *corpus callosum,* and the *Sylvian fissure,* and the communicating artery of the *circle of Willis.* Usually they are about the size of a pea or a hazel nut, but sometimes are larger before they rupture.

Symptoms of Tumors—1. *In the Brain,* are not characteristic as distinguished from *softening, abscess,* or local lesions, already described. They present the greatest variety, and all produce certain common changes in the contiguous cerebral substance. All of them tend to some extent to derange the capillary circulation of the brain and its membranes, and to that extent they tend to produce symptoms of cerebral irritation. The greater the vascularity and congestion of the tumor, the greater will be the infiltration of the whole brain by serum. In connection with the membranes, excitement and congestion of the *pia mater* and grey substance of the hemispheres are attended with peculiar psychical phenomena, and often by the symptoms of effusion into the ventricles. Chronic headache and epileptiform attacks are most common, and when the vascular excitement of tumor and brain is at the greatest, then loss of memory and of intellect, and the occurrence of convulsions or palsy, are the results. In some cases headache is one of the earliest symptoms in association with vertigo and vomiting. If the growth of the tumor is slow, and its size small, little influence may be exercised on the general functions of the brain; but according to its locality will certain special indications occur. There is an utter absence of constant symptoms pathognomonic of tumors of the brain; and the diagnosis must be made (*a.*) from the history of the case; (*b.*) the physiological phenomena indicating the location of lesions at a point where tumors are known to be common and other lesions rare; (*c.*) from peculiarities in the course of the disease. The *history of the case* may show an exciting cause for brain disease—e.g., *parasites, syphilis, diseases of the heart* (hypertrophy, or of the valves), *or of the arteries* (degeneration), *cancers, scrofula, injury to head, caries of petrous bone.* If it is considered that a tumor is the lesion, its nature may also be recognised from some of the causes of brain disease. The symptoms may be of brain disease or lesion, but cannot be otherwise regarded as indicating a tumor; and tumors may reach a large size without inducing symptoms, provided their growth is—(1.) slow; (2.) not in the vicinity of the central ganglia; (3.) not in the vicinity of vessels, so as to interfere with the flow of blood to and from the brain; (4.) not vascular in themselves, so as to swell at times from overfulness of blood, or suddenly decrease from containing less blood; (5.) not of such a kind as will compress capillaries, so as to cause such a change in the brain as will lead to loss of function of the part and its connections where the tumor is located.

The more important symptoms which tumors may induce are:—(1.) Severe, persistent, and sometimes intermittent headache is often for a long time the only symptom indicative of brain disease—presumably tumor, if the headache is usually intense and severe. The continuousness of the headache may be interrupted in the case of vascular tumors, when fulness or emptiness of vessels may cause exacerbations of pain. Constant pain referred to the back of the head may point to a stretched tentorium as the probable site of a tumor in the posterior cerebral fossa. (2.) Giddiness and vomiting accompanying headache also point to brain-lesion, and confirm the probability of tumor. Vertigo is rarely absent; sometimes it is the first symptom, and often it is the most constant. It is of frequent occurrence in the tubercular tumors of children. (3.) Local symptoms consist of *hyperæsthesia, neuralgia, formication, twitching, partial anæsthesia, partial paralysis*—all of which may commence before headache. Such symptoms are referable to disturbances of circulation in the vicinity of the lesion, and are not pathognomonic, because they are associated with so many other lesions; but if these phenomena are limited to *cranial nerves,* they are suspiciously indicative of brain disease—probably tumor. (4.) Lastly, cerebral tumors sometimes cause blindness; and when blindness is due to cerebral tumor, the ophthalmoscope furnishes important information, as shown in the changes of the optic disc. (*a.*) Simple swelling of the optic papillæ, with great tortuosity of the *vena centralis,* indicates obstructed venous circulation, most frequently seen with tumors encroaching on the orbit from within. (*b.*) Slight inflammatory swelling of the optic papillæ, with less distinct venous hyperæmia and inflammation of retina, occur with inflammation of optic nerve and its membranes, extending to the retina. (*c.*) Atrophy of optic nerve from a tumor, or from meningitis. (5.) If the symptoms have come on so slowly and gradually as that the commencement cannot be fixed, the fact points to tumor rather than to any other lesion ; unless where the tumors are vascular, when the symptoms may appear suddenly in consequence of vascular changes within the cranium. (6.) Epileptiform convulsions occur more frequently from cerebral tumors than from any other cerebral lesion, and especially when the tumors are located in the cerebrum, and near the cortical substance. When the tumor perforates it is usually through the parietal or temporal bones; less often through the occipital, when it may sometimes be seen to move with respiration. (7.) The general functions and organic processes of the body are sluggish, pulse slow, and respirations under the normal; bowels constipated, and urine scanty. The body may increase in bulk, or marasmus may be excessive, with bedsores and dropsy of the feet and ankles. (8.) In a large proportion of cases there is increasing failure of memory and hebetude. Aphasia may occur; or the patient may become incoherent in speech, or fatuous or furious as to his conduct. Under these circumstances he may lose or fail to exercise control over his evacuations, or he becomes delirious or maniacal, or he may give way to boisterous laughter, or become low-spirited and apt to cry (BRISTOWE).

2. *In the Cerebellum,* tumors may exist without causing any symptoms. They occur only when the middle lobe is affected. The most important signs are :—(1.) The *reeling gait,* the ataxy being almost invariably confined to the legs and body. (2.) A peculiar form of *vertigo,* extremely severe, and sometimes permanent. It sometimes occurs before the reeling gait is noticed; and also when the patient is in the recumbent position; but it is most marked when he sits up in bed or attempts to walk. It gets worse on closing the eyes, and is relieved by support. These two signs must be coexistent to indicate cerebellar tumor (ALTHAUS, *Lancet,* May 8, 1880).

3. *In the Spinal Cord,* symptoms of tumor are due to irritation or destruction from compression of its substance, or involvement of the roots of the nerves, which have been already described under "Paralysis."

Prognosis.—Always unfavourable. Death by *coma* is the most usual termination, either directly or by intercurrent disease.

Treatment.—Under such circumstances medicinal treatment can be only palliative: the patient may be protected as much as possible from hyperæmia of the brain aggravating the morbid state. His nutrition and mode of life must be regulated, and also the functions of the bowels. Apoplectic or local inflammatory attacks may be met by local blood-letting and cold compresses. Hypodermic injections of morphia are also to be used in suitable cases. If syphilis exists, or is suspected, anti-syphilitic remedies are at once to be adopted.

(5.) *The influence of nervous diseases over processes of nutrition* consequent on lesions of the cerebro-spinal axis and of the peripheral nerves, exhibits itself mainly in affections of the skin, the connective tissue, the muscles, the articulations, and the viscera, especially the bladder. These secondary affections are at first inflammatory. They make their appearance with incredible quickness, and proceed with great rapidity in their development after the lesions of nerves, or nerve centres, are set up which cause these trophic affections. They are like the ulcerations of the cornea which come on after section of the fifth nerve. Eschars, for example, may form on the sacrum the second or third day after an accident causing compression and irritation of the spinal cord; or a few days after cerebral hæmorrhage. The primary lesions of the nerve-centres which influence the trophic conditions are irritative or inflammatory, and ultimately destructive in their effects. These secondary trophic affections are mainly brought about by—(1.) Lesions of peripheral nerves, either traumatic or idiopathic. (2.) Lesions of spinal cord and medulla oblongata. (3.) Lesions of certain portions of the encephalon. The nerve lesions give rise to—(1.) *Skin affections*, of the nature of eruptions (limited to the region of distribution of a single sensory nerve), such as vesicles and bullæ in the form of *zona* (*herpes zoster*), pemphigoid eruptions, cutaneous redness, glossy skin (smooth, pale, bloodless), which wastes and becomes very thin. (2.) *Affections of the muscles*, in rapid wasting and loss of electrical contractility. A form of *progressive facial hemiatrophy* may be referred to here, in which the trophic functions of the fifth nerve are especially implicated, the atrophy being limited to the parts supplied by this nerve. In some cases there are evidences of positive lesions of other cranial nerves, such as paralysis or irritation of the *sympathetic*, of the *abduceus*, of the *auditory* and *glosso-pharyngeal*, as well as alteration of nerve nuclei in the medulla (BANISTER, *Journ. of Nervous and Mental Diseases*, Oct., 1876). (3.) *Affections of joints—e.g.*, subacute inflammations, terminating in anchylosis. (4.) *Affections of the bones—e.g.*, periostitis, followed by necrosis. These secondary lesions occur spontaneously without the intervention of pressure; and after irritative lesions of the spinal cord and medulla oblongata. It is only when the lesions affect the system of motor nerve-cells in the cord that deterioration of muscular nutrition takes place. The affections of the spinal cord which determine grave disorders in the nutrition of the muscles are—(1.) *Acute central myelitis.* (2.) *Spinal apoplexy.* (3.) *Irritative lesions caused by fractures and luxations.* (4.) *Affections of the grey matter of the anterior cornua, as in infantile spinal paralysis, progressive muscular atrophy.* In *locomotor ataxia*, cutaneous affections may be noticed of the nature of—(*a.*) *papular* or *lichenoid eruptions;* (*b.*) *urticaria;* (*c.*) *zona;* (*d.*) *pustular eruptions analogous to erythema.* Partial lesions of the encephalon may also give rise to zona, as after hemiplegia. Eschars of rapid development: *decubitus acutus*, or *acute bedsore*, shows itself as an erythematous patch, on which vesiculæ and bullæ rapidly develop, terminating in mortification of the skin and subjacent tissue. It usually occupies the sacro-gluteal regions; or on parts subjected to very slight pressure for a short time, or even on parts without pressure. It occurs in cases of apoplexy from cerebral hæmorrhage, or from softening of the brain, and spinal lesions. It occurs on the paralysed side, from the second to the fourth day after the attack, and is symptomatic of circumscribed cerebral disease. Its appearance gives ground for an unfavourable prognosis. Bedsore from myelitis and injury of the spinal cord affects especially the sacral region in the middle space extending symmetrically on either side. Affections of the joints and joint-ends of bones are either acute or subacute. They appear in cases of traumatic lesion of the cord, and occupy the joints of the paralysed limbs; in myelitis from tumors or Pott's disease, primary syphilis, and in recent hemiplegia connected with cerebral softening. The chronic forms depend on lesions of the anterior cornua of the grey axis, and occur chiefly in posterior sclerosis (locomotor ataxy), and in certain cases of progressive muscular atrophy. Other forms of these secondary lesions are—*visceral ecchymosis, nephritis,* and *cystitis,* as complications of irritative spinal affections, with discharge of purulent, bloody, ammoniacal, and fetid urine. These visceral and cutaneous lesions are mainly due to involvement, either of sensory nerves or the grey matter of the cord posterior to the central canal, or the immediately adjoining portions of the posterior columns of the cord or ganglia at base of brain. The lesions which determine muscular and arthritic mischief are situated either in the course of motor nerves or in their nuclei of origin.

(6.) *Progressively ascending, descending, and collateral lesions* are often the slow result of the many circumscribed affections of the brain, spinal cord, and nerves, which have been the subject of consideration in some of the previous pages. Degenerative changes follow the existence of the primary lesions in the slow course of time, either in the nervous tissue above them, below them, or in both directions. The degeneration may also spread laterally. When, for example, hæmorrhagic foci are of long standing they give rise to various secondary changes, not only of the parts in their immediate vicinity, but of others in distant regions (see pp. 95, 395, 422, 467, *ante*). This secondary degeneration (first made out by Türck in 1851, and subsequently confirmed by Vulpian, Charcot, and Bouchard) consists essentially of an atrophy of the nerve fibres, and the development in their place of connective tissue, or the substratum of a structureless homogeneous material (Dr. FRED. TAYLOR). When the extravasation is situated in the corpus striatum involving the internal capsules, the path of descending degeneration seems to be in a band-like form, along the motor tract, through the crus cerebri, the pons, and the anterior pyramids. Within the latter, the degenerative process passes over, in its descent, to the other side of the medulla, and may thence continue its course downwards through the entire length of the spinal cord, occupying especially the posterior portion of the lateral white column. It may also affect the cord, to some extent, on the same side with the lesion, when it is confined to the inner part of the anterior column (NOTHNAGEL). In a case of brain disease with degeneration of the cord, described by Dr. Fred. Taylor, involving all the white matter of the posterior half of the cerebrum, and running down tongue-like into the temporo-sphenoidal lobe, involving the posterior extremity of the lenticular nucleus of the corpus striatum, the corpora geniculata and posterior portions of the optic thalami were involved in a mass of gelatinous material. The spinal degeneration was so complete, that in every part of the spinal axis the whole area of every section in the pyramidal strands was diseased; and the degeneration corresponded precisely in the crus, pons, medulla, and spinal cord, with the course of the pyramidal tracts (*Guy's Hospital Reports*, Vol. XXIV., p. 179, 1879). Lesions situated outside the ganglia in the centrum ovale, if they attain certain dimensions, also produce descending sclerosis, if they are not too far off the base of the corona radiata, also cortical lesions of some depth and extent (implicating grey and white matter) of the ascending parietal and ascending frontal convolutions, and the adjacent parts of the parietal and frontal lobes determine consecutive sclerosis. When lesions occupy the lower part of the cord, degeneration changes gradually ascend through the posterior columns, on either side of the posterior median fissure.

General atrophy of the brain is a secondary change which also sometimes occurs.

DISSEMINATED SCLEROSIS; MULTIPLE SCLEROSIS—Syn., INSULAR SCLEROSIS (MOXON), CHARCOT'S DISEASE (ALTHAUS).

FRENCH EQ., *Sclérose en plaques disséminées* (CHARCOT).

Definition.—*Clinically, the condition is characterised by disorders of vision (mainly from oscillation of the eyeballs), speech, and intellect; and by tremors of the head, neck, trunk, and limbs, which supervene only on purposive movements. There is also gradually supervening paralysis (with contraction), mainly of the lower extremities. These conditions are due to hardening which invades the cerebrum, the pons Varolii, the cerebellum, the medulla oblongata, and the spinal cord, in small round spots or patches of a greyish colour and of irregular outline.*

Pathology.—(*a.*) *History.*—Cruveilhier and Carswell first illustrated this disease. Türck, Rindfleisch, Zenker, and Frommann, afterwards contributed data for its recognition. Up to 1858 it had not found any description in this country; and to Charcot especially we owe almost all our knowledge of the morbid anatomy and the recent clinical details which pertain to it.

(*b.*) *Causation.*—Women rather than men are its subjects, between twenty and twenty-five years of age. It rarely occurs after thirty; and its patients rarely reach the age of forty. Wet and cold, prolonged anxiety, moral influences, and certain acute diseases, such as cholera and enteric fever, are also said to have been active agents in setting it up.

(*c.*) *Morbid Anatomy.*—The sclerotic patches are of a grey colour, forming a striking contrast to the healthy tissue (white) in which they are imbedded. They resemble the grey matter, but become of a pink colour on exposure to the air, and contain numerous blood-vessels. They are found chiefly in the central ganglia and in the antero-lateral columns of the cord; and, also, in certain peripheral nerves, such as the olfactory, optic, and fifth; and in the anterior and posterior nerve roots. There is thickening of the neuroglia round the patch, the nuclei being more bulky and numerous; hence the nerve tubes are separated, and lie at a greater distance from each other. The myeline cylinder, or white substance of Schwann, is wasted, and the cylinder axis may be hypertrophied. Centrically, the nerve tubes become more and more wasted; the cylinder axis has enlarged, while the white myeline cylinder has almost quite disappeared. The neuroglia becomes more transparent, and in some places is replaced by ordinary connective tissue. In the centre of the patch the neuroglia appears predominant, or at any rate the elements of sclerosis; the nuclei are less numerous and bulky. Carmine ceases to colour them. No myeline is seen; and the cylinder axis is reduced in size. The coats of the blood-vessels are thickened in the peripheral zone, mainly from germination of nuclei, which continue more numerous in the central portions of the patch, so that the calibre of the vessels is considerably reduced. Free oil-globules and granule-cells stud the outer portions of the patch, and accumulate within the lymphatic sheaths of the vessels. Corpora amylaceæ tend to become developed in the course of the vessels. It is thus a chronic form of *interstitial myelo-encephalitis.*

Symptoms.—The expression and mental condition of the patient are changed. The glance vague and uncertain, the lips pendulous and apart, give a sad, weak or fatuous aspect. Memory fails, the conceptions are slow; intellectual and emotional faculties are generally impaired. There is general indifference to all that goes on. Mania or dementia may supervene. *With regard to paralysis and contracture,* paresis affects especially the lower limbs at an early period, commencing generally in one leg. The movements do not suggest inco-ordination. After the other leg becomes affected, the weakness extends to the arms, and increases generally till motor power is absolutely lost, and tremors diminish till they finally cease. Muscular sense, reflex and electrical contractility, remain unimpaired previous to the paralysis. There is no paralysis of bladder or rectum, and the muscles retain their form, bulk, and tonicity. They retain galvanic and faradic contractility. The lower limbs, under various forms of excitement, or without any apparent cause, become suddenly stiffened in extension and pressed against each other · and so continuing for some hours or days. These attacks are at first separated by long intervals, but sooner or later the rigid extension of the muscles becomes permanent, and the limbs lie in close apposition, and cannot be separated. The limbs may sometimes be flexed at the joints. At a later period the arms may be affected like the legs, and pressed against the sides. The disease usually commences with cerebral symptoms, such as double vision and vertigo, followed by embarrassment of speech and nystagmus. Weakness of the lower extremities, more or less remittent, may precede the cerebral symptom for some months, or even years, without pain or loss of sensation. Towards the end the appetite fails, and the patient is liable to diarrhœa. Intelligence fails, and the patient becomes fatuous. Evacuations are passed involuntarily, the bladder inflames, and bedsores form. The pulse is quickened, and the temperature rises to 103° or 104° Fahr. The symptoms may be arranged in groups:—(1.) A peculiar form of tremors, somewhat rhythmical in character, and only manifest when purposive movements of a certain extent are made. They cease completely during sleep, and when the limbs are at rest. (2.) Signs which point to intracranial lesions. (3.) Paralysis and contracture. *As to the tremors,* any muscular effort whatever causes them, and the more precise the object to be attained, the more tremulous are the tremors, which increase in tumultuousness as the task approaches completion. Efforts to restrain movement cause the movements to become more intense. *The signs which point to intracranial lesions* are especially—(1.) Affections of the eyes, such as double vision, nystagmus. (2.) Defects of speech, such as slow and drawling utterance—a pause after each syllable, each of which is slowly spoken and imperfectly pronounced. Speech may become unintelligible. (3.) Vertigo is an early symptom in three-fourths of the cases. It is generally gyratory, in paroxysms of short duration. All the objects seem to spin round the patient and he with them.

Diagnosis.—It is to be distinguished from locomotor ataxy by the nature of the tremors, and by the presence of nystagmus; and from tabes and paralysis agitans by the presence of vertigo. The trembling, combined with the cerebral phenomena, are pathognomonic. Anatomically a certain number of axes cylinders are present in the otherwise completely altered tissue of the sclerosed patches; which do not exist in the sclerosis of the posterior columns which is characteristic of progressive locomotor ataxy, in which want of co-ordination is increased when the patient closes his eyes, which is not the case in this disease.*

Prognosis.—The disease may last from six to ten years. Death generally takes place by pneumonia, phthisis, cystitis, dysentery, or apoplectic attacks, bedsores, or failure of the nutritive powers.

(7.) *Peripheral, central, and reflex sources of disease*, with some of their consequences, have been already illustrated under the topic of *Reflex Paraplegia* (page 370, *ante*).

IMPAIRMENT AND LOSS OF POWER OF SPEECH: APHASIA, APHEMIA, AMNESIA.

The nervous arrangements for the physiological movements of articulate speech are situated in the medulla oblongata, pons, and corpora striata; and the diseases connected with lesions in these parts have been already described, especially in relation to impairment of the muscular and nervous apparatus. The utterances of intelligent language in the expression of syllables, words, and sentences, depend on the integrity of a definite portion of the substance of the hemispheres. The main current of speech flows through the left hemisphere; less through the right. Hence, the left hemisphere is chiefly trained to speech in infancy and youth with right-handed people, as well as for combined voluntary movements. Most people are left-brained and right-handed. Dr. Broadbent has usually found the third frontal convolution larger on the left side than on the right. The posterior part of this convolution has also more abundant and complicated connections with other convolutions than any other part of the brain. Besides central and callosal fibres (and of the former, very many pass directly down to the lenticular nucleus), it receives numerous fibres from the fasciculus uncinatus, from the convolutions of the insula, and from the adjacent convolutions along the upper edge of the fissure of Sylvius; it is further connected by associated fibres with the frontal and parietal gyri. In the complicated structures of this convolution, its anatomical fitness may be recognised for the outlet of language. In structural relations there is no marked difference between the convolutions of the two sides (DODDS, l. c., p. 652). If the power is lost on the left side by the subsequent education of the right hemisphere, it may still be made efficient for speech (see SHARPEY on "Education of the Brain in Old Persons," in *Brain,* April, 1879). After destruction of the area for language, emotional language and the power of using interjections—to swear, and to say "yes" and "no"—are still possible.

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* In 1875, Dr. W. Erb, Professor at Heidelberg, described a form of sclerosis which in its symptoms is sharply characterised, and distinguished from the great group of spinal paralyses—the "*tabic diseases.*" He named the affection "*spastic spinal paralysis,*" equivalent to the "*tabes dorsalis spasmodique,*" or "*amyotrophic lateral sclerosis,* of Charcot. The *gait* of the patient is characteristic, and to be distinguished from the gait in common paralysis, where the patient walks in a shuffling manner, dragging his feet after him, with the tips of the toes pointed to the ground and his heels raised up. It is also to be distinguished from the *ataxic* gait, in which the patient throws his feet forwards and outwards with considerable force, bringing the heel to the ground first, and if his eyes are not on the ground to see his feet, he reels and staggers like a drunken man. In the *spastic spinal paralysis* of Erb, the gait is described as "spastic." The patient walks, or rather runs. The feet while they seem to stick to the ground, yet the tips of the toes come in contact with obstacles, and catch in every hindrance or inequality of the surface of the ground, so that the gait is a "hopping elevation of the whole body," which is bent forwards, the legs keeping close together at the knees. The legs are thus dragged behind, the feet appearing to stick to the ground, and to be moved forward with great difficulty. There is great muscular tension, and reflex contractures occur in the various groups of muscles which are set in action in walking. The *sclerosis* is said to be in the lateral columns of the cord; but the existence and position of the lesion have not yet been verified by *post-mortem* examination; and it remains still to be proved whether the group of symptoms of this affection described by Erb and Charcot, belongs to lateral sclerosis or not. The irritative phenomena consist of heavy, dragging, and tearing pains in the loins and legs, burning feelings or sensation of cold in different parts of the skin. The pains are not so severe as in *tabes.* The initial feebleness begins in the lower extremities, first in one leg, and then in the other. In the older and graver cases there is complete paralysis. There is an early tendency to

There have been several attempts to localise the faculty of speech. Those of Bouillaud, Dax, and Broca, may be taken as representative of these different theories.

(1.) Bouillaud's theory places the psycho-motor centre for the expression of language by speech in the anterior lobes, including the ascending frontal and the central lobe or insula. His views have been, on the whole, amply vindicated (DODDS, *Journal of Anat. and Phys.*, Vol. XII., p. 464, 1878).

(2.) Dax's theory maintains that the faculty of language (the psycho-motor centre for speech) is seated in the left hemisphere only—a theory which cannot be maintained in its entirety; but which, in the great majority of cases, apparently holds good (DODDS, l. c.).

(3.) Broca's theory holds that the speech-centre is always and only present in *the posterior part of the third left frontal convolution*—that is, the part whose integrity is necessary for intelligent language. It is that convolution which is trained for the verbal expressions of thoughts and ideas. The area of lesion destructive of speech is limited, and generally due to embolism of the left middle cerebral artery. The theory is open to criticism (DODDS, l. c.): (1.) It may be disproved so far as it avers the constant and uniform left-sidedness of the speech-centre, which is not so unilateral

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cramps (CHARCOT). There is increased reflex excitability of the tendons, which increases with the duration of the disease; especially is this so with the patellar tendon. This disease is to be distinguished from chronic transverse myelitis, tabes dorsalis, chronic atrophy of the anterior cornua, multiple sclerosis, and paralysis of the cauda equina, and from cerebral hemiplegia (SAUNDBY, in the *London Med. Record*, Nov. 15, and Dec. 15, 1877).

as is generally supposed; and, (2.) it is doubtful whether the posterior part of the third frontal convolution includes the actual limits of the centre. In some instances the speech-centre is on the right side. Aphasia has occurred with complete integrity of the third left frontal convolution, and cases of positive lesion of this convolution without aphasia.

Dr. Dodds would therefore extend and enunciate Broca's theory as follows:—"*That there are two speech-centres—one on either side—contained in the island of Reil, the lowest part of the ascending frontal, and the posterior part of the third frontal convolution; that in the great majority of cases the centre on the left side (though exceptionally that on the right), is of pre-eminent importance; but in either case, the other centre always plays an active, though secondary part in the production of speech.*" He is also of opinion that from the bilateral co-ordination of the lower centres, speech may be carried on though either centre be destroyed—with difficulty if it be the leading, with comparative ease if it be the non-leading centre that is destroyed. In the former case the difficulty (amounting frequently to practical impossibility) of re-establishing the power of speech, will depend upon the age of the patient, his mental condition, the state of the channels of sensation (*e.g.*, hearing and sight), through which he may be reached, and on his aptitude for learning to take the first step, as it were, with the opposite side (DODDS and HUGHLINGS JACKSON). Precision in localisation of lesion in aphasia is of paramount importance. Broca's theory thus extended has so far demonstrated positively that disease of the left supposed centre can produce, and almost invariably does produce, ataxic aphasia; and it has also been shown, that in rare cases aphasia can likewise be caused by disease of the *right* supposed centre. Hence it cannot be reasonably doubted that the speech-centre is included in the island of Reil, the lower part of the ascending frontal, and the posterior part of the third frontal of either side, destruction of which *must* cause ataxic aphasia. But there are still cases unexplained even by this comprehensive theory. Nevertheless, there are facts which lend probability to the view that *the co-ordinating centre* is in the posterior part of the third frontal, and the adjacent part of the ascending frontal; and that the seat of *the "motor" memory of names* is in the island of Reil, while the anterior frontal convolutions are specially related to the higher intellectual processes. The speech-centre is, therefore, not so unilateral as is generally supposed. Dr. Barlow records the case of a boy who had an attack of right hemiplegia and aphasia, from which he had almost recovered when he had left hemiplegia, with difficulty in deglutition, and complete aphasia. The limbs gradually improved, but the speechlessness remained, though after a time he was able to write replies on paper. *Post-mortem*, there was found embolism of both middle cerebrals, with very limited and symmetrical softening of the lower part of the ascending frontal, and of the second and third frontals of either side.

Aphasia is of two kinds, namely—(1.) *Amnesia* or *amnesic aphemia*, in which there is difficulty in remembering the spoken or written words. The idea exists, but the symbol of its expression cannot be called forth. There is a loss of memory for words. With perfect vision the patient cannot read; he cannot write, nor make himself understood from what he may try to write. He cannot recall facts nor concentrate thoughts, nor reason in sequence. (2.) *Aphemia*, or *atactic aphesia*, in which there is loss of motor co-ordination of words. Typical cases are rare. The memory is not at fault, but the patient cannot pronounce words, and can generally say only "Yes" or "No," or monosyllables. The wish to speak does not evoke the combined automatic movements on which speech depends. When the patient can express himself correctly in writing, although unable to speak, his condition is described by the term "aphemia." He understands the words addressed to him, but he is unable to speak in response, for various reasons. Some can express in writing what they cannot speak. The clinical conditions under which *aphasia* or defect of speech occurs, are especially—(1.) Right hemiplegia from cerebral hæmorrhage (in rare cases left—the patient probably being left-handed); 2. unilateral convulsions; (3.) chorea; (4.) enteric fever in children; (5.) General paralysis; spinal paralysis, locomotor ataxy, and disseminated sclerosis; glosso-laryngeal paralysis, and lesions of one or more motor nerves of the organ of speech.

DISORDERS OF THE INTELLECT—INSANITY, INTELLECTUAL AND EMOTIONAL DISTURBANCES.

Definition.—*Disorders of the mind consequent on brain lesions, or attendant on affections in which the brain is secondarily or remotely implicated. Feelings, Emotions, Intelligence, or Will of the patients, conjointly or separately, are exalted, perverted, or impaired.*

Pathology.—(*a.*) *History.*—The term "Insanity" has not been recognised in the nomenclature of the College of Physicians, and the subjects hitherto described under that popular term are now comprehended under the general heading of "Disorders of the Intellect." Under this title the following six varieties are specified by the College,—namely, *Mania* (acute and chronic), *Melancholia, Dementia, Paralysis of the Insane, Idiocy, Imbecility.* With the exception of that species of insanity associated with general paralysis (already described, p. 342, *ante*), the other named varieties are but symptoms of one or other species of insanity (W. H. O. SANKEY, *Journal of Psychological Medicine*, April, 1877). It is difficult, or impossible, to define in words the terms "*insanity*" or "*lunacy.*" *Insanity* comprehends all unsoundness of mind. *Lunacy* is a legal term, representing only those deviations from a standard of mental soundness which is universally recognised, although difficult of definition, in which the person, the property, or the civil rights may be interfered with; where incapacity, or violence, or irregularities threaten danger to the lunatic himself or to others. *Insanity* is therefore the more comprehensive term, including all states of the mind which are inconsistent with the original and ordinary character and habits of the individual, and with his relations to the family or community of which he is a member. Etymologically, the term implies the negation of something, and not a positive entity. It has been said that nobody is quite sane; and, indeed, the old Latin poet and satirist wrote: "*Orandum est ut sit mens sana in corpore sano.*" He did not say that a sound mind ever existed, but he said it was a thing to be prayed for (Dr. W. ORANGE, *Brit. Med. Journ.*, p. 511, 1877).

Many theories have been propounded to explain the nature of *disorders* embraced by the term "*insanity.*" They may be resolved into two, as at present entertained, namely,—(1.) The *metaphysical, functional,* or *spiritual* theory, which inculcates the belief that these disorders imply an affection of the immaterial principle. Such a belief is at variance with all reasoning, and in direct opposition to positive and well-recognised data. The brain is the material instrument by which the mind acts, whether it be by the unseen phenomena of *conception, judgment, reasoning,* and *instinct,* or by the more obviously expressed phenomena of *volition, emotion,* and *sensation.* We know of no mental manifestations except through the brain. We know nothing of the actions of the *mind*, except through the interposition of that material organ; and that no mental process ever emanates except from such an organism, or is received except through a similar piece of machinery. *Spiritualism* is not in vogue with us as a profession (WILKS). To consider, then, those *subjective* phenomena which constitute "mind" (an immaterial essence) as liable to disease apart from all derangement of the material organ,—namely, the brain (the instrument with which it works), is to believe in an incongruous unphilosophical, unphysiological doctrine. (2.) The *cerebral* theory, which is the more consistent one regarding the nature of insanity. The belief which it inculcates is that the instrument through which the phenomena of mind are expressed is the part diseased;—that the encephalic nervous textures are primarily implicated.

The *legal* value of the ordinary terms used with reference to the insane ought to be clearly understood. (1.) There is no difference between "*insanity*" and "*unsoundness of mind,*" none between "*insane*" and "*unsound.*"

Delusion.—Every sense may express, by the mode in which it performs its function, the existence of cerebral disease; as when light things feel heavy, small things seem large, hot things feel cold; or when the special senses become morbidly *active*, the patient seeing persons or hearing discourses when no such person is present, and no such discourse is being related. The term "delusion" generally includes all those phenomena: and although much objection has been taken to the term as signifying merely an unfounded belief, the "symptoms of delusion are still accepted in our courts of justice as the most authentic mark of insanity, and as the essence of cerebro-mental disease." *A delusion has been defined as a belief in the existence of things which have no existence in reality, or an erroneous perception of the nature of things, or of their relation to each other, occasioned by cerebro-mental disease;* or again (as Dr. BUCKNILL defines it), *an intellectual error caused by the pathological condition of the mind, and displaying itself in false sensation, perception, or conception* (BUCKNILL and TUKE, p. 317). The larger number of cases of well-marked insanity that fall under our notice are characterised by delusions —a leading feature of which is their persistency. There are two classes of delusions thus symptomatic of insanity:—(1.) *Illusions,* as when the sight or the hearing are the media of morbid or mistaken perceptions; as when individual persons are seen when they are not then present. Or, when the ear is affected, hearing "*voices*"—"the airy tongues that syllable men's names." (2.) *Hallucinations* arise when no impression has been produced on any of the senses. They are new creations due to an abnormal condition of the sensorium. Illusion and hallucination thus have reference only to disordered perceptions. Both separately or together may exist without insanity. *Illusions* are objective; *hallucinations* are subjective. Hallucinations sometimes occur when the organ is itself destroyed through which they would be *objectively* expressed, thus showing their *subjective* nature. The images thus excited are described to be as vivid as those produced by objective causes; so that the patient, when insane, entirely believes the empty and false form he sees, the ideal sounds he hears, to be real and substantial. Auditory hallucinations are of common occurrence in the insane, and are of bad omen. *Illusions* differ from *hallucinations* in this, that the *optic nerve*, or the *auditory* nerve, has conveyed an impression which thought or idea perverts. They are "false appreciations of real sensation" (BRIERRE DE BOISMONT). They are the misapprehensions or false interpretations of external objects which really do exist, so that the most harmless beings may become objects of great terror to patients, and especially to children. Hence the danger of disordered intellect arising from fright. *Dizziness* is a familiar instance of a simple *hallucination.* It consists in a vivid representation of movement, either of the body of the patient himself, or of surrounding objects which are actually at rest. It is sometimes difficult to distinguish between *illusions* and *hallucinations* when they relate to general sensation. "Whatever their origin, should the patient's reason still enable him to discard them as false and unsound, he is sane in relation to them, and this happens with illusions, though seldom with hallucinations. If he cannot be convinced of their unreality, he is very generally insane; but not always, as early training, peculiar religious views, and other special circumstances may account for apparent mental unsoundness" (FINLAYSON, l. c., p. 229; also BLANDFORD, *Journ. Mental Science,* Vol. XIX., p. 507).

(*b.*) *Causation.*—In modern times disorders of the intellect are unhappily of frequent occurrence, and they have been supposed to be extending in proportion to the degree of civilisation. On the other hand, however, an exhaustive inquiry by Dr. C. L. Robertson (*Journ. of Men. Science,* April, 1869), and another by Dr. H. Maudsley (*Brit. Med. Journ.,* Jan. 13, 1872), warrant the following conclusions :—

1. There is no satisfactory evidence of an increase in the proportion of occurring cases of insanity to the population; and no evidence, therefore, of an increased liability to insanity. 2. It is not necessary to assume such an increase in order to account for the undoubted great increase in the number of registered insane persons. 3. The difference between one insane person in 802 of the population in 1844, and one in 400 in 1870, is mainly, if not entirely, owing to the fact that in the former year the returns included only half the existing insane persons in the country, while in the latter year nearly all of them have been registered. 4. Some part of the difference is owing to the fact that certain patients are registered as lunatics now who would never have been thought so in times past. 5. A lower rate of mortality and a lower percentage of recoveries may account for a part of the increase in the total amount of insanity. 6. The proportion of admissions to the population, which

represents approximatively the occurring cases of insanity, does not, when the necessary allowances are made, yield evidence of any serious increase.

The remote causes of disorders of the intellect are of a moral or physical nature. Of the patients principally admitted into the different hospitals of France, Italy, and Belgium, about *one-tenth* have their insane state attributed to falls, blows on the head, the abuse of mercury, or other physical causes not determined. The remainder of the cases have their disorders ascribed to moral influences—such as religion, love, jealousy, family disputes and family cares, reverses of fortune, wounded pride, disappointed ambition, anger, fright, excess of study, libertinage and drunkenness. It is sometimes difficult, if it is always desirable, to separate physical from moral causes in our speculations on the etiology of insanity. The action of moral influences in producing insanity is so striking, that the passing events of the day often impress upon the disease its peculiar characteristic. The causes of disorders of the intellect are of a nature producing in the patient, in the first instance, *emotional changes* only, either by the sudden and violent agitation of the passions, or by the long-continued influence of circumstances operating more insidiously upon the mind, and producing an habitual state of abnormal feeling. There is no description of disordered intellect which, if traced to its source, may not be found either to consist in perverted emotion, or to emanate from that origin (BUCKNILL in *Med.-Chir. Review* for January, 1854). Dr. Hood's statistics give the proportions as follows:—*Moral causes*, such as anxiety, grief, uncontrolled emotions, in 40·8 per cent. of the males, and 32·7 per cent. among females. *Physical causes*, in 19·8 per cent. of males, and 21 per cent. of females. The chief of these physical causes are *old age*, abuse of alcoholic liquors, bodily illness, and the critical period in females.

The principal predisposing causes are *age*, *sex*, *hereditary descent*, and *inherited disease*. (*a*.) *Age*.—Infancy is nearly exempted from insanity, and so also is childhood, except in congenital cases. Esquirol, however, gives the case of a child between five and ten years old whose *monomania* lay in attempting to destroy both her father and mother. But disordered intellect seldom makes itself obvious till after puberty, when the passions are fully developed. The greatest liability exists at the ages between twenty and thirty; while the statistics of Hanwell make the period from thirty to forty to be the most liable to insanity (THURNAM). (*b*.) *Sex*.—Males are more liable than females to attacks of disordered intellect, in the proportion of 53 per cent. of males to 46 per cent. of females; and a larger proportion among the unmarried than among the married population (THURNAM). (*c*.) *Hereditary Origin*.—The testimony of almost universal experience establishes the fact of a very general hereditary transmission of insanity, varying from 26 to 69 per cent. This is remarkably instanced among the high nobility of countries who intermarry, and are allied by blood to each other. The form of disordered intellect generally transmitted appears to be *dementia;* the mother's intellectual disorder being chiefly transmitted to daughters, and that of the father's to sons. Legislative enactments regarding the intermarriage of persons tainted by disordered intellect are greatly to be desired; and the concealment of such disorder, with a view to marriage, ought to render marriages null and void which are concluded under such circumstances. *Melancholia* is often hereditary; and all its varieties are disposed to be remittent. (*d.*) Certain diseases are powerful predisposing, or even exciting causes of disordered intellect, such as *epilepsy*, which gives rise to a large number of the most incurable cases. Derangement of function or structure of the uterus is another powerful predisposing cause. Many persons also become deranged after severe fevers or attacks of "*sunstroke*," in climates like India. Dyspepsia is also a frequent forerunner of intellectual disorder. Constipation, long-continued and habitual, associated with solicitude and family cares in persons formerly healthy, and of excitable and lively dispositions, appear in some cases to have acted as an exciting cause of *melancholia* (Dr. W. H. O. SANKEY, *Medical Times and Gazette*, Sept. 19, 1863).

(*c.*) *Morbid Anatomy.*—The brain is an organ of such exquisite delicacy, both of structure and of function, that important and extensive lesions may, and often do exist in it, which neither our naked eyesight nor our touch can appreciate, and which can only be demonstrated by various complex methods of research.* In those cases where the manifestations of the mental phenomena are simply disordered and perverted, but not abolished, it is consistent with the pathology of disease in other parts to expect *the very slightest* structural change in the brain—analogous to the local morbid state which attends *inflammation*, and to that form of it to which Virchow gives the name of *parenchymatous*, appreciated by the microscope as a mere "cloudy swelling" of the minute elements of tissue. The phenomena of disorders of the intellect also furnish most conclusive data which show that the brain-tissue is in paired, sometimes by *irritative* and sometimes by *destructive* lesions. Dr. Batty Tuke has given the result of the examination of eighty brains of persons who had been the subjects of chronic insanity. The most common lesion has been fatty degeneration and atrophy of the nerve-cells, commencing at the periphery and extending inwards. The cell is represented by a mass of débris, and in extreme cases of senile mania, by masses of fatty globules, which become diffused throughout the tissue. The neuroglia is increased.

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* I am indebted to Dr. Allen Thomson for the following account of a new and greatly improved process for the preservation of the brain and nerves in the moist condition, which has lately been described by Professor Giacomini of Turin. This process consists essentially in a preliminary induration of the brain by means of a saturated solution of chloride of zinc (or other reagents), a subsequent immersion in alcohol, and, lastly, impregnation with glycerine. The brain prepared in this way retains much of its natural appearance, and is only tougher and somewhat harder than a fresh brain. The distinctive appearances of the white and grey substances are retained, and though the minuter histological appearances are not sufficiently preserved for microscopic examination, yet for all the purposes of macroscopic observation and dissection the brain so preserved is quite as useful as one recently extracted from the body, or one successfully preserved in alcohol. From the peculiar hygroscopic properties of the glycerine, these brains do not dry, and in fact in the ordinary atmospheric conditions undergo little or no change, though kept for years. They lose only a small part of their weight by the preserving process, and they may be most conveniently kept under cover in series as subjects of observation and study of the outward form or internal structure, or as records of the effects of pathological lesion or physiological experiment. When the specimens are to be handled, Professor Giacomini recommends their being covered with one or more layers of a caoutchouc or marine glue varnish, which, however, it may be added does not alter materially the external appearance of the specimens.

Dr. H. Major has described the changes in the grey matter of the convolutions of the brains of the insane. He found that the corpuscles in the outer layers of the convolutions, immediately beneath the pia mater, increased in number, and that the neuroglia had lost its delicate homogeneous appearance. Increased germination generally had taken place; and in the deeper layer the large nerve-cells were comparatively few, and did not form distinct rows, as in the healthy brain. Some had disappeared, and those remaining had lost their sharpness of outline, and were altered in shape. The vessels were dilated, thickened, and tortuous (*West Riding Lunatic Asylum Reports*, Vols. II. and III.). Schroeder Van der Kolk does not remember to have performed, during twenty-five years, the dissection of an insane person which did not afford a satisfactory explanation of the phenomena observed during life. There is, therefore, good reason for the belief that no mental aberration can exist without some positive lesion of brain-substance, be it temporary or permanent. Drs. Tuke, Rutherford, and Skae, describe a special lesion seen by them in the brain of an insane person, of which they have given a description, with drawings, in the *Edinburgh Medical Journal* for September, 1868. It consists of miliary spots, or areas of tissue, in the white matter of the corpus striatum, in that of the cerebrum and cerebellum, the pons and medulla oblongata, surrounded by well defined fibrous-looking walls. These spots occur chiefly in the white tissue, a single spot only having been seen in the grey. The first stage of this lesion seems to be the appearance of somewhat opaque, white, molecular material, which gives rise to a *cloudiness* of the tissue. It does not seem to have any close relation to blood-vessels, as it pushes them aside; and there was no evidence of germination of the nuclei of the *neuroglia*, or of the vascular walls. The molecular material, while it leaves the fibres of the *neuroglia* intact, takes the place of its nuclei and of the nerve-tubes. The molecular matter of the spots seems to be the result of active growth taking place either within or in the periphery of the tissue. The lesion is the result of a growth which leads to the formation of molecular material, which, by encroachment, causes the disappearance of nerve-tissue; and it differs from that lesion which Rindfleisch has described as the essential change in grey degeneration.

The further testimony from *morbid anatomy* which illustrates the pathology of *disorders of the intellect*, has resulted from observations made upon the *bulk* of the brain, upon its *absolute* and *specific* weight, and upon the relative bulk and weight of the grey and white substance of which it is composed. Such observations show generally—(1.) That the absolute weight of the brain is slightly increased in the insane—a conclusion which is also consistent with the fact, established especially by Bucknill, that, in many cases of disorderly intellect, the absolute *size* of the brain is materially diminished relatively to the capacity of the cranium. (2.) This increase in absolute weight appears to depend chiefly on an increase in the weight of the *cerebellum* compared with the *pons Varolii*, the *medulla oblongata*, and the *cerebrum*. The general result is, that the *cerebellum* in the insane is heavier in relation to the *cerebrum* than it is in the sane. (3.) Almost invariably the weight of the left cerebral hemisphere exceeds that of the right by at least the eighth of an ounce (Dr. BOYD, Royal Society, Feb. 28, 1861; also *Med.-Chir. Trans.*, Vol. XXXIX.) (4.) On arranging the weight of the brain according to the form of insanity under which the patients laboured, it has been found that the absolute weight is greatest in *mania*, and least in the *general paralysis of the insane;* while the cerebellum decreases similarly in weight through the same series, with the exception that, in the *general paralysis of the insane*, it presents the highest average. Generally, it appears that in cases of *acute mania* there is the smallest amount of increase in the relative weight of the cerebellum; while in *general paralysis*—a disease of more prolonged duration—the greatest increase takes place. (5.) Observations on the specific gravity both of the grey and white matters of the brain show an increase in the *insane* compared with the same textures in the *sane* (SANKEY, SKAE). (6.) The *mode of death* has also an influence upon the specific gravity; and, generally, it may be stated that when cerebral symptoms are well marked, such as by convulsions, and when the case terminates by *coma* or by *apnœa*, the specific gravity is higher than when the symptoms are associated with exhausting disease, such as phthisis, and when the case terminates fatally by *anœmia* or *asthenia.* The high specific gravity of the grey and white matter in the former class of cases averaged 1·041, while the average specific gravity of the whole brain in similar cases varied from 1·040 to 1·052; the average specific gravity of the whole healthy brain being only 1·036 (BUCKNILL). (7.) The most essential change consists in shrinking of the substance of the brain, with degeneration of the nerve-cells, or a relative atrophy of its substance by a deposit of inert matter (BUCKNILL, *Med.-Chir. Review*, Jan., 1855). (8.) On comparing the specific gravity of the grey substance in the different forms of mental disease, the lowest appears to occur in cases of *dementia*, where, however, it is 0·003 above the average in the *sane.* The next highest specific gravity occurs in cases of *melancholia*, the next in *general paralysis*, the next in *mania*, and the highest in *epilepsy.* In some of these, however, and probably in all, the *mode of death* appears to influence the specific gravity. For instance, in the *paralysis of the insane* terminating by *coma* or *apnœa*, the specific gravity of the whole brain has been 1·040; while in similar cases terminating by *syncope* or *asthenia*, the specific gravity of the whole brain has not exceeded the average, nor gone beyond 1·039. The average specific gravity of the white substance is lowest in cases of *mania*, next in *dementia*, higher in *general paralysis*, higher still in *monomania*, and highest in *epilepsy* (*Edin. Monthly Journ. of Med. Science*, October, 1854).

The general results of the more crude examinations of the cranium and its contents in cases of insanity, in this and other countries, show that, in a very large proportion of cases, there are found some degree of thickening and opacity of the arachnoid, serous effusions into the subarachnoid tissue, into the arachnoid sac and ventricles of the brain, or of the serum with lymph more or less gelatiniform, accompanied sometimes with increased, sometimes with diminished vascularity of the brain and its membranes, thickening and adhesion of *dura mater*, gritty state of the *pia mater*, turgescence of cerebral vessels, or of *puncta vasculosa.* Lesions are also more frequently found at the anterior and superior portions than at the base of the brain. Some cases prove fatal by a series of apoplectic seizures when the blood-vessels are extremely atheromatous. In that form of paralysis erroneously supposed to be peculiar to the insane, there also appears to exist a peculiar softening of the grey matter, not indicated by any change appreciable to the eye, but by layers of the grey matter stripping off easily with the membranes, to which it often adheres; by the readiness with which the grey matter is broken up by a stream of water; and by changes in the contents of the brain-cells, as observed microscopically (see p. 342, *ante*). The morbid state may extend to a considerable depth into the grey matter composing the hemispherical ganglia; but the whole of these ganglia are generally more or less implicated, in conjunction with the tubular fibres passing from the hemispheres through the vesicular neurine. Dr. Lindsay has noticed œdematous softening of the central parts of the brain in certain of the cases he has examined. Such changes are generally associated with great vital and nervous depression. In a valuable paper "On the Blood in the Insane," Dr. Lindsay has shown that, although the blood is in some cases characterised by particular morbid states, such states are nevertheless not peculiar to insanity; and save in extreme and exceptional cases, there are no specific abnormalities of the *urine, blood, post-mortem appearances, craniological measurements*, or of *facial characteristics* in the *insane* as compared with the *sane.* There is evidence to show, however, that while the whole morbid condition of the blood in disorders of the intellect is not peculiar to such disorders, the general fact exists, that there is a diminished quantity of fibrine. A discrasial condition in the more severe and hopeless cases of mania seems evidenced by the peculiar blood tumors of the ear, so often seen in that form of disorder of the intellect.

Symptoms and Forms of Disorders of the Intellect.—No unobjectionable classification of cases of insanity can be propounded; and "a very large proportion—perhaps a majority—of hospital cases of insanity cannot be referred *simpliciter* to the heading of any *nosology.*" Changes of type in the disease are also not uncommon; such, for example, that a woman, previously suffering from *melancholia*, may become *erotomaniacal* or *maniacal;* and *dementia* is a frequent sequel of all the other forms of intellectual disorder. It is by no means unusual to find the same case of disordered intellect, at different stages of its progress, presenting the characters successively of *mania, monomania, melancholia,* and *dementia.* The differences between *forms* of intellectual disorder cannot therefore be regarded as *essentially* very great; and by no means sufficient to warrant their being regarded as distinct diseases. The existing manifestation of disorder may be very partial, or very extensive, but the disease is the same.

Besides the simple classification given by the College of Physicians, the following classification and synopsis of the dominant symptoms, principal phases, or general conditions of disorders of the intellect, by Dr. Lauder Lindsay, may be useful to the student, as being at once comprehensive and general, accurate (so far as it goes) and practical. It makes no pretence to rigid philosophical or scientific exactitude, or to exhaustive completeness—an exactitude and completeness which its author believes to be unattainable. But it may claim to be more simple and less technical, and hence more *likely to be easily remembered*, than the majority of the many and varied classifications or nosologies of insanity that have been offered during the last half century:—

I. Insanity may be—(*a.*) *General* [Examples, Mania, Dementia,]; or, (*b.*) *Partial* [Example, Monomania]. It may—*apparently*, though perhaps not *really*—affect (*a.*) *All* the faculties or powers of the mind [Examples, Mania, Dementia]; (*b.*) or any *one* or *two* of their groups, viz.:—1. Intellect proper [Example, Delusional Insanity]; 2. Moral sentiments [Example, Moral Insanity]; 3. Propensities or passions [Examples, Dipsomania, Kleptomania, Erotomania].

II. It may be—(*a.*) *Congenital* [Example, Idiocy]; or, (*b.*) *Acquired* [subsequent to birth and generally to puberty].

III. Its characteristic may be—(*a.*) *Excitement* [Example, Acute Mania]; (*b.*) *Depression* [Example, Melancholia]; (*c.*) *Neither excitement nor depression* —No departure from normal character or conduct [Examples, Some cases of Delusional and Moral Insanity].

IV. It may or may not be accompanied by *Delusions*, which may be either—(*a.*) *Partial and uniform*, comparatively [Example, Monomania]; or, (*b.*) *Numerous and variable* [Examples, Some cases of Delusional Insanity.

V. The following *Propensities* or tendencies may be exhibited singly or in combination:—(*a.*) *Destructiveness*—1. *To life*—Suicide, homicide; 2. *To property*—Clothing, furniture, glass, &c. [Example, Pyromania]; (*b.*) *Alcoholic intoxication* [Example, Dipsomania]; (*c.*) *Theft* [Example, Kleptomania]; (*d.*) *Sexual aberration* [Example, Erotomania];* (*e.*) Abstinence from food.

VI. Insanity may be—(*a.*) *Simple*, or, (*b.*) *Complicated* [with other diseases]. The chief complications are—1. *Epilepsy;* 2. *Paralysis*, which may be—(*a.*) *Partial;* or, (*b.*) *General* [Example, General Paralysis or Paresis]. 3. Various—(*a.*) *Functional*, or, (*b.*) *Organic* affections of the—(1.) *Lungs* [Example, Phthisis]; (2.) *Stomach* and intestines [Example, Dyspepsia]; (3.) *Uterus*, &c.

VII. Each of the main forms of insanity—(*a.*) Mania, (*b.*) Melancholia, (*c.*) Dementia, (*d.*) Amentia—may be developed in *different degrees of intensity* [Example, Dementia is represented by mere facility of character —by silliness, imbecility, or weak-mindedness of every grade, onwards to utter fatuity].

These are, in fact, but some of the varieties described in text-books on insanity. In reality they are but symptoms, and often but passing symptoms, in the course of one continuous disease. Many of the phenomena are not only transient, but succeed each other in the progress of a single case. They denote rather the phenomena or stages of one disease, and not separate diseases. A case of ordinary insanity at its outset commences by a stage of melancholy, considered at first acute. It may terminate in one of four ways:—(1.) In recovery; (2.) in death; (3.) by passing into mania; (4.) by becoming chronic (*chronic mania* or *melancholia*) (W. H. O. SANKEY, l. c.)

I. MANIA has its origin in disordered emotions, and is essentially a disorder of the impulses or propensities in the first instance, tending to more or less "*disorder of the intellect with excitement.*" One or more of the passions is almost always exalted. There are two forms—namely, *acute* and *chronic mania.* Furious expressions of passion, of prolonged duration, are very generally present in the acute form. It has in almost all instances its stages of incubation. At first there may be only apparently trifling irregularities in the affections. The maniac may be at the outset of his disease either sad or gay, active or indolent, indifferent or eager, but he soon becomes impatient and irritable. He neglects his family, forsakes his business and household affairs, deserts his home, and yields himself to acts which strikingly contrast with his ordinary mode of life. Periods of composure and agitation succeed each other, and so do acts the most strange and extravagant. The kindest love and tenderness of domestic life serve but to irritate and provoke, so that to remain in the bosom of his family excites the patient by slow degrees to the highest pitch of fury. It is seldom in *mania*, as in *monomania*, that the patient is insane on one subject only. His mind is a perfect chaos; all is violence, effort, perturbation, and disorder. He confounds time and space, associates persons and things the most unnatural, creates images the most unreal, and lives isolated in feelings and reasoning from all the rest of the world. He hates all whom he was wont to love, and wishes to overthrow and to destroy everything. The female maniac, perhaps in health the model of candour and virtue, gentle and modest, an affectionate daughter, a devoted wife, and a good mother, becomes in this disease bold and furious, exposes her person unmoved to the gaze of every eye, is blasphemous and obscene, respects no law either of decency or humanity, threatens her father, strikes her husband, or perhaps murders her children. In ordinary mania the premonitory symptoms are characterised by gloom and despondency, upon which the maniacal excitement supervenes. There is generally a marked departure from the patient's former state of health. *Insomnia* is one of the most important and earliest symptoms. The functions of the body are more or less deranged, and fever may prevail, sometimes severe, with a hot, dry skin; brown, parched, and dry tongue, and teeth covered with sordes. There is an absolute refusal of solid food. The patients fall off in condition. The bowels are generally confined, and the stools offensive. The pulse varies from 80° to 90°, and is diminished in volume.

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* *Satyrasis* and *nymphomania* ought to be distinguished from *erotomania*—the former being an insatiable desire in man to have frequent connection with females, and with the physical power of doing so without exhaustion, with constant erection and desire for venery. It corresponds with *nymphomania* in the female.

Erotomania is a disorder of the intellect (love melancholy), associated with, if not caused by, the more refined passion of love.

Special or partial forms of mania may be noticed, namely—(1.) *Suicidal* and *homicidal mania,* which imply a tendency to act under insane impulses, in some cases the result apparently of delusions of suspicion, or of implacable enmities against supposed foes. A plausible reason is generally assigned for the attempt. Such impulse and attempts of the most persistent and dangerous kind may co-exist with a perfect knowledge of right and wrong, and their bearings on human actions—with perfect ability also to manage business affairs, though of a complex pecuniary character—with perfect propriety in maintaining most of the relationships, or of discharging most of the social or public duties of life—with deportment often the most polished and gentlemanly, the most considerate and kind (Dr. LINDSAY, *Rep.*, XXXVII., p. 42). In a large number of cases the suicidal propensity is developed in connection with (2.)

Religious melancholia—a form most difficult to eradicate or conquer; and from its inveteracy, the forerunner often of incurable forms of disordered intellect. (3.) Arson is one of the most common forms of mania (*pyromania*); and (4.) *Kleptomania* is an irresistible desire to steal. (5.) *Recurrent mania* is characterised by attacks of insanity coming on without obvious cause, followed by depression and recovery (transitory mania), the period of continuance being quite uncertain. The excitement may last for a week, a month, or longer, and may be as violent as in acute mania. After a short period of depression recovery may take place, more or less complete; and the patient may not have another attack for years; but these are most unsafe cases to be at large. (6.) *Monomania* comprehends various forms or phases of intellectual disorder, attended with *delusions*. Some one passion or idea so entirely possesses the patient as often to lead to dangerous conduct. The modes by which the monomaniac gives expression to his mental affliction are endless; and they are especially indicated by *delusions* of endless variety. Some prevailing morbid sentiment lies at their root, and precedes formal delusion,—*e.g.*, exaltation of feelings, suspicion. When accompanied by hallucinations of hearing it is an intractable form of insanity. "Voices" are dangerous hallucinations. As time passes additional delusions spring up. The morbid circle widens, till the intellect is entirely involved. Incoherence comes on, and the mental state passes into incurable dementia. The general health is usually good. (7.) *Puerperal mania* is a form from which the pregnant female suffers at the period of her confinement, and which may repeat itself at each period of parturition. It usually occurs within two or three weeks after delivery; and the fourth or fifth day is the most common time for the appearance of the symptoms. These are of an acute type; and the condition may be that of delirious mania—one feature of the psychical state being that the language used by the patient is often filthy and obscene. Early in the disease both the lochia and mammary secretion are arrested, and the urine may be albuminous. (8.) *Epileptic insanity* is a mental disorder induced by continued epilepsy, most commonly the result of the *petit mal*, where fits are frequent and of short duration. Increasing weakness of memory and irritability of temper are early indications of the impairment of mind. In the course of time, however, the mind is a complete wreck. An acute attack of mania may occur with an impulsive and sudden disposition to violence to any one in the proximity of the patient, murder, or suicide. (9.) *Climacteric insanity* ought to be recognised as apt to occur in females at the age when the catamenia cease to flow; when at each recurrence of the monthly period corresponding to the time at which the catamenia would have appeared, subacute meningeal inflammations may supervene, with a highly congested condition of the blood-vessels (Dr. ORANGE, l. c.). (10.) *Chronic Mania* becomes established when, the acute stage having passed away, delusions become more definitively formed, incoherence is still considerable but less; and the excitement is not so constant, but is more paroxysmal in character. Illusions and hallucinations, especially of hearing, are commonly present; and others of the special senses may be also involved. By slow degrees the mind becomes more and more enfeebled, till at length its powers are irretrievably lost in the ruin of complete dementia (Dr. A. ROBERTSON).

II. MELANCHOLIA stands next in importance to mania as a leading symptom of insanity. It is often the first indication of mental disease. It may precede *mania*, and be associated with or supervene upon other forms of disorder of the intellect. It may supervene suddenly, as when it is the immediate consequence of grief; or it may come on gradually, as the mere exaggeration of a naturally melancholy frame of mind without delusion; or it may be the outcome of some visceral or constitutional disease. Premonitory symptoms generally indicate a period of incubation more or less prolonged, and sufficiently obvious. A state of depression often follows upon a state of *mental elation*, or on prolonged mental exertion and occupation, which suddenly ceases. It also succeeds the mental exaltation produced by inebriating drinks. These effects, however, are generally slight and transient, and the cases of this kind ought to be separated from those cases in which the depression becomes persistent with delusions. In *persistent melancholia* the "relish for existence" becomes less and less, the spirits become depressed, and the man feels unequal to the ordinary duties which call him into public life. In the domestic circle he becomes silent, and seeks entire solitude (TUKE, *op. cit.*, p. 148). His propensities are to indolence and general indifference. He reads nothing, writes to nobody, shuns all exertion. One dominant propensity alone is too often active—namely, self-destruction; while obstinate abstinence from food and drink is a common feature. All cases of melancholia must be regarded as having a suicidal tendency, whether or not there have been threats or attempts indicating it. Every such patient must be looked on as suicidal (CONOLLY, BLANDFORD, MILLAR, BRUSHFIELD). There is greatly increased susceptibility of the emotions. The subjects of *melancholia*, in its early stage, are easily moved to tears by trivial circumstances. All consolation is disregarded. Nothing can subdue their will, convince them of their error, quiet their alarms, or allay their fears; remove their prejudices, overcome their repugnances, or conciliate their aversions. Nothing can divert them from the engrossing thoughts that occupy their mental energies, and take possession of their emotions. Occasional remissions of the affection may deceive the patient's friends for a time; but the disease progresses till the patient is either placed under the care of competent guardians, or he voluntarily seeks the tranquillity of an asylum. Fortunately for the chances of cure, a love for and appreciation of the ridiculous is often associated with the tendency to *melancholia*. Cowper, who penned "John Gilpin," is a familiar example. Carlini, a French comic actor of reputation, consulted a physician to whom he was unknown, on account of attacks of profound *melancholia* to which he was subject. Amongst other remedies, the doctor recommended the diversion of the Italian comedy. "Your distemper," said he, "must be rooted indeed if the acting of the *lively Carlini* does not remove it." "Alas!" said the miserable patient, "I am the *very Carlini* whom you recommen me to see; and while I am capable of filling Paris with mirth an laughter, I am myself the dejected victim of melancholy and chagri (PREFECT'S *Annals of Insanity*, p. 404, quoted by Dr. TUKE, *op. cit.*, 150). The ultimate course of cases of *melancholia* varies with the constitu tion of the patient, his age, the degree in which the disorder is complicated, and the existence of hereditary predisposition to mental disease. It tends to pass into *dementia;* but less marked than in *mania.* It is to be distinguished from grief or simple depression—(1.) by being disproportionate in degree or duration to the grief or calamity with which it is connected; (2.) by its intractability and independence of external influences; (3.) by its periodic exacerbations and remissions; (4.) by the impairment of voluntary power which attends it—a paralysis of will; (5.) by irritability of temper; (6.) by morbid impulses; (7.) the attitude is characteristic. The head is bowed on the chest; answers are given to questions with effort and in monosyllables, or after a considerable pause. The patient is apathetic, taciturn, or absolutely silent.

The special forms which *melancholia* sometimes assumes are—(1.) *Religious*—in which the impress is given to its character by the religious tendencies of the patient, the exciting cause being not rarely traceable to the fiery denunciations of well-meaning but injudicious clergymen, whose great power in the pulpit is mainly attributable to the excitement and alarm they are able to produce upon too susceptible listeners. They delight, metaphorically, to suspend such persons over a bottomless pit, in order that they may drag them up again when they fancy they have sufficiently impressed them with most unchristian terror. Such preachers were wont to be banished from the country, even in Pagan times, by a law of Marcus Aurelius. (2.) *Hypochondriasis,* in which the morbid mental state is expressed by *exaggeration, or increase to a morbid degree of intensity,* of that property which every one possesses more or less of creating around him, or within himself, sensations which are not the result of external impressions or corporeal conditions, but which, having their origin in the mind (subjective), are represented and appreciated by the material organs of the body. It consists essentially in the transference of a phenomenon (subjective or mental in its origin and essence) into what appears to be a real material change, appreciable sometimes by others (REYNOLDS). It is often expressed by the sense of touch, combined with a morbid imagining, so that the patient believes himself to be strangely metamorphosed, changed into some inanimate thing, or he loses all knowledge of his personal identity. This form of disease is sometimes combined with other *delusions.* More commonly the affections are subverted, and those who ought to be most dear to the patient by the ties of relationship become most hateful. The mind is most commonly swayed by some destructive passion to effect some object criminal in itself. *Delusions* are neither necessary nor essential symptoms. *Hypochondriasis* is often one of the worst concomitants or sequelæ of dyspepsia (WATSON); and when disorders of the intellect assume the form of *hypochondriasis,* and are at the same time associated with real lesions of the body, the complication is often very embarrassing to the physician, from the distorted, exaggerated, absurd, and false statements of the patient. The imaginary troubles that affect the patient become actual delusions, out of which he cannot be reasoned. Such patients, however, seldom attempt suicide, and are never weary of seeking fresh medical advice, and of trying new treatment. It is only when delusions arise that hypochondriasis becomes insanity, as when such hallucinations exist as that the heart is standing still, or that a limb is withered, or that the body is putrefying. They really believe they can feel that the heart does not beat, that the skin is dried up, or that they can smell the putrid emanations from their body. In spite of such mental aberration and morbid fancies, most hypochondriacs are able to transact their business, and to take care of their house and family. This is one reason why hypochondriasis usually has not been regarded as a disorder of the intellect, being looked upon rather as a nervous affection. But in the worst forms the patient loses all interest for matters which do not bear upon the state of his health. He becomes abstracted, forgetful, and negligent of his affairs, gives himself no further concern about his family, and often remains idle in his bed for years. It is often a long time before his nutritive condition begins to suffer. Gradually, however, especially in bad cases, the patient grows thin, and acquires a sickly appearance, and derangement of the secretory and digestive functions arises. (3.) *Nostalgic Melancholia* (*nostalgia*) makes itself obvious by an inordinate desire to return to one's native country when far away from home, to which is added the apprehension, on the part of the patient, that he may never be able to return. The prophecy of the inspired writer seems ever ringing in his ears,—"Weep ye not for the dead, neither bemoan him; but weep sore for him who goeth away: for he shall return no more, nor see his native country" (Jeremiah xxii. 10). Army surgeons often witness such cases of home-sickness. The mental faculties in nostalgic patients are the first to undergo a change (LORREY). Decided aberration of mind is expressed by exaltation of imagination, especially in extravagant delusions respecting their homes, accompanied by increased heat of the head and acceleration of the pulse. There is redness of the conjunctivæ, and unusual movements of the patient are frequently observed. Uncertain pains occur in various parts of the body, and the bowels are constipated. There is a general feeling of oppression and weariness; an inability to fix the attention, and conversation is unconnected. A sense of weight and pain pervades the viscera. There are also sometimes partial paralysis of the stomach and diaphragm, and symptoms of *gastritis* or *gastro-enteritis* supervene. Under these circumstances prostration of strength ultimately becomes extreme, mental depression keeps pace with the decline of the body, the patient lies weeping, sighing, or groaning, and a propensity to suicide is not unfrequent when the debility becomes extreme. General paralysis is common; but death is the result of a gradual exhaustion of the vital powers (TUKE, *op. cit.*, p. 156). The Dutch, the Swiss troops, the Highlanders, and the Irish, are those soldiers amongst whom this form of insanity has been mostly noticed; and the disorder is apt to be prevalent during the extreme height of the barometer.

III. DEMENTIA is "a disorder of the intellect characterised by absolute loss or feebleness of the mental faculties," ranging from failing memory and slight confusion of thought onwards to utter fatuity. Its varieties are, *acute* and *chronic dementia*. Acute dementia occurs in the young, more frequently in women than in men, and is prone to affect the naturally feeble-minded and excitable, especially when there is a hereditary tendency. The direct cause is usually a severe mental shock, such as fright, and its occurrence is sudden and soon after the shock. In its *chronic form* dementia is usually the sequel to other forms of insanity, as already mentioned. It is characterised by dulness of apprehension, weakness of memory, inability to follow out any subject of thought, or by general silliness of language and conduct. In its extreme state there is inability to comprehend the simplest questions, and the patients require all their wants to be attended to like an infant. *Senile dementia* is characterised by failure of memory respecting recent events, while the long past may be remembered with accuracy. Ideas become incoherent. Delusions arise which are seldom persistent, but which, while they last, may incite to violence; and maniacal excitement may sometimes last for days. There is feebleness of purpose, with facility of disposition; and indecency of conduct may be manifest (Dr. ALEXANDER ROBERTSON). Dements may become paralytic, and a thickness of speech may be the first symptom of its approach. After a time the speech is more manifestly affected, followed by a loss of power in the limbs of one side, more marked in the lower extremity, so that the step is feeble and straggling. In the last stage they are completely paralytic, and only able to utter a few unintelligible sounds.

IV. IDIOCY (*Congenital*) arises from an abortive condition of the intellect from defective development of the brain. Subjective phenomena are not capable of being exhibited, and therefore cannot be inquired into (ESQUIROL, COKE, PRICHARD, BUCKNILL, TUKE). The characters of the idiot vary according to the degree in which the cerebro-spinal system is involved (TUKE). In the lower, or most abject forms, the functions of *organic* or *vegetative* life are ill-performed; nutrition is imperfect, the power of reproduction small, and the patient would perish were it not for the assistance of others. The functions of animal life are likewise impaired, and he may be scarcely alive to external impressions, nor possess the power of executing spontaneous acts. The abject condition of the idiot is obvious in his vacant stare, the thick everted lips, the slavering mouth, the irregular teeth, the gums often swollen, the frequent strabismus, the general want of symmetry, the absence or defect of the senses of sight, hearing and speech, taste and smell. Notwithstanding the staggering gait, he is constantly moving, if on his feet; and if seated, he has a difficulty in balancing himself (TUKE, *loc. cit.*).

Diagnosis.—The diagnosis of insanity requires consideration from two points of view—namely, (1.) From its medico-psychological aspects; (2.) from its medico-legal aspects. These aspects, unfortunately, do not at present coincide.

I. *The Medico-psychological Diagnosis of Insanity.*—Premonitory indications of insanity are to be traced in *morbid alterations of temper*, *continued depression of spirits, amounting sometimes to simple melancholia; headache, severe giddiness, inaptitude for business, loss of memory, confusion of mind, defective power of mental concentration, the feeling of brain lassitude and fatigue, excessive ennui, a longing for death, a want of interest in pursuits that formerly were a source of gratification and pleasure, restlessness by day and sleeplessness by night.* Any one or more of these symptoms indicate an unhealthy state of the functions of the brain; but their insidious mode of approach, and the unwillingness of friends to believe that anything is wrong with a relative, rarely if ever permit the symptoms to attract attention till some form of insanity becomes unmistakably developed.

If a person previously in a state of bodily and mental health is conscious that changes are taking place in the working of his mind, such that trifles worry and irritate him, that he feels his brain unfit for work, that his spirits flag, that he tends to magnify all the evils of life; if, moreover, he is observed to be fanciful, imagining things to exist which have no existence apart from himself; if he believes that kind friends ill-use and slight him; if, besides, symptoms like these, or analogous to these, are associated with headache, derangement of the digestive organs, want of continuous sleep, the friends of such a sufferer may rest assured, and the patient may perhaps be convinced, that the state of his brain is abnormal, and he may be induced to commit his case to the consideration of a physician. Symptoms of severe bodily fatigue, associated with extreme depression of spirits, mental exhaustion, reverie, paroxysms of melancholy, partial somnambulism, or hallucinations manifesting themselves at an early period of life, must be regarded as important psychical phenomena. To obtain a correct and early diagnosis, one "*only safe rule*" is to be observed—namely, a close and thorough appreciation of the physical and mental aspects of the existing condition of the presumed lunatic at the period of his supposed insanity, compared with his prior physical and mental manifestations, which were regarded as his natural and healthy state, and which had not been observed to be different from those of other men—"*a comparison of the individual with his former self.*" The necessity of making the mind of the individual the standard of comparison by which to determine sanity or insanity cannot be too strongly urged. The man must be the measure of himself; and this principle is found to be of universal application in all physiological and pathological investigations. For example, before the physician can judge of the condition of the urine passed by a man in disease, he must know the condition of that man's urinary excretions when they are in a normal state. So, also, before the physician can judge as to the impairment or disorder of intellect in a given individual, he ought to acquire or possess some knowledge as to his intellectual condition previously. If mental phenomena are ascertained to exist of a morbid kind, compared with those which have existed before, and especially if there be any characteristic symptom of cerebral disease, the individual may be fairly deemed insane, and irresponsible for his acts. But there is no general and universal test of mental unsoundness; and it is unfortunate that the legal mind continues to assume that such tests do exist. Definite criteria cannot possibly be laid down applicable to every case: and the medical proofs of insanity must vary according to the distinctive features of each (Dr. A. ROBERTSON, *Town's Hospital and Asylum*, Glasgow).

II. *Medico-legal Diagnosis of Insanity.*—That "it takes all kinds of folk to make a world" is a proverbial saying. On this principle the world and its inhabitants may be regarded as embracing—(1.) Sane people; (2.) insane people; and, (3.) folks inhabiting a borderland, whose sanity or insanity is doubtful, and who may become subjects of anxious inquiry in this respect. They furnish the chief and most abundant cases for litigation in our courts of law, while they often contribute fearful legends to the annals of criminal jurisprudence. They are also met with in asylums, or as criminal lunatics in jails, in considerable numbers. Soundness or unsoundness of mind is therefore a diagnosis which the general practitioner is often called upon to make; and to do so he ought to follow imperatively such rules as will lead him to as safe a result as the nature of the individual case will admit of. Sanity or insanity, soundness or unsoundness of mind, is simply a question of fact. But it is a question of fact which must be judged of not as a question of law alone, or of medical science alone, but on the ordinary rules which are applied in daily life, aided by the most advanced knowledge of cerebral disease, and especially regarding the working condition of the brain as the instrument of the mind. The plea of insanity in civil or in criminal courts of law (as a plea of innocence, or as a bar to disposing power), ought to establish the existence of such a degree of mental disorder as excludes the free determination of the will, and which renders the application of the penal code no longer efficacious. It is the duty of the medical man to make such a critical examination of every individual case on its own merits, with the view of ascertaining whether such a degree of mental disorder does or does not exist: and then to place the result of his examination before the court in the plainest and clearest language that will suffice to convey his meaning (Dr. ORANGE, l. c.).*

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* When the plea of insanity is put forward in a criminal court of law, in bar of trial, of sentence, or of punishment, the grounds on which decisions have been arrived at have varied greatly at different times, and the following statements in this note are compiled from the writings of Dr. Alexander Robertson, Physician and Medical Superintendent, Town's Hospital and Asylum, Glasgow; Dr. W. Orange, Medical Superintendent of the Broadmoor State Lunatic Asylum; and other authoritative sources of information, to which references are made.

In capital charges the plea of insanity is not usually admitted in bar of trial, unless the prisoner is very obviously insane—maniacal, or distinctly the subject of dementia—to such an extent that he cannot form a conception of the nature of the charge against him. In no class of criminal trials has "the glorious uncertainty of the law" been more strikingly manifested. Cases seemingly parallel (so far as the medico-psychological evidences of mental unsoundness, or of the irresponsibility of the agents for their acts were concerned) have had opposite results; so that some panels have been found guilty and executed, while with others the plea of insanity has been sustained, and the criminal lunatic has then been consigned to an asylum during Her Majesty's pleasure, usually, but not always, equivalent to confinement for life. Such contradictory conclusions have been mainly due to the differences in "*the tests of insanity*" propounded by the various judges in their charges to the juries, and to the fact that the juries have not always given a verdict in accordance with the legal deliverance of the judge. But of late years, compared with 150 years ago, a great change is apparent in the character of the charges to the juries by the judges. In 1723, Arnold, a lunatic, was tried for shooting at Lord Onslow. The presiding judge in his charge to the jury said:—"It is not every kind of frantic humour, or something unaccountable in a man's actions, that points him out to be such a madman as is exempted from punishment; it must be that a man is totally deprived of his understanding and memory, and doth not know what he is doing, no more than an infant, than a brute, or a wild beast; such a one is never the object of punishment." At that time, therefore, nothing but a total deprivation of reason could save any lunatic from suffering the full penalty of the law for criminal acts. The doctrine of *partial insanity* was not then admitted in any of its various forms in palliation, however clearly it might be evident that the offence was the direct product of delusion, and as much the offspring of disease as though it were the action of a raving maniac (Dr. ALEXANDER ROBERTSON). In charging the jury at the trial of an insane man, in the spring of 1876, for the murder of his wife, the learned judge, Lord Blackburn, is reported to have said that "he had read everything that was written on the subject, and had thought the matter over, and that he never could come to understand the precise amount of insanity that rendered a man unaccountable." The present aspect of the law towards medical evidence in cases where insanity is pleaded has greatly changed of late, and some measures have recently been proposed, having for their object to produce an improvement in the existing state of things. The legal notions of insanity have not been always the same; and in the present century, the chief landmarks indicative of the current of opinion with respect to insanity in criminal cases are—(1.) The trial of Hatfield for shooting at the King, in the year 1800, in the Drury Lane Theatre, when the doctrine of total deprivation of reason was again laid down as requisite and necessary to sustain the plea of insanity. But although urged by the Attorney-General it was not adopted, and the panel was acquitted on the ground of insanity. He was ably and eloquently defended by Mr. Erskine, afterwards Lord Erskine, who showed that the act had been due to delusion, though the derangement was only partial. This advance on the opinions which ruled in Arnold's case was not lasting, as, (2.) The trial of Bellingham for shooting the Prime Minister, Mr. Spencer Perceval, in the lobby of the House of Commons, in 1812, resulted in his conviction and execution for the murder, though the crime was shown to have been the direct result of a delusion. The test of insanity, or criterion of responsibility, submitted to the jury by Chief-Justice Mansfield in this case was, "*the capability of distinguishing between right and wrong*—NOT IN REFERENCE TO THE PARTICULAR ACT, BUT IN GENERAL." Bellingham was thus less fortunate in the counsel who defended him and in the judge who tried him. Had the test been applied to the particular charge on which the prisoner was being tried, he could not have been condemned, as the delusion which prompted the act rendered him unable to distinguish right from wrong in regard to it. (3.) The trial of Macnaughten for shooting Mr. Drummond, mistaking him for Sir Robert Peel, in 1843. The result of this trial caused much excitement throughout the country, and was the means of strongly directing public attention to the condition of the law respecting insanity associated with crime. Macnaughten laboured under a *monomania of suspicion*—a form of partial insanity most difficult to recognise. He fancied that he was being dogged on the streets, and that different persons (of whom Sir Robert Peel was one) had conspired to ruin him. Hence the motive and the murder. The judges were then unanimously of opinion that "the jury ought in all cases to be told that every man should be considered of sane mind until the contrary was proved in evidence; that, before a plea of insanity should be allowed, undoubted evidence ought to be adduced that the accused was of *diseased mind*, and that at the time he committed the act *he was not conscious of right or wrong*." Macnaughten was acquitted on the ground of insanity, notwithstanding the fact that he was not obviously insane, and had transacted business a short time before the deed. The House of Lords, participating in the feeling of alarm which this decision gave rise to throughout the country, and seeing that the law was thus uncertain, propounded to the judges certain questions with regard to the law on insanity when it was alleged as a defence in criminal actions, the object being to obtain from them an exposition of the law for the guidance of future courts. The answers to those questions constitute the law upon the subject at the present time. Lord Justice Bramwell has condensed them thus:—"When a man's state of mind is such that he does not know the nature and quality of the act he is doing—for instance does not know that cutting a man's head off will kill him, like the man who cut off the head of another person in order to see how he looked when he woke; or when his state of mind is such that, although he may know what the result will be, he does not know that it is wrong, then he ought to be acquitted. So, also, if he is labouring under a delusion of such a character that, if the delusion were true, he would be justified in the homicidal act, he would also be entitled to acquittal." He further added—"The common notion that a man may be acquitted merely because he is mad, is erroneous."

The statutes relating to criminal lunatics give nowhere any definition of the term "*insane*." Any definition may be judicial, but is not statutory. And further, the Act passed in the year 1800, which directs the mode in which persons indicted for any offence and found insane are to be dealt with, speaks of the accused person *appearing to the jury to be insane*. The mode of legal procedure is, for the judge to tell the jury that what they have to consider is, not whether the accused person is insane, but whether he is insane according to a certain definition, that definition being the one which was enunciated by the judges in Macnaughten's case. The legal test of insanity was then resolved into, (1.) The question whether the accused at the time he committed the criminal act knew what he was doing; and, (2.) did he know that what he was doing was wrong? In this form the rule is usually laid down by the judge in criminal trials, and it is founded upon the following *dictum* of Chief-Justice Hale:—"Partial insanity is no excuse. This is the condition of very many, especially melancholy persons, who for the most part discover their defect in excessive fears and griefs, and yet are not wholly destitute of reason; and this partial insanity seems not to excuse them in committing any offence, for it is matter capital. It is very difficult to determine the invisible line that divides perfect and partial insanity; but it must rest upon circumstances to be duly weighed and considered by both judge and jury."

With regard to the questions put by the House of Lords to the judges, Sir James Fitzjames Stephen has observed that very eminent judges had greatly doubted the constitutional propriety of putting abstract questions of that kind to the judges, and getting such answers from them; and that he had heard more than one of the most eminent judges on the bench express themselves as being doubtful with regard to that authority on that ground. Lord Lyndhurst, then the Lord Chancellor, in the discussion in the House of Lords, declared that there was no doubt as to the law—that it is clear, distinct, and definite, that a man is not to be held judicially insane, when tried criminally, if he know right from wrong. And in the recent trials of Mr. Watson and Miss Edmunds, the legal *dictum* was laid down by Mr. Justice Byles and by Baron Martin in the usual form. The legal test is, therefore, perfectly definite. It is the metaphysical test of mental capacity founded on a knowledge of right and wrong. But, as science has advanced, many thinking minds have been brought to doubt the sufficiency of this test; and particularly the question has been raised by medical jurists and medical experts in lunacy. In true insanity, the voluntary power to control thought and action, and to regulate conduct by a knowledge of right and wrong, is impaired, limited, or overruled, by insane motives. A lunatic may have the power of distinguishing right from wrong, while he has not the power of choosing right from wrong. The settlement of this question, then, is one rather for lawyers and statesmen, than for medical men in their special capacity. But it is requisite that the medical witness should be fully acquainted with the legal definition or character of that degree of insanity which the judges have decided shall be admitted as evidence of irresponsibility. That this definition of the kind and degree of insanity which confers irresponsibility is defective; that it is not in conformity with the ascertained facts of medical science; and that some modification of it is now required, are opinions which are now largely shared by high legal authorities. In the year 1874, a Bill to amend the law of homicide was introduced into the House of Commons by the late Mr. Russell Gurney, the learned Recorder of London; and one very conspicuous amendment proposed in that Bill had reference to the plea of insanity. Instead of the existing state of the law as expounded by the judges in the House of Lords, it was proposed to enact that homicide should not be deemed criminal if the person by whom it is committed is, at the time when he commits it, prevented by any disease affecting his mind—(*a.*) from knowing the nature of the act done by him; (*b.*) from knowing that it is forbidden by law; (*c.*) from knowing that it is morally wrong; (*d.*) from controlling his own conduct. But that homicide is criminal, although the mind of the person committing it is affected by disease, if such disease do not, in fact, produce some one of the effects aforesaid in reference to the act by which death is caused, or if the inability to control his conduct be not produced exclusively by such disease. The proposed Bill went on to say that, if a person be proved to have been labouring under any insane delusion at the time when he committed homicide, it shall be presumed, unless the contrary appear or is proved, that he did not possess the degree of knowledge or self-control hereinbefore specified. In practice, the question has been raised most often in the case of *homicidal insanity*, and particularly in those instances where there are no actual delusions or no known motive, but where, as in Esquirol's third degree of homicidal mania, the impulse to kill is sudden, instantaneous, unreflecting, and uncontrollable. Homicidal insanity is commonly defined to be a state of partial insanity, accompanied by an impulse to perpetrate murder. It may make its appearance at all ages, even in children. Sometimes the impulse is long felt, but concealed and restrained. Occasionally the act of murder is perpetrated with great deliberation, and apparently with all the marks of sanity; and the perpetrator may conceal himself, hide his weapon, and, like a sane criminal, endeavour to obliterate all traces of the crime. It is exactly in such cases that the insufficiency of Hale's dictum is felt, for it cannot enable us to distinguish the insane homicide from the sane criminal; for if an insane person commit homicide in consequence of his insanity, he is thus amenable, provided at the time he committed the act he knew right from wrong.

Mr. Russell Gurney's Bill was referred to a Select Committee, and was ultimately abandoned. The chief witnesses examined by the Committee were—Lord Justice Bramwell, Lord Blackburn, Mr. Justice Crawford, and Sir James Fitzjames Stephen. The Lord Chief-Justice submitted a statement in writing, but was not examined orally. Lord Justice Bramwell, although approving of the existing law, said, in the course of his examination, that he thought "the law lays down such a definition of madness that nobody is hardly ever really mad enough to be within it." The majority of medical writers agree with this deliberate statement, that the law, as now expounded, lays down such a definition as to render it capable, if applied in its full strictness, of including only a very small percentage of insane persons, and it is well, therefore, to have this matter so boldly and authoritatively stated. Compare with it the evidence of Lord Blackburn, who says,—"We cannot fail to see that there are cases where the person is clearly not responsible, and yet knew right from wrong. . . . In the case of a woman who was tried for wounding a girl with intent to murder. The facts were these. The woman had more than once been insane, the insanity being principally brought on by suckling her child too long; that was the cause that had produced it before. She was living with her husband, and had the charge of this girl, an impotent girl of about fifteen, who lay in bed all day; she was very kind to her, and treated her very well; they were miserably poor, and very much owing to that she continued to nurse her boy till he was nearly two years old; and suddenly, when in this state, she one morning about eleven o'clock went to the child lying there in bed, aged fifteen, and deliberately cut her throat; then she went towards her own child, a girl of five or six years of age, of whom she was exceedingly fond, and the girl hearing a noise, looked up and said, 'What are you doing?' 'I have killed Olivia, and I am going to kill you,' was the answer. The child fortunately, instead of screaming, threw her arms round her mother's neck, and said, 'No; I know you would not hurt your little Mopsy.' The woman dropped the child, went down and told a neighbour what she had done, that she had killed Olivia, and was going to kill Mary, 'but when the darling threw its arms around my neck, I had not the heart to do it.' She clearly knew right from wrong, and knew the character of her act; for some little time after that she talked rationally enough, but before night she was sent to a lunatic asylum raving mad; and, having recovered, she was brought to be tried before me at a subsequent assizes. She did know the quality of her act, and was quite aware of what she had done; but I felt it impossible to say she should be punished. If I had read the definition in Macnaughten's case, and said, 'Do you bring her within that?' the jury would have taken the bit in their own teeth and said, 'Not guilty on the ground of insanity.' I did not do that. I told them that there were exceptional cases, and on that the jury found her not guilty on the ground of insanity, and I think rightly."

Sir James Fitzjames Stephen expressed himself thus:—"Baron Bramwell thinks that the law of England is such that insanity hardly ever, under any circumstances, excuses a man from crime; in fact, in one of his answers he goes so far as to say that he holds the definition of insanity to be logical and correct, but does not believe that anybody was ever mad enough to fall within it. Practically, that comes to the same thing as saying that madness makes no difference as to responsibility. Mr. Justice Blackburn says, on the other hand, that the section drawn in the Bill pretty nearly represents the existing law as it is, and, if it errs, it errs in defect, because it does not take in certain cases which ought to be taken in. He adds that, in a particular case which he had to try, thinking that the existing law was, I suppose, in a very elastic condition, he took upon himself to tell the jury that there were exceptional cases which came under no rule, and that they ought to acquit the woman who was on her trial on the ground of insanity, although no authority could be found for it; and although Baron Bramwell, an equal authority, considers that the woman under such circumstances ought not to be acquitted, as it was perfectly certain she was by law guilty, I do not wish to follow the matter out, because I do not wish to be considered as saying a word implying disrespect to either of those learned judges. But when you find two learned judges of the highest eminence directly contradicting each other on matters of the first importance, matters on which the life and death of persons tried before them might depend, and one of them praising that state of things as a proof of the elasticity of the common law, I can only say that I feel surprised, and cannot agree with that learned judge's praise of its elasticity. Baron Bramwell speaks quite in the opposite sense, and expresses entirely my own opinions on the matter, namely—that it is eminently desirable that you should have definitions, and that those definitions should state plainly what the law is." Where we find so high a legal authority saying of two learned judges that they directly contradict one another on matters of the first importance, it ought, in some measure, to reconcile medical men to being twitted with occasional want of unanimity of opinion amongst themselves. The matter was further discussed by three judges—Judge Maule, Lord Cranworth, and Lord Wensleydale. Judge Maule said:—"If you are satisfied that the prisoner committed this offence, if you are also satisfied that he was insane, and that he did not know right from wrong, he should be acquitted on that ground; but if you are of opinion that at the time he committed the offence he did know right from wrong, he is responsible for his acts, though he may be of weak intellect." Lord Cranworth, then Baron Rolfe, said:—"If a prisoner seeks to excuse himself upon a plea of insanity, it is for him to make it clear that he was insane at the time of committing the offence charged. The onus rests upon him, and the jury must be satisfied that he clearly was insane. If the matter is left in doubt it is their duty to convict him." In another case Baron Parke, afterwards Lord Wensleydale, said:—"The question whether a man is in a state of mind to be responsible would depend on the question whether he at the time knew the nature and the character of the crime he was committing; and this circumstance ought to be taken into consideration, that it is difficult to see how they can establish insanity in a case where there was a total absence of delusion." The Lord Chief-Justice of England, in a letter addressed to the Committee, expresses himself thus:—"As the law as expounded by the judges in the House of Lords now stands, it is only when mental disease produces incapacity to distinguish between right and wrong that immunity from the penal consequences of crime is admitted. The present Bill introduces a new element, the absence of the power of self-control. I concur most cordially in the proposed alteration of the law, having been always strongly of opinion that, as the pathology of insanity abundantly establishes, there are forms of mental disease in which, though the patient is quite aware he is about to do wrong, the will becomes overpowered by the force of irresistible impulse; the power of self-control, when destroyed or suspended by mental disease, becomes, I think, an essential element of responsibility." In Leeds (Autumn Assizes, 1865), Mr. Justice Mellor observed that he thought that the definition of insanity which would excuse from criminal responsibility, as given in Macnaughten's case, hardly went far enough. He agreed with the universal conviction of medical observers, that a man might know that he was doing an act which was wrong, and still might be labouring under such disease of mind as not to be able to restrain his impulse to do that act, and he should, therefore, not be amenable to the criminal law. The opinion of one judge, however, cannot alter the law; and, as lately stated by Mr. Justice Byles, if the law be altered, it must be done by Act of Parliament. Thus, the legislature, on the one hand, enacts that medical men shall be the judges of insanity, for no one else can sign a certificate; on the other, it formulates for them a legal test. Can there be any reason for this but the fact that lawyers and legislators still refuse to acknowledge insanity to be a disease? Who ever heard of a legal test of concussion of the brain? or apoplexy? or epilepsy? But if a patient be suffering from insanity, it is not enough for a medical man to state this to be the fact, but he must further state whether the individual knows right from wrong. It is not, however, with regard to the law in the abstract that the difference of opinion has arisen. If the premises upon which the legal dictum in regard to insanity is based be correct, no conclusion could be more logical or just; but the medical profession who differ from it, hold that the premises upon which it is founded are false, and that, if the abstract idea of the law be "the perfection of reason and common sense," then the legal dictum is inconsistent with the abstract idea of the law. The spirit of the law undoubtedly is that the legal responsibilities of an individual may be abolished, provided that it be proved that he is mentally incapacitated for his duties by either bodily disorder or defect. It is obvious that this proof must rest upon medical testimony; but the line drawn by that dictum will admit of no degree of irresponsibility short of idiocy or raging madness, whilst the advance in modern study of diseases of the brain and aberrations of intellect has revealed to physicians various phases of mind which are incompatible with responsibility. At a meeting of the Medico-Psychological Association, held at the Royal College of Physicians in London, in July, 1864, the following resolution was agreed to—"That so much of the legal test of the mental condition of an alleged criminal lunatic as renders him a responsible agent because he knows the difference between right and wrong is inconsistent with the fact, well known to every member of this meeting, that the power of distinguishing between right and wrong exists frequently among those who are undoubtedly insane, and is often associated with dangerous and uncontrollable delusions." This was also the view entertained by the committee of medical officers of asylums in 1868, and in substance is contained in the memorial which was presented in the name of the whole of that branch of the profession to the Home Secretary. It expresses briefly the conclusions, which are, in truth, beyond doubt, that insanity may exist, and insane and criminal acts may be and are committed by-persons of demonstrably diseased mind and physically diseased brain, who are yet conscious of the difference between right and wrong (*Brit. Med. Journ.*, Jan. 27, 1872). These extracts are sufficient to show that, on the part of some of the most learned judges in England, there exists the conviction that the time has come when a revision of the law relating to insanity, as expounded in the Macnaughten case, is urgently required; and it is now, therefore, a specially opportune time for medical men to contribute as far as they can from their knowledge of the natural history of insanity, in order to secure that the change, when made, shall be such as to bring the law as nearly as may be into harmony with science, and also in harmony with the general opinion of the educated public. What the public require, in order to form their opinion, is knowledge. If the present legal tests are now to be laid aside as antiquated, it becomes an interesting question whether the principle that *inability to control the conduct*, if caused by any disease affecting the

It would be of the greatest importance to have an authoritative understanding as to the precise differences between the legal data on which a person is to be judged insane, and the medical tests of the insane state. It is clear from the contents of the note, that one legal fiction has been practically ignored,—namely, in the recent case of the Rev. J. S. Watson, when the absence of delusion was not dwelt upon by the counsel for the prosecution, nor commented on by the judge as being fatal to the theory of insanity. It was tacitly assumed that insanity might exist without delusions. The other legal fiction is also ceasing to exist in the form in which it was originally enunciated—namely, the knowledge of right and wrong as a test in criminal cases. Some agreement ought now to be come to on the following points:—(1.) As to tests of insanity—(*a.*) that based upon the existence of delusion; (*b.*) that turning upon the knowledge of right and wrong, and of the consequences of actions. (2.) As regards the doctrine of partial insanity, and its responsibilities. (3.) A revision of the distinction between responsibility

mind, ought to carry with it irresponsibility. This appears to be really the essence of the whole matter. It is curious to observe that, although *conduct* is the one thing with which the law is concerned, yet that in this question of insanity the legal tests have hitherto dwelt so entirely upon knowledge and beliefs, and have altogether omitted all mention of *conduct*. The other important modification proposed, has reference to the value of the existence of delusion as a symptom of insanity in the legal sense of the term. The existence of a delusion is a most important element in the diagnosis of insanity; and it is also true that, where a delusion exists, it is almost impossible to affirm the absence of any connection between the delusion and any particular act performed; and it may be added that delusions often exist without being detected. Still it must be admitted that there are certain forms, or rather stages, of insanity in which there are no delusions, or at any rate, none in the ordinary and plain meaning of the word. And, therefore, as delusion, although a most important symptom of insanity, is a symptom which is not invariably present, it is questionable whether it would not be better to omit the clauses relating to it, lest it should come to be thought, upon the principle "*expressio unius est exclusio alterius*," that the proof of insanity would not be complete in the absence of that particular symptom. Another objection is that, in requiring proof of the existence of an insane delusion, instead of simply requiring proof of the existence of insanity, the difficulty is only shifted, and not solved. The question immediately arises, What is an insane delusion? The medical witness, if called upon to assist the court at all by his evidence, should be left entirely unfettered as to the means of diagnosis of insanity which he is to employ; and he should be only required, first, to satisfy his own mind, and then to submit his own opinion to the consideration of the court, supported by such proofs as will bear the test of cross-examination upon the point, whether, when the offence was committed, there did or did not exist in the accused person such mental defect or disease as deprived him of the power of regulating his conduct in accordance with the laws of the land. To tie down the medical witness, by precise legal rules, as to the mode in which he is to form his opinion, is very much like telling a physician to make a diagnosis of lung-disease, and at the same time limiting him to such information as he can gather from the pulse and the tongue, and strictly precluding him from making any use of such instruments as the stethoscope or the thermometer, upon the plea that such things are not mentioned by Hippocrates or Celsus. English lawyers have commonly expressed the opinion, and expressed it strongly, that to leave the question of irresponsibility to be decided by the court in this general manner, without some strict definition to guide it, would be attended with grave inconvenience; but this feeling, perhaps, in some measure, springs from legal tradition, and it is a question whether any inconvenience could be so great as that which attends the existing practice, under which it constantly happens that the jury cut the knot by bringing in a verdict of insanity in opposition to the judge's charge; or the court finds itself not unfrequently compelled first to pass sentence upon a manifest lunatic, and then to invoke the aid of the advisers of the Crown in order to prevent the execution of the sentence (Dr. ORANGE, Address on "The Present Relation of Insanity to the Criminal Law of England," *Brit. Med. Journ.*, Oct. 13, 1877).

for criminal acts and capacity for civil acts. (4.) As to the mode of dealing with those whose mental condition is impaired, but who are not, in the popular sense of the word, "insane." (5.) As to the possibility of dealing with those bordering upon insanity, as at present recognised. (6.) As to the mode to be adopted in dealing with cases, both civil and criminal, when insanity is alleged as a plea of innocence, or as a bar to disposing power. (7.) As to the doing away with the present mode of investigation in a court of law—viz., by the calling of skilled witnesses on different sides. (8.) As to the possibility or desirability of a court or of a commission to report on all cases of impending legislative inquiry in regard of those who are alleged to be insane (*Brit. Med. Journal,* June 29, 1872). Mere depravity is to be carefully distinguished from actual mental disease. The term *moral insanity* is better avoided in a criminal court of law. In almost all forms of insanity (as Esquirol long ago pointed out), the moral faculties become impaired more or less; and, in some cases, the impairment of the moral faculties is disproportionately greater than that of the intellectual faculties. *Moral perversion* also is sometimes the earliest noticeable symptom of the invasion of insanity; and it is often also found, strongly marked, in some members of a family of which other members are actually insane. In forming his diagnosis, the medical witness must give to *moral perversion* its due weight as a symptom. A mere deficiency of moral sense will not warrant a person being certified as insane, even when descended from insane parents (Regina *v.* Edmonds, 1871). Unless connected with such moral perversion there be also found other distinct evidences of mental impairment, it would be better to speak of absence, defect, impairment, or obliteration of the moral faculties, and not to make use of the term "insanity" at all; or that any inability to obey the law arises from defect of judgment or of will, or of self-control, and that therein consists the insanity. As the law now stands, however, the medical witness is not allowed to place his opinions before the court in this way. He is required to answer a certain set of questions, sometimes ingeniously framed to bring out only one side of his opinion of the case. Serious injustice may thus be done if counsel on the opposite side do not return to the subject so imperfectly elucidated. If the case be one of homicide he will probably be asked :—(1.) Whether the accused knew that the act of stabbing or shooting, &c., would produce death? Our knowledge on this point amounts to this, that only when raving, delirium, or utter dementia is present, is there real unconsciousness of the act done. (2.) Whether at the time of committing the act, the person's state of mind was such that he did not know it was wrong to kill? The medical witness must be prepared to give a definite opinion with reference to the application of this test to the particular case under investigation. If applied in its full strictness and according to the plain meaning of the words, the opinion of Lord Justice Bramwell is "that hardly anybody is really mad enough to come within it." On the other hand, legal ingenuity is capable of enlarging the meaning of terms to any amount. By some the knowledge of right and wrong implies also the power of refraining from the act—the power, in fact, of *controlling* conduct. In this respect the witness cannot testify to such a matter of fact. It can only be a matter of inference, a speculation, if he testify that in his belief the power of appreciating the nature of the crime was absent at the time of its commission. (3.) Whether, if he be suffering from a delusion, it is a delusion of such a character that, if it were true, the act of killing would be justifiable? Delusion is a most important symptom of insanity, and it should be searched for with the greatest perseverance and care. But the existing law expressly declares that the existence of any number of delusions is not sufficient to render a person irresponsible unless they are of such a nature that, if true, they would justify the commission of the act—a legal dictum which supposes that an insane man is able to think and act reasonably with respect to his delusions. When a man kills his wife and children under the delusion that they are likely to die from want, because he believes himself to be ruined; even if he were ruined, would that be a justification for killing anybody if he were sane? Over and over again juries have given verdicts of "not guilty on the ground of insanity" in such circumstances without a moment's hesitation, so that juries have acquitted persons manifestly insane, yet whose insanity did not come within the strict legal definitions. "The true value of the existence of delusions as a symptom of insanity is the evidence which they give of a grave defect of the reasoning faculties. If there are many delusions, and if they are of recent origin, they afford evidence that the faculty of judgment is acutely deranged; whereas, if the delusion be limited to one, or to a few closely kindred subjects, and if it be of long standing, it perhaps is possible for a person so afflicted to be capable of exercising fair judgment upon matters not immediately connected with the delusion. Still, wherever it is possible to demonstrate the connection between a known delusion and a criminal act, it is satisfactory to the jury at any rate; and this connection can be found more frequently with time and patience than might be anticipated" (Dr. ORANGE). It is also to be kept in mind that the connection between the delusion and the act may not be made out at the trial of a criminal lunatic, and that the exact bearing of any delusion can often only be ascertained with certainty through the statements of the accused himself; and when, either on account of increasing dementia, or from any other cause, the accused is disinclined to enter into conversation, the point must necessarily remain in the region of conjecture and probability. Hence the absence of direct proof of the connection between any delusion and any insane act ought by no means to be interpreted as equivalent to evidence that no such connection exists.

Insanity must also be distinguished from mere *delirium* (see p. 411, *ante*). It is specially important practically in the early stages of general paralysis (page 342, *ante*), which in the stage of delirium so frequently comes under the notice of the general practitioner.

How are cases, civil and criminal, to be inquired into by the general practitioner:—(1.) As to the existence or not of insanity? (2.) when insanity is alleged as a plea of innocence, or as a bar to disposing power? With a view of drawing up a "statement" on these points, or furnishing a medical certificate of insanity, the following rules have been compiled as a guide, mainly from the following sources:—

namely, the writings of Bucknill and Tuke, Bushnan,[*] Alexander Robertson, Blandford,[†] Millar,[‡] Brushfield.[§] (1.) It is obvious in the interests of truth and justice, that the medical inquirer must use his utmost endeavours to prevent himself becoming a partisan. If the subject of legal inquiry has been a patient well-known to himself, he may naturally become so; but it will be well if he is able to prevent his evidence being led *on mere matters of opinion* by counsel on either side. "Facts observed by himself," and carefully made a note of, are the only elements on which his reputation can be safe in a witness-box. (2.) Every legitimate effort should be made to obtain full information of all facts which may throw light on the mental condition of the individual who is the subject of inquiry. Learn as thoroughly as possible the antecedents and history of the patient. One of the difficulties to overcome in the inquiry, is the risk the physician runs in being misled by the interested statements of friends and relatives. They too frequently act on the principle that "what they wish to be, that they believe." They may wish their relative to be considered sane, or the contrary; and they may believe him to be so when he is not, or the reverse. They may at first disguise, misrepresent, or deny circumstances which might be thought discreditable to the subject of inquiry or themselves. The physician may therefore find himself surrounded by persons from whom he can derive little information which is unbiassed and trustworthy. He may find the household divided against itself. Seeing that such is the case, his best policy is to become a patient listener; and if he has a good memory and quick perception, he will arrive at conclusions the more readily that he avoids all cross-examination. With prudence and caution he ought, then, to seek out people who have known the patient, but who are neither friends nor relatives, but only neighbours and acquaintances, whose evidence will often be more truthful and useful to him. One great aim of such inquiry is to ascertain particulars respecting—(*a.*) conduct and habits of life; (*b.*) causes of excitement; (*c.*) the existence or not of hereditary predisposition, and of previous attacks of insanity. (3.) Copious notes should be taken of the *exact replies to specific questions at the time of the examination:* and it is of importance to record and to quote the exact language and authority. (4.) The inquiries should extend over the whole life, and include what may be called the medical and family history of the subject of inquiry. Ascertain, (*a.*) if a hereditary tendency to insanity or other inheritable nervous affection exists; such as, epilepsy, paralysis, chorea, or habits of intemperance; (*b.*) inquire especially regarding epilepsy; (*c.*) inquire as to the existence of physical signs of general paralysis in its early stage. (5.) *Estimate the value of the hereditary tendency upon the following principles*, that—(*a.*) The insanity of one parent indicates a less degree of predisposition than that of a parent and an uncle; and still less than that of a parent and a grandparent, or of two parents. (*b.*) The insanity of a parent and a grandparent, with an uncle or aunt in the same line, may be held to indicate even stronger predisposition than the insanity of both parents. (*c.*) The insanity of a parent occurring after the birth of a child, without predisposition, is of no value in the formation of a hereditary tendency. (*d.*) If several brothers or sisters, older and younger than the patient, have become insane, the fact tells strongly in favour of predisposition, although neither parent nor grandparent may have been so. (*e.*) The insanity of cousins cannot yet be determined as worth anything in favour of predisposition, except in corroboration of other and weightier facts (BUCKNILL, *op. cit.*, p. 272). (6.) *Ascertain if there has been any change of habits or disposition.* The physician will thus learn what kind of a man, in habits and conduct, the patient has been when in health. (7.) If the medical man is introduced to a person of whom he has no knowledge, and whom he may never have seen before, it is necessary that he should have several interviews (three at least) before he forms his final opinion. This is obvious from the nature and the extent of the inquiries, which the exigencies of the case demand. The first interview ought not to be of shorter duration than one hour. It may even be necessary to visit the subject of inquiry at a subsequent time quite unexpectedly, late in the evening perhaps, or at night. (8.) *Exercise the greatest tact and discretion in the personal examination of probably insane patients, and obtain an introduction in as natural a way as possible.* Avoid commencing any conversation which will tend to divulge the object of the visit. In commencing insanity the patient is generally suspicious, and some contrivance and great tact are required to open up a conversation. It may even be necessary to be a party to some deception; but it must be borne in mind that the discovery of even the slightest deception by the patient may have a most prejudicial influence on future management. As a general rule, it is best to engage the attention, and obtain at least the good-will of the subject of examination by sympathising inquiries respecting bodily ailments, or concerning such things as he knows the patient takes a lively interest in. The first aim of the physician must be to placing himself on good terms with his patient. For this no general directions can be given. He must employ that tact, derived from good sense and knowledge of mankind, without which he will find himself lame and impotent in the field of medical practice amongst the insane. The most difficult cases to be inquired into (*de lunatico inquirendo*) are those in which differences of opinion and of interest exist among the members of the patient's family,—when perhaps the patient has been told by some one of his family that it is wished to prove him insane, and to place him under confinement, and that a doctor is coming to examine him for that purpose. (9.) The examination should be conducted when no one else is present with the patient. The medical inquirer (legally) must separately from any other medical practitioner examine the patient, and form an independent opinion as tothe existence of in sanity (16 and 17 Vic., cap. 96, §§ 4 and 10). Too much caution cannot be exercised in rigidly carrying out this statutory requirement. In a recent case (Novell *v.* Williams) the judge remarked with reference to the two certificates being on the same paper that,—"Anything more calculated to lead to the law being broken I cannot imagine." [This is a mere detail of stationery, which it is the province of the Commissioners in Lunacy to arrange with the Stationery Office for the use of medical men.] The medical men who certify, should not be together in the same room during the examination of a case. They should not see each other's certificate before writing their own, or hold any converse about the case until each has finished his own certificate. (10.) *No question must be asked the subject of inquiry as to whether or not he has committed any specific crime.* (11.) *Study and make a note of the appearance, demeanour, and general conduct of the patient.* (12.) *Notice any peculiarities of bodily condition,* as regards plumpness or emaciation, the state of the skin, the pulse, the tongue, and the temperature; and condition of the eye, as indicating impaired bodily health; and inquire about the *catamenia,* as furnishing a guide to the existence or otherwise of *climacteric insanity.* (13.) *Observe any peculiarities of residence or of dress.* Many circumstances testify to a want of order and direction in household affairs where the head of the family is insane. In the room occupied by the patient things are liable to be out of place, especially as regards the decoration of the walls and the arrangement of the furniture. A love of order is rarely seen among the insane. (14.) *Observe any peculiarities of gesture,* and the expression of the countenance of the patient. (15.) A medical certificate may be declared invalid if it does not specify the name and number of the street where the patient was interviewed. (16.) Every certificate should be an independent and complete document, and no reference should be made in it to any other. (17.) Every medical man must state his reasons in detail for declaring any person to be insane under the two following heads:—(*a.*) *Facts indicating insanity observed by himself* (personal observation); (*b.*) *Other facts indicating insanity communicated to him by others* (hearsay evidence). (18.) He may be called upon to defend all his statements and facts in a court of law. Hence the extreme caution required in all written statements. (19.) All personal facts recorded in a legal certificate must have been observed *on the day of examination:* and a certificate of insanity is not valid unless the symptoms testified to in it are observed by the physician *on the day the examination took place.* (20.) Hearsay facts must be carefully recorded, giving the name of the informant and all other particulars. (21.) The certificate will be invalid if the medical man is unable *from his own personal knowledge* to adduce such facts as will in themselves show the patient to be insane. (22.) One delusion, however well-marked, to the exclusion of others, might run a chance of being disputed as to its tenour and correctness. (23.) All the leading symptoms, and especially dominant delusions, should be stated as tersely as possible. (24.) If a delusion is said to exist, and that a single one, especial care and industry is necessary for its elucidation. The asserted delusion might be true. The nature and character of the delusions must be fully recorded.

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[*] "Hints for certifying in Cases of Lunacy," *Med. Times and Gazette*, August, 1862.

[†] *Insanity and its Treatment*, 2nd Ed., 1877.

[‡] *Hints on Insanity*, 2nd Ed.

[§] "Medical Certificates of Insanity," *Lancet*, Vol. I., 1880.

Those who have to do with insane patients would do well to follow the recommendation of Drs. Brushfield, Robertson, and others, and make a written record of such cases. One can never tell whether or not they may become the subjects of inquiry in a court of law, or when the time may come for them to be consigned to an asylum. Dr. Brushfield emphatically refers to the unsatisfactory nature generally of the medical certificates of insanity. He suggests for adoption the following:—

Ainsi, lorsqu'on entreprend le traitement d'un anévrysme, on ignore, d'une part, quel est le degré de stagnation qu'il faudrait produire dans le sac pour y faire déposer telle ou telle espèce de caillots; on ignore, de plus, quel est le degré d'influence que la ligature exercera sur la rapidité de la circulation anévrysmale. En d'autres termes, on ne sait ni ce qu'il faut faire, ni ce qu'on pourra faire, et c'est dans de pareilles conditions qu'on se décide à entreprendre une des opérations les plus sérieuses de la chirurgie!

Pour sortir de cette fâcheuse incertitude, il faudrait pouvoir graduer l'action de l'obstacle qu'on oppose au cours du sang artériel; il faudrait pouvoir réduire peu à peu l'activité de la circulation anévrysmale, afin de faire naître les conditions favorables à la coagulation fibrineuse, avant d'arriver au degré de stagnation qui fait précipiter les caillots passifs; au lieu d'appliquer une ligature ordinaire, serrée une fois pour toutes, et agissant avec une aveugle brutalité, il faudrait exercer sur l'artère une action progressive. La ligature graduelle, déjà si avantageuse au point de vue de la gangrène, l'est bien plus encore au point de vue de l'oblitération de la tumeur. En tenant compte de ces deux avantages, on arrive à se demander si elle mérite bien le mépris où elle est tombée, et si le bénéfice qu'elle procure sous ce double point de vue ne compense pas largement les chances, très-contestables d'ailleurs, qu'elle ajoute, suivant quelques personnes, en faveur de l'hémorrhagie consécutive. Pour ma part, je pense que ce procédé, ou, pour mieux dire, que cette méthode de ligature a été abandonnée sans motifs suffisants; que les faits, jusqu'ici, lui sont favorables (1); que la théorie lui est plus favorable encore, et qu'on serait pleinement autorisé à en tenter de nouveau l'application. Mais, en attendant que l'expérience ait définitivement fait connaître le degré de gravité de la ligature graduelle, considérée sous le rapport de l'hémorrhagie consécutive, on peut dire qu'elle agit sur les anévrysmes d'une manière irréprochable, et on peut en dire autant de la compression graduelle médiate, ou *compression indirecte,* exercée à travers les téguments sur le tronc de l'artère qui se rend à la tumeur.

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(1) *Voy.* plus haut, p. 495-499.

Revenons maintenant à la ligature ordinaire ou ligature brusque. On vient de voir qu'il y a des conditions imprévues ou inconnues qui en font varier le mode d'action. Il y a d'autres conditions qu'on peut connaître à l'avance et qui viennent se joindre aux précédentes pour favoriser l'oblitération du sac par des caillots passifs.

On n'a pas oublié que les anévrysmes diffus ne se prêtent pas à cette circulation régulière qui préside au dépôt graduel des couches fibrineuses (1). La ligature ne peut donc faire déposer dans ces anévrysmes que des caillots passifs. Cela ne prouve ni pour ni contre la méthode d'Anel. Toutes les autres méthodes en sont là. Cependant certaines méthodes, telles que la compression indirecte et le traitement de Valsalva, agissant avec une plus grande lenteur, permettent à l'anévrysme de décroître notablement avant de s'oblitérer, et peuvent même réussir à faire déposer, dans la cavité de la tumeur, quelques masses fibrineuses qui diminuent d'autant le volume des caillots passifs et les chances de l'inflammation ultérieure. Mais je m'empresse de reconnaître que le mode d'action de la méthode d'Anel sur la tumeur des anévrysmes diffus, n'est pas notablement inférieur à celui des autres méthodes.

Il faut tenir compte aussi de l'ampleur de la poche anévrysmale. Toutes choses égales d'ailleurs, les anévrysmes volumineux, traités par la méthode d'Anel, sont plus exposés que les autres à l'oblitération passive, parce que le filet de sang qui y pénètre après la ligature, n'en renouvelle pas le contenu avec une rapidité suffisante.

Enfin, le choix du procédé de ligature exerce la plus grande influence sur la nature des caillots qui se déposent dans les poches anévrysmales. Le procédé de Hunter possède ici une grande supériorité sur le procédé d'Anel, parce qu'il porte une atteinte beaucoup moins profonde à la circulation de *l'anévrysme.* Le procédé de Hunter lui-même agit d'une manière d'autant plus favorable, qu'on laisse un plus grand nombre de collatérales entre le sac et la ligature. Ces propositions n'ont pas besoin d'être développées. En dépouillant les très-nombreuses observa-

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(1) *Voy.* plus haut, p. 145.

been given, are also to be recognised. Of this there are two varieties; one is connected with epilepsy, and proceeds directly from an epileptic attack, or may take the place of it. Under the influence of it great crimes have been committed. In the other form of transient mania the attack, with the usual violence, delirium, and delusions, runs a rapid course, terminating in recovery in a week or less. It is of importance to be able to decide whether the attack will be of this transient kind, or run the usual course of *acute* mania of four or five weeks' duration. Time is an element in solution, and in two or three days will solve the difficulty. The attack is likely to be transient if the invasion is very sudden, and if there is a definite and sufficient mental cause, such as a shock or fright. It is likely to be prolonged if its approach has been very insidious and gradual, and if there is no assignable cause. If the bodily condition is much affected, if the tongue is brown and dry, the urine scanty and high coloured, and if the bowels can hardly be moved by the strongest purgatives, it is *not likely* to pass off in a few days. If, on the other hand, the bowels are easily and freely opened, if the urine is copious and pale, and the tongue pale and moist, we may hope that the attack will be soon over, especially if there is extreme violence, bearing no proportion to this slight bodily disturbance. If sleep occurs in a day or two the attack soon subsides. Sound and long sleep is not to be expected so soon in *acute mania.* Removal to an asylum is not to be carried out till it is certain that the illness is *not* transient, and such a measure inevitable (BLANDFORD).

Intercurrent bodily diseases in the insane are of great importance in forming a prognosis, and demand careful study. They are exceedingly insidious in the insane as compared with the sane. In the insane their characteristics are masked or obscured by the inertia or trophic torpor which attends morbid states of the nervous system. The febrile type is generally typhoid or asthenic, and there is an absence of symptomatic fever. No complaint may be made, and no external evidence of pain or suffering may be given in cases even of *acute phthisis,* where subsequent *necropsy* shows the lungs riddled with vomicæ and full of pus (Vol. I., p. 280); or in cases of *phlegmonous erysipelas* going on to the formation of pus in the limbs; or in *pneumonia,* where the lungs are solidified, and normal respiration impossible; or in *organic diseases of the heart,* in *gastritis,* and in other painful diseases, such as *cancer, enteritis,* or *peritonitis.* The possibility of Bright's disease should not be forgotten. Surgical operations may be submitted to without a murmur, as if feeling were completely obsolete (*analgesia*). The most serious chest diseases may run their course without cough or expectoration; the excito-motor nervous system would seem to be nearly inert or torpid. Sooner or later progressive emaciation and debility, languor, lassitude, and indolence, perhaps anorexia or sleeplessness, direct attention to the state of the patient, in whom the physical signs then show the extent of the apparently latent disease, perhaps rapidly advancing for some time before towards a fatal termination (Dr. LINDSAY). The usefulness of the thermometer in detecting such latent disease ought not to be forgotten (see Vol. I., under "Fever"). The type or aspect of insanity may be quite changed by fatal bodily disease. A patient, from having been passionate, fretful, and abusive, may become affable, mild, and docile. The dying patient sometimes becomes sane towards his last moments, death being preceded by a bright though transient flicker of the light of reason. The sufferer has even expressed himself serenely, contentedly, happily, as to his latter end, and his transition from life, with all its troubles and diseases, to eternity, with all its joys and comforts (*Thirty-first Report of the Murray Royal Institution*, near Perth, p. 14). The mind may even then be soothed by the hopes and consolations of religion. "The cloud, though not wholly removed, may yet be tinged with a silver lining, and the music of the Gospel truth may help, like the harp of David, to charm the troubled spirit to repose" (Rev. W. D. KNOWLES, *Thirty-seventh Report*, l. c., p. 297). Such *euthanasia* for the insane is surely to be desired. That insanity also is sometimes vicarious, or alternates with other diseases, must be remembered in prognosis. It is so sometimes with phthisis, one of the most common complications of insanity. When the phthisis becomes acutely developed, the patient may become temporarily sane, and *vice versâ.* A return of the correct exercise of judgment is a fallacious indication of cure, so long as the emotions are perverted, even in a slight degree, from their normal condition; but immediately the emotions are controllable and proper, the cure may be considered complete (BUCKNILL, *op. cit.*).

The mortality among the insane (based on the average population of a mixed county asylum) is considered decidedly unfavourable if it exceeds 9 or 10 per cent.; a mortality less than 7 per cent. is highly favourable. The largest mortality is from *dementia*, the least from *monomania;* in the latter, indeed, where there is no tendency to suicide, the duration of life is little abridged; so that premature death is almost in all cases owing to accidental and often preventible causes (Dr. THURNAM). Expectancy of life in the insane, in relation to life insurance, may involve questions of great pecuniary importance. Popular as well as professional errors exist on the subject. The insane are separable into classes for the purposes of life insurance, each of which, within certain limits, is characterised by a different chance or expectancy of life. Statistics on the subject are greatly required (see Dr. LINDSAY'S *Thirty-second Report*, p. 17).

The **Treatment** of insanity resolves itself into the *medical* and the *moral* management of the case. Medicine indirectly acts upon the brain, as it does upon other organs, so as favourably to influence the course of the disease. It regulates the different actions and secretions of the viscera of the body, and thus improves the general health, so that the happiest results are often obtained by the early and judicious use of medicinal agents. No uniform method of treatment can be taught. The condition is essentially one of asthenia, feebleness, anæmia, or exhaustion. Local bleeding may be adopted in some cases with reference to a distant viscus. Leeches to the *vulva* and thighs are beneficial in some cases of *mania, monomania,* or *melancholia,* in adults, when such disorders of intellect are concurrent with the menstrual period; and to the *sphincter ani* in those cases obviously connected with suppressed hæmorrhoids or hepatic congestion. In some instances leeches may be applied with benefit to the Schneiderian membrane, particularly in those cases occurring in early life, and in persons of plethoric constitution and of sanguine temperament. Illusions of hearing or of vision, which had embittered the patient's life, have been removed by leeching behind the ears or over the superciliary ridges.

The utility, in *acute mania,* of prolonged hot baths was much insisted upon by the late Dr. Winslow. The patients remain from eight to fifteen hours in them, at 82° to 86° Fahr., whilst a current of water at 60° is continually poured over the head. Various details of what is now known as hydropathic treatment have recently been introduced, with great advantage, into English asylums, such as the Russian or vapour bath, the wet sheet, wet pack, and the like. Packing in the wet sheet, warm baths with cold to the head, will often procure sleep more certainly than medicines of the sedative or hypnotic class.

Sedatives, or agents which modify directly the condition of the cerebral tissue, and which procure sleep or rest to the brain, constitute very valuable remedies. In recent acute cases they are generally admissible; and are only contra-indicated if constipation keeps up the excitement and sleeplessness; or if local cerebral irritation exists; or if the general health and secretions are disordered. In the various chronic forms of *melancholia* they are most useful, and in suicidal insanity. The *meconate* and *hydrochlorate of morphia* should be *uninterruptedly and perseveringly given,* in gradually increasing doses, until the nervous system is completely under their influence. The preparation Dr. Millar has found most useful and the least productive of constitutional disturbance, is a *solution of opium in water, acidulated with citric acid* (containing about *one-sixth* of crude opium) administered every four or six hours, in doses of from 10 to 20 minims, (equal to 10 grains of crude opium daily), and continued until the patient feels he can sleep without it. So also will *chlorodyne* and *chloro-morphine* be of use in some cases, as well as *chloral, bromide of potassium,* and *hyoscyamus. Indian hemp* is an extremely useful sedative, not hitherto appreciated sufficiently for many reasons. (See an excellent paper on its uses by Professor RUSSELL REYNOLDS, in BEALE'S *Archives,* Vol. II., p. 154.) It relieves pain, is *soporific, anodyne, antispasmodic ;* and while conducing to sleep, promotes at the same time *diaphoresis* and *diuresis,* without producing headache, vertigo, constipation, or impairing the appetite. The dose varies from *one-sixth to one-half grain for a child,* and from *one-third to one grain and a half for an adult.* In cases of mental or emotional disturbance it will be found extremely useful, especially where there is deranged cerebral circulation, with pain and delirium; in cases of incipient insanity after fever or *sunstroke ;* and in cases of *senile ramollissement.* Recently *digitalis, conium, belladonna,* have been extensively employed as calmatives. *Endermic medication* in insanity offers numerous advantages; and *hypodermic injections,* especially of calmatives and soporifics, have also recently been employed. Dr. Reissner's experience leads him to recommend *morphia,. codeia,* and *narceia,* the former being much the most useful, and the others only to be used in special cases when morphia fails. *Narceia* he recommends in those cases where the injection of morphia produces unpleasant symptoms of an uncomfortable kind—not to say of a poisonous tendency. *Narceia* he considers a special remedy in such cases. Preparations of opium introduced into the system by the hypodermic method, are more speedily manifested by the results than when administered by any other mode. The *acetate of morphia* is the best form with a minimum of *acetic acid* in hot distilled water, in the proportion of *five* grains of *acetate of morphia* to one fluid drachm of water or of glycerine. *One minim* of this represents $\frac{1}{12}$ of a grain—a safe and useful minimum dose. *Two minims* (equal to $\frac{1}{6}$ of a grain), is the best commencing dose for relief of severe pain, and as a hypnotic in states of nervous irritability, whether connected with disorder of the intellect or other diseases. *Three minims,* or $\frac{1}{4}$ of a grain, *is an unsafe dose* TO COMMENCE WITH; dangerous and even fatal results have resulted from such a dose. It should not be given till smaller doses have been tried. Used *endermically,* the salts of opium are reckoned to be three times as powerful as when swallowed (WARING). *Hydrate of chloral* is of use in subduing the delirium of mania, and sometimes in obtaining sleep, combined or not with *bromide of potassium.*

Purgatives may be regularly required. When the bowels are constipated the form is best determined by the state of the tongue, and sometimes by the idiosyncrasies or proclivities of the patient in regard to medicine-taking. Supposing the tongue to be white and coated, the *sulphate of magnesia,* or other neutral salt, combined with *tincture of hyoscyamus,* in the proportion of ʒj. of the former to ♏ xv. to ♏ xxx. of the latter, in *camphor mixture,* is a formula to be recommended; so also is *calomel* (*five* grains) with *compound jalap powder* (ʒi.), and a little powdered ginger; and also *watery extract of aloes* nightly. If, on the contrary, the tongue be clean, the cathartic should be given with some slight bitter, as the *infusion of orange peel* or of *gentian.* In some cases the bowels are not only exceedingly obstinate, but the patient may be greatly averse to all medicines. In such cases one or two drops of croton oil placed on the tongue or introduced in food produces free evacuations. Mild purgative treatment formed the basis of cure in the school of Pinel and of Esquirol, usually combined, in cases of violence, with the application of cold to the head, and of warmth to the lower parts of the body; such as placing the patient in the warm bath, and giving him the cold douche—a remedy since more particularly insisted upon. The further treatment consists in restoring any other functions that may be in defect or in excess, as the functions of the uterus in the female, and of the liver or heart in both sexes, by the usual remedies applicable for these purposes.

When there is anæmia, vegetable tonics, combined with the preparations of *iron,* are of great service. At the same time the patient ought to be supplied with an abundance of nutritious and easily digested food. It quiets the noisy, and lessens the dirty, mischievous, and destructive propensities of others. Where there is an absolute refusal of solid food, as in acute mania, it is essential that the liquids (which the patient for the most part readily takes) should contain as much nutritive material as possible. Strong soup and broth, beef-tea mixed with milk and with arrow-root, must be given often. The best form of food is that in which eggs and milk largely enter. Two or three eggs beaten up, and mixed with half a pint of warm milk, warm ale, or warm porter, may be given with a sedative every four to six, or eight hours, and continued daily till excitement abates, or the ability to take solid food returns. Such stimulant food is well borne, and absolutely necessary in cases of acute mania. In *melancholia,* three to twelve ounces of wine may be necessary in the twenty-four hours; and there is also a tolerance of *opium.* Ten minim doses may be given three times a day. If impulsive tendencies are manifest with despondency, *bromide of potassium* should be given in thirty to forty grain doses, with an *iron* mixture. If the tendency is to atonic lethargy, large doses of *quinine*, or small doses of *strychnia*, are to be given with the *iron*. *Easton's syrup* is valuable. In neuroses of the stomach and gastralgia, *arsenic* and *bismuth* are both of much service in small doses. *Muriate of ammonia* is of special use in those cases in which the depression is intense and paroxysmal in character.

The moral management of disorders of the intellect is by many supposed to constitute the more efficient mode of cure; and it must be admitted to be a most important adjunct. The first important rule is to remove the patient at once from his family. In slight cases this step is necessary in order that he may be induced to exercise such command over himself as he possesses, and to remove him from influences which may have been aggravating his morbid state; such as finding himself a prisoner, instead of being master in his own house. In severe cases it is necessary, in order to prevent his doing mischief either to himself or others. In melancholia no form of treatment is so efficacious. The great object is to break up the monopoly of grief and fear which has obtained possession of the mind; and if a new interest can be awakened, or a fresh hope insinuated, this object is in a great part obtained. At the same time solitary brooding and idleness must not be permitted; and at least cheerful companionship must be secured. It is important also to seek out and to treat visceral or constitutional diseases and relieve them—*e.g.*, gout, syphilis, uterine disorders, climacteric conditions, and the like. The main feature in the moral management of the insane in this country is the abolition and absence of mechanical coercion or restraint. The beneficial action of this system, generally known as "*the non-restraint system*," is now thoroughly recognised in England and Scotland, where it has been gradually established in every asylum since 1847–48. But there are certain exceptional phases of insanity, in which some mechanical restraint is the most humane mode of treatment—the only mode, indeed, of avoiding certain catastrophes of too common occurrence. In certain conditions of excitement it is proper to place the patients at once in a darkened room, remote from noise and the means of injury to himself or others, so that as few objects as possible may irritate him, just as a patient with his eyes affected is kept in a darkened room. The effect of such seclusion is generally of a soothing character; and in not a few cases of periodic mania it is eagerly sought for by the patients themselves. When a case of melancholia is of severe type from the first, *muriate of ammonia* and *tincture of opium* are the most valuable agents. There is a tolerance of *opium* in simple melancholia. So long as depression lasts it is taken with benefit to every function. When depression terminates it may be abandoned at once without inconvenience. As convalescence advances the patient should be induced to undertake some manual labour, or some office in the asylum or household, which, by amusing his mind, will invigorate his body, and greatly tend to restore the healthy working of his brain. There are no more powerful moral medicines than "occupation," "recreation," and "education." Occupation should be such that no time is left for idleness, or for sitting brooding over morbid fancies. The curative results of well-chosen means of recreation cannot be over-estimated. When the circumstances of the patient admit of it, travelling, which embraces change of air and change of scene as well as exercise, is often highly salutary in incipient cases; and much has of late been done by the judicious introduction of music and other amusements into asylums. Change of scene acts often like a tonic to the depressed and jaded powers; but where there is great restlessness and feverish perturbation, travelling and change of scene should not be resorted to (Dr. J. CRICHTON BROWNE). Concerts, balls, conversaziones, evening entertainments, pic-nics, excursions, *fêtes champêtres*, athletic games, pedestrian excursions, public amusements in towns, and carriage drives, are all legitimate and well-approved means of maintaining a constant and varied succession of recreation adapted for all classes of the insane. When reason is restored, and the affections again fix themselves on their natural objects, *and when the emotions are under control;* the patient may be allowed to see his friends, and have his attention directed to the affairs and interests of his family; but it should be remembered that the mind remains weak and enfeebled for some time after apparent recovery; and, consequently, the patient's restoration to society should be gradual.

An hospital for the insane, or a lunatic asylum, is the most fit and proper place for a "person of unsound mind." But every such asylum ought to be governed by one superintendent, who shoud be a medical man—an officer of health to the community over which he presides. He ought to have the means of controlling all sanitary arrangements in whole and in detail—of avoiding overcrowding—of preventing and destroying effluvia—and of examining the quality of the food, the water, and the drugs furnished to the establishment. Chemical and pathological appliances ought therefore to be at his disposal for his use. The number of patients who may be thus under the supervision of one medical head in an asylum ought not to exceed 200 (ESQUIROL, AMERICAN COMMISSIONERS IN LUNACY). Premature removal from asylum treatment, in opposition to medical advice, is greatly to be deprecated; and its baneful results are frequently to be seen, especially in the sad endings of cases of *suicidal melancholia*. In such cases an acknowledgment should be required of the recipient of the patient, or his friends, that he is removed notwithstanding the assurance given by the medical superintendent that the patient is not recovered, and is unfit for removal (Dr. LINDSAY, *Thirty-second Report*, p. 14). Such removals not unfrequently induce a change of type from acute and curable to chronic and incurable mental disease.

The subject of insanity has been treated of in this text-book because it is a subject which medical men must study as they do any other disease; and which they may be called upon to treat and to deal with in a medical and also in a medico-legal point of view. They have, however, few opportunities at our schools of medicine of studying insanity as they would any other disease in a general hospital. They are, therefore, often called upon to give certificates of insanity, without perhaps having ever seen or studied a case of disorder of the intellect; and many recent legal decisions have shown that it is not always safe for a medical man to incur the responsibility of signing such certificates, in the present state of the law relating to lunacy. Seeing, also, that the legal and medical views of insanity are not in unison, and that the legal views are not in accordance with what is known as the correct pathology of the disease, the subject has been treated of as fully as space would permit. To learn this important subject in all its bearings, the student is recommended to study the comprehensive works of Maudsley, Bucknill, and Tuke; and the valuable reports,—(1.) of the Murray Royal Institution for the Insane, near Perth, prepared by Dr. Lauder Lindsay; and, (2.) those of the West Riding Lunatic Asylum, edited by Dr. Crichton Browne, both of which have ceased to be published. They are each in their way models of reports from the scientific point of view.

CHAPTER XIV.

DISEASES OF THE EYE IN RELATION TO DISORDERS OF THE NERVOUS SYSTEM AND TO GENERAL DISEASES.

THE numerous morbid states to which the eye is liable, as well as the peculiar and varied structures of which it is composed, enable us to see almost all its diseases in miniature, as if through a glass, and "to learn many of the little wonderful details in nature of the morbid processes, which, but for the observation of them in the eye, would not have been known at all." So wrote Dr. Latham many years ago, relative chiefly to the phenomena of inflammation, and before the ophthalmoscope was devised. Sir Thomas Watson commenced his course of Lectures on Medicine with disease of the Eye; and for this reason,—"that we find in the eye more satisfactory and plain illustrations of the *general* facts and doctrines of pathology, than in any other single organ of the body," (*Lect.* xvii.) Since the ophthalmoscope has enabled us to look into the interior and very innermost depths of the eye, it is found that visible indications of disorders of the nervous system may be seen in its structures which can be seen in no other part of the body. Hence, the great importance to the physician of examining the eye by the ophthalmoscope in all constitutional diseases as well as in those of the nervous system, as first pointed out in England by Dr. John Ogle. Since then Drs. Hughlings Jackson and Gowers strongly urge the routine use of the instrument in all diseases in which ophthalmoscopic changes are even occasionally met with. It is thus that unexpected information may be gained regarding the nature of the disease and its probable consequences (GOWERS' *Medical Ophthalmology*, p. 4).

Certain changes in the eye are known to accompany certain diseases of the brain and spinal cord, and also certain lesions of organs more remote, which are also the expression of *general* and *constitutional disease.* But, like the subject of syphilis, the subject of diseases of the eye has suffered from the unscientific division of the field of medical practice into surgery and medicine; and, at the same time, the uncombined and partial study of eye diseases by the physician and the surgeon have been a loss to each, preventing each from taking the comprehensive view of the whole facts which belong to each particular case. One-half the facts are overlooked on the one side or on the other; for the ground has been neutral at one time, at another claimed as the exclusive province of the surgeon, the physician, or the oculist. Although the diseases of the eye form now almost a distinct specialty, and are claimed by the ophthalmic surgeon, nevertheless pathology recognises no such artificial distinctions. The student of medicine must study diseases of the eye with the aid of the ophthalmoscope as a general practitioner of the healing art; and physicians are learning every day to appreciate the advantages of this instrument as a valuable aid to diagnosis and prognosis, especially in cerebral affections. The medical schools have now recognised the necessity of teaching its use. The innermost depths of the eye of patients suffering from any acute or chronic affection of the nervous system ought to be examined, whether they complain of defect of sight or not; and practical experience in the use of the ophthalmoscope is absolutely essential for the physician. To the works of Bader, Ernest Hart, Jonathan Hutchinson, Hulke, Hughlings Jackson, George Lawson, Macnamara, Liebreich, and Gowers, the reader is referred for guidance and directions in acquiring a practical knowledge of this instrument, and what it teaches.

The eye furnishes important *indicia* of cerebral disease, and requires to be examined,—(1.) By comparing and observing the state of the pupils; (2.) by ascertaining the extent of the field of vision in both eyes; and, (3.) by the ophthalmoscope. This instrument shows—(1.) The termination of an artery and the commencement of a vein, and the blood circulating within them, the columns of which are seen as red lines. The walls of the vessels are, as a rule, invisible. (2.) The termination of a nerve, which, from its close proximity to the brain, and from other circumstances, possesses significant relations to the rest of the nervous system. (3.) A nervous structure—the retina—which suffers in a peculiar way in many general diseases. (4.) A vascular structure—the choroid—which also presents changes in certain constitutional affections. All these can be looked upon if the student can use the ophthalmoscope.

One of the first and most important observations, in a pathological point of view, is the recognition of the *close relations which exist between the cerebral and intra-ocular circulation;* and especially the changes in the *optic disc*, the *retina*, and the *choroid*, happening in the course of cerebro-spinal, or any other disease. The following account of these changes has been compiled mainly from an article by Dr. Allbutt on "Medical Ophthalmoscopy," in the *Brit. and For. Medico-Chir. Review*, Vol. XII., 1868, p. 127; from papers in the *Medical Times and Gazette*, for 1868, by the same author; from the works of Hughlings Jackson, Bader, Hutchinson, Hulke, and, more recently, Dr. Gowers' work on *Medical Ophthalmoscopy*, the first work of the kind in this country.

The *optic disc* may be *seen* to be the seat of simple congestion, and of congestion with effusion within or around it, of inflammation of its sheath, of inflammation in its substance, of anæmia, and, lastly, of atrophy. The circulation of the optic nerve is thus judged of from the condition of its disc and of the retina, so that any change of vascularity evident in these parts of the *fundus* of the eye may be to some extent an index of changes in the cerebral circulation of which it is a part. Remarkable variation is met with in the colour of the *optic disc.* Its tint may vary as much as the tint of the cheek, and is always redder in the young than in the old (GOWERS). The *optic tract, chiasma,* and *nerve* receive their blood-supply chiefly from the *choroid plexus* and *pia mater;* the *optic tract* generally receives also a branch from the middle cerebral artery; and close to the sclerotic the *optic nerve* receives the short *ciliary arteries* given off from the *ophthalmic* soon ofter the *arteria centralis retina.* Thus the blood-supply influences the appearance of lesions as they proceed from *optic disc* or *retina.* The *retina* may be *seen* to be the seat of fibrous and fatty exudations or patches, more especially in the course of the vessels, also of hæmorrhages. The *choroid* may be *seen* to be the seat of disturbance, or loss of its pigment, and of hæmorrhages. These affections of the nerve-vascular parts of the eye may denote—(1.) *Simple hyperæmia of the discs and retinal blood-vessels;* (2.) *Anæmia of the same parts;* (3.) *Ischæmia of the disc and its consequences;* (4.) *Acute interstitial neuritis and consecutive atrophy;* (5.) *Neuro-retinitis;* (6.) *Perineuritis, chronic neuritis;* (7.) *Primary or simple progressive atrophy* (ALLBUTT).

The blood-vessels within the eye may be *seen* to undergo many characteristic changes, such as diminutions or obliterations, aneurismal dilatations, miliary aneurisms, tortuosities, pulsations, varicosities, blood stasis, embolism, thrombosis, or rupture. Examples and descriptions of these various morbid lesions are to be seen in the valuable Atlas of Liebreich, and in the chromo-lithographs of Bader, Galezowski, Bouchut, and the illustrations to Dr. Gowers' work on *Medical Ophthalmology.* In severe congestions within the eye there is generally a decided difference between the condition of the two eyes. The *papilla* is scarcely distinguishable in colour from the *retina,* and perhaps is to be traced only by the convergence of the vessels. The veins tend to become swollen and tortuous, sometimes varicose. When the centre of the disc remains white, this form of congestion has been named *peri-papillary;* and is often seen to be confined wholly or partially to one part of the circumference of the *disc* with the corresponding district of the *retina.* The retardation of blood in the veins produces in them every degree of change, from simple dilatation to varicosity, elongation, and even rupture. Ruptures are most frequent in cases of *albuminuria.* The effused blood degenerates more or less quickly, so that these hæmorrhages appear as whitish blotches, patches, or streaks in the course of the vessels (*choroidal atrophy* and *opaque nerve-fibres*), and are very characteristic of that state of the system in which the small rough kidney is found. Simple *hyperæmia* may be due to orbital disease, to *choroiditis,* or to Bright's degeneration and *alcoholismus;* but in by far the greater number of cases it is due to encephalic disease—to tumors, acute or chronic meningitis, or to changes in cerebral vascularity, attended with convulsions.

The optic nerve and disc.—The lesions which attract most attention in this part, as connected with cerebral disease, are:—(1.) *Optic neuritis,* with its *consecutive atrophy;* and, (2.) *Primary or progressive atrophy.*

Optic neuritis is very generally co-existent with *meningitis* of the base of the brain, with *tumors, abscess,* and *hydatid disease;* also *softening of the* *brain,* from vascular obstruction, and with large *hæmorrhages.* Other causes outside the brain are, *albuminuria, lead poisoning,* certain *febrile diseases* and *blood states.* Hence it is a valuable symptom. It is to be distinguished from the *retino-neuritis* of albuminuric patients, and from the *retino-choroiditis* of syphilis, mainly by the history of the case, and the limitation of the affection for the most part to the papilla and converging vessels. The relation of optic neuritis to encephalic disease is still the subject of much divergence of opinion. Dr. Hughlings Jackson maintains that encephalic irritation best explains the occurrence and phenomena of optic neuritis, and that its occurrence is not related to increased intracranial pressure. Galezowski believes that neuritis is always descending, and maintains, contrary to most observers, that the intra-ocular change is in all cases the visible manifestation of an inflammation propagated by continuity from the brain. Dr. Gowers comes to the following conclusions:—(1.) *Optic neuritis,* limited to, or most intense in, the optic papillæ, may occur without any obvious cranial or cerebral disease. (2.) Facts generally make it difficult to connect papillitis with increase of intracranial pressure. (3.) Evidence of descending neuritis may be generally traced in cases of cerebral tumor; and in cases of meningitis the evidence of descending neuritis is almost invariable.

An important stage in the process of *optic neuritis* is the commencement of the *consecutive atrophy.* The intense vascularity in and about the disc subsides—the infiltrations are absorbed—the nerve whitens, and the capillaries slowly shrivel and vanish. The edges of the disc become distinct, but are deformed; and patches of organised lymph are to be seen upon and about them. The atrophy may be a primary change, or secondary to some lesion. In the former, loss of sight coincides in origin and progress with the visible atrophy; in secondary atrophy the loss of sight occurs first; and the signs of nerve degeneration are not observed until a subsequent period. Primary atrophy is sometimes distinctly hereditary. Males are more prone to the lesion than females. The neuropathic dyscrasia is often well marked, an indication of heredity. Cold, sexual excess, the toxæmia of alcohol, lead, and tobacco, blows in the neighbourhood of the orbit, and overuse of the eyes, are all occasional causes of atrophy. An isolated degeneration of the optic nerve may also be associated with a similar degeneration somewhere else in the nervous system, as in locomotor ataxy—a parenchymatous—*i.e.,* a primary neural form; also in general paralysis of the insane; in disseminated (insular) and lateral sclerosis of the cord. Other causes are, gastric intestinal affections, syphilis, diabetes, menstrual disturbances, migraine, and malarious fevers. Primary atrophy usually affects both eyes, and one much more and earlier than the other. *Secondary* atrophy results from lesion of the optic centres or fibres. A cortical lesion in the brain, about the supra-marginal gyrus (FERRIER), may entail loss of sight of the opposite eye. A lesion outside the hinder part of the optic thalamus causes loss of sight of the opposite eye, and of the opposite half of the field of vision of the same side, after an interval of time. Lesions of one optic tract cause bilateral, symmetrical hemiopia, unaccompanied by ophthalmoscopic changes until after some years. Very nearly 50 per cent. of the cases of atrophy are associated with disease of the brain and cord (GOWERS, l. c.). Examination shows a change of sight in three directions

—(1.) diminished acuity of vision; (2.) alteration in the field of vision; (3.) altered perception of colours (GOWERS, l. c.).

Simple primary or *progressive atrophy of the optic disc* is the most hopeless of lesions. It is a process of degeneration from within, mostly ending in utter blindness. It must be distinguished from atrophy with *glaucomatous excavation* or *posterior staphyloma*, and from the changes in the disc which may accompany pigmentary retinitis and choroiditis. The appearance of *progressive atrophy* is characteristic. The fine capillaries which give the rosy tint of the healthy disc slowly wane, and a dead white or pearl-like white is left. Vision is lost, passing steadily and surely away. The border of the disc is sharp, clearly defined, flat and even, and it strongly contrasts, by its whiteness and definition, with the red tissues surrounding it. The lesion depends nearly always on some disease of the cerebrum, cerebellum, or spinal cord. In many the atrophy is of a mixed kind, and there may be slight effusion. *Hemiopia* (lateral) is always due to cerebral disease. The *optic nerves* are almost always involved in the process of *meningitis* at the base, and in *tuberculous meningitis optic neuritis* almost always exists. Not so meningitis confined to the *fissure of Sylvius*, or upon the convex surface of the brain. In the first period of *meningitis* dilatation of the veins of the *retina*, peripapillary congestion, and often effusion, are common. In the second period, tortuous veins, stasis, thrombosis, and even rupture of the vessels—lesions which appear more or less quickly, according to the amount of obstruction to the circulation in the sinuses occasioned by the meningitis. It always tends mechanically to oppress the venous circulation. Lesions in the back of the eye are also to be seen in Bright's disease and syphilis. In *Bright's disease* the nutrition of the *optic nerve*, as well as of the *retina*, is profoundly altered; and atrophic changes of the nerves have been found, even at the corpora quadrigemina, with fatty patches in and about the *chiasma* (GALEZOWSKI). Upon the *retina* extravasations are found in the course of the vessels. These extravasations are slowly effused, and pass into degenerative states, forming white patches or striations along the margins of the veins, while other patches may be due to degeneration of the retina itself. These *retinal* degenerations are the first in order in *Bright's disease;* while in *cerebral disease* the *optic disc* is generally the first part to show signs of change. In *syphilis* the *choroid* is the chief seat of lesion; and patches of many colours are to be seen at the back of the eye, some of a brilliant white, others of darker tints, such as red or brown.

The most important indications of cerebro-spinal organic lesions capable of recognition by ophthalmoscopic examination of the eye, may be summed up as follows, as significant of general diseases :—

(1.) *Optic neuritis* and *neuro-retinitis*, *choroiditis*, and *papillary atrophy* accompany the greater number of acute and chronic affections of the brain and spinal cord. (2.) The law of coincidence of *optic neuritis* and organic lesions of the nervous system may be explained by the anatomical and physiological relations of the eye with the brain and spinal cord. (3.) Whenever any positive hindrance occurs in the cerebral circulation, in consequence of a lesion of the brain or spinal cord, there may be *papillary* and *retinal hyperæmia*. (4.) When there is acute or chronic inflammation of the brain, it may extend to the eye by following directly the course of the optic nerve. (5.) Affections of the anterior spinal cord may, on account of the union of its nerves with the sympathetic, at a level with the two first dorsal pairs of nerves, produce the phenomena of *papillary hypertrophy* in the eye, followed later by *atrophy* of the *optic nerve.* (6.) *Optic neuritis* and *neuro-retinitis*, caused by acute and chronic diseases of the nervous system, are generally found in both eyes. (7.) In affections of the brain and its meninges, *optic neuritis* is most often more marked in the eye corresponding to the hemisphere most severely affected. (8.) Changes in the *optic nerve* and the *retina,* accompanying disorders of sensibility, of intellect, and of motility, always indicate organic diseases of the brain. (9.) These changes in the *optic nerve* and the *retina* should not be separated from other symptoms of the existing disease; their presence, however, is an element of diagnosis of positive certainty. (BOUCHUT, French Academy of Sciences, June 8, 1868.)

Ischæmia of the discs occurs in all intracranial affections which more or less directly distend the ophthalmic veins, as *meningitis*, *hydrocephalus*, and *intracranial tumors.* In *meningitis* the exudation at the base of the brain may press upon, or the inflammation may involve, the cavernous sinus, in which case there is only *ischæmiæ.* Or the inflammation may creep down the nerve and cause *neuritis optici*, or it may mainly follow the sheath of the nerve and cause *perineuritis;* or it may both affect the sinus and the optic vein, and may creep down the nerve, in which case there will be both *ischæmia papillæ* and *neuritis.* The changes in the eye often occur early enough to be the first certain signs of the existing disease. The mirror also shows the presence, or the traces, of meningitis in a large number of children, and of adults, who survive, and in whom the disease may or may not have been suspected. Such persons who have survived the disorder, tubercular or not, may wholly recover from a state of obscure and protracted ill-health; or a want of full mental power, or a capriciousness of temper may remain; or the reason or the affections may be changed to the degree of insanity (Dr. CLIFFORD ALLBUTT). Hydrocephalus is very destructive to the optic nerves. If it be extreme, the discs and retinæ become wholly disorganised, and the optic nerves atrophied by pressure. *Ischæmia papillæ* is the earliest change.

Intracranial tumors, including not only malignant growths but cysts, aneurisms, and all local enlargements and thickenings, are, after meningitis of the base, the most frequent cause of *ischæmia* and *neuritis.* Disease of the cerebellum—tumors, inflammatory changes, and softening—cause mischief to the optic discs. Acute and chronic cerebritic softening alone never produces *ischæmia*, the changes in the optic nerve being *neuritis*, acute or chronic, and atrophy. In cerebral hæmorrhage, when the effusion is large, it causes stasis and infiltration in and about the optic discs by obstruction, and this is more marked in the eye corresponding to the site of the clot. In *symptomatic epilepsy*, with coarse, cerebral lesion, optic atrophy is common, being connected with the organic affection, but it is never associated with essential epilepsy; some circulatory troubles occur in the latter, and venous dilatations are said to have been seen in the central ganglia in severe cases. Dr. Hughlings Jackson has recorded many instances of convulsions beginning unilaterally, in which there was double *optic neuritis.* In *general paralysis*, atrophy of the discs is almost constant; but it is not an early symptom, appearing generally about the end of the first stage or the beginning of the second. In *disease of the spinal cord*, simple progressive atrophy of the optic nerve is not uncommon. It is frequent in locomotor ataxy—sclerosis of the posterior columns of the cord—and it has been observed in some cases of chronic myelitis not attended with shooting pains. It probably depends upon an affection of the great sympathetic, through its communication with the anterior roots of the spinal nerves. Coloured vision has been seen in epilepsy and in sunstroke.

Colour-sensibility is of great importance to be recognised at the commencement of nerve atrophy, and in some forms of syphilitic disease. In *retinal apoplexies* the *chromatic* faculty is liable to alteration when the patches are very large, or when they affect the central parts of the retina. In *syphilitic retinitis* or *neuritis*, with or without choroiditis, the general impairment of sight is attended by loss of perception of green, and sometimes of red. In the *retinitis* of *albuminuria* or of *diabetes*, there is no colour-blindness until the disease has reached an advanced stage, so as to involve the external layers of the *retina* and *macula lutea*. In *atrophy* of the nerve the chromatic function suffers from the outset, especially with regard to red and green.

The student is referred to the special treatises on ophthalmology, for an account of the substantive diseases of the Eye.

CHAPTER XV.

DIAGNOSIS OF DISEASES OF THE HEART AND LUNGS.

The pathology and diagnosis of diseases of the heart and lungs—of the thorax generally, its contents and parietes—are so intimately related to each other, that some preliminary sections are necessary, the object of which is to show how these diseases are recognised and appreciated by certain *symptoms*, local and constitutional, as well as by *physical signs.* The topics may be arranged in the following order (see also pages 24, 25 Vol. I.).

Section I.—Relation of the Thoracic Viscera to the Walls o the Chest.

(A.) *Regions of the Thorax.*

In order accurately to describe and record observations, and mo conveniently to communicate precise information respecting the s and signs of internal diseases, it has been customary to map out exterior surface of the chest and abdomen by lines into different comp ments, termed *regions.* These lines have not always been drawn in same manner; and therefore, there has ever existed great discrepancy among writers and teachers in this and other countries respecting the number, the extent, and the names of the regions defined by such lines. The regions are arbitrary, and the lines described upon the surface are understood to correspond with imaginary planes passing towards the centre of the body. The points, or *landmarks*, on the surface through which these lines are drawn ought to be fixed points and obvious; so that the boundaries and contents of every region may be accurately defined, with the view of localising physical signs as precisely as possible. As it is necessary that a complete inspection of the body shall take in simultaneously the abdomen and the chest, the topography of these regions is given together. The annexed regional plans of the trunk are those devised by Mr. Paxton, of Oxford; and given by the late Sir John Forbes, in his paper "On the Exploration of the Chest and Abdomen," in the first volume of the *Cyclopædia of Practical Medicine.** Prominent points of the skeleton are made the basis of the system by which the regions are mapped out by vertical and horizontal lines.

The *vertical lines* having relation to the chest, are *eight* in number, as follows:—(1.) Along the middle of the sternum, from its upper to its lower end; (2.) from the acromial end of the clavicle to the external tubercle of the pubes (right and left); (4.) along the spinous processes of the cervical and dorsal vertebræ; (5.) along the posterior or spinal border of the scapulæ, from the clavicular transverse line to the mammary transverse line. (See Figs. 19, 20, and 21).

The *horizontal*, or *transverse lines*, are four in number:—(1.) Around the lower part of the neck, sloping downwards to the upper end of the sternum anteriorly, and to the last cervical vertebra posteriorly; (2.) around the upper part of the chest in the line of the clavicles; (3.) around the middle of the chest by the lower edge of the *third* rib, above the line of the male nipple, and touching the inferior border of the scapulæ behind; (4.) around the lower part of the chest, on a level with the xyphoid cartilage. (See figures, as before.) By these lines the lower part of the neck and the chest is divided into three horizontal and eight vertical bands; and by their intersection various compartments or regions may be indicated, as follows:—*Anterior*, *lateral*, and *posterior* regions being recognised, the ANTERIOR are named the *supra-clavicular*, *clavicular*, *infra-clavicular*, *mammary*, *infra-mammary*, *supra-sternal*, *superior sternal*, and *inferior sternal*. The LATERAL regions are,—the *axillary* and the *infra-*

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* To facilitate accuracy in this method of recording physical signs, blank outlines of the trunk of the body were first used by Piorry, and more recently they have been largely used both in clinical teaching and in recording cases for publication, by Professors Bennett, in Edinburgh, Gairdner, of Glasgow, and Beale, of London; the latter of whom has especially described the great advantages from their use, in the sixth number of the valuable *Archives* which he published (Vol. II., p. 97). The *outlines* merely of the trunk (without the dotted lines and the numbers), given in the text to illustrate the topography of the chest and abdomen, have been engraved on wood; so that the student can readily indicate the areas of physical signs by pencil, ink, or coloured chalk lines. My publishers are prepared to supply *fifty* blank copies of these outlines, at the cost of one shilling and sixpence, under the following title:—"Outline Figures for indicating the Areas of Physical Signs in the Clinical Diagnosis of Disease;" for the use of Students and Practitioners of Medicine. Arranged by WILLIAM AITKEN, M.D., F.R.S., &c. Charles Griffin & Co.

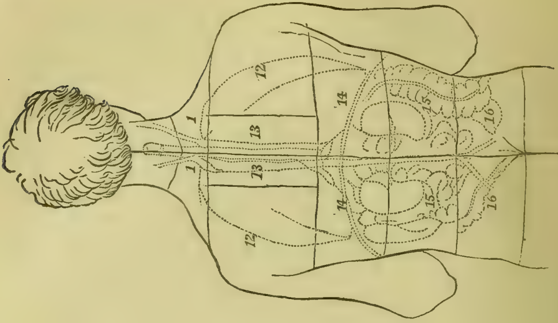

Fig. 21.

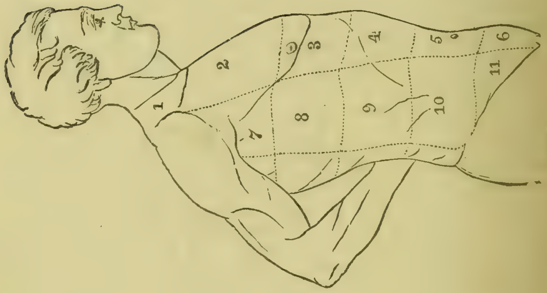

Fig. 20.

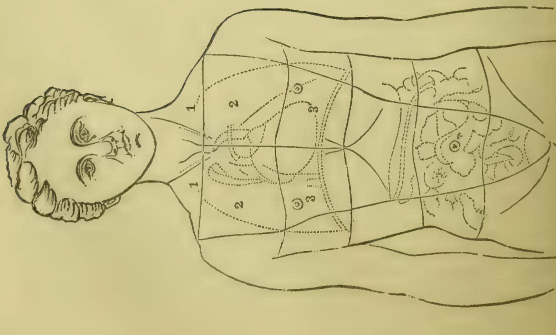

Fig. 19.

axillary. The POSTERIOR regions are,—the *supra-spinous;* the *infra-spinous* (sometimes called the scapular); the *inter-scapular;* and the *infra-scapular* (sometimes called the upper dorsal). Of these regions the three sternal are single; all the rest are double.

The Supra-Clavicular Region is a small triangular space *above* the clavicle on either side, with its base internally at the trachea, its apex towards the outer end of the clavicle, and bounded below by the upper edge of that bone. A line drawn from the outer part of the clavicle to the upper rings of the trachea will limit its upper border. In this region is found the triangular apex of the lung (Figs. 22 and 23), sometimes reaching on the right side a little higher than on the left, with portions of the subclavian and carotid arteries, and of the subclavian and jugular veins. The floor of this region internally is formed by the upper surface of the first rib.

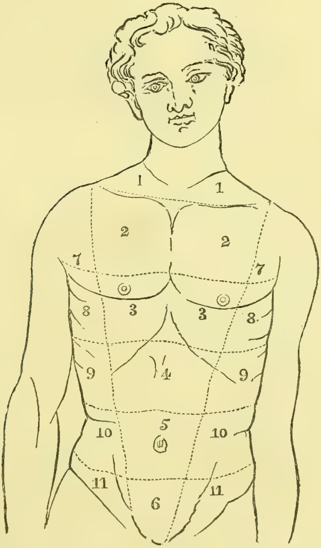

Fig. 22.

The Clavicular Region is very narrow and oblong, corresponding to the inner two-thirds of the collar-bone. Behind the bone lies on both sides lung-substance; but on the right side, at the sterno-clavicular articulation, is the *innominate artery,* and the *subclavian artery* crosses the region at its outer edge; on the left side the *carotid* and *subclavian arteries* pass upwards, almost at right angles to the bone.

The Infra-Clavicular Regions are nearly square, corresponding to No. 2 in the figures. Each region (right and left) is bounded above by the inferior border of the clavicle; below by the lower border of the third rib, where it joins the cartilages of the sternum; it is bounded on the outside by the vertical line passing from the acromial end of the clavicle downwards towards the external tubercle of the pubes (on either side); on the inner side the subclavian region is bounded by the edge of the sternum.

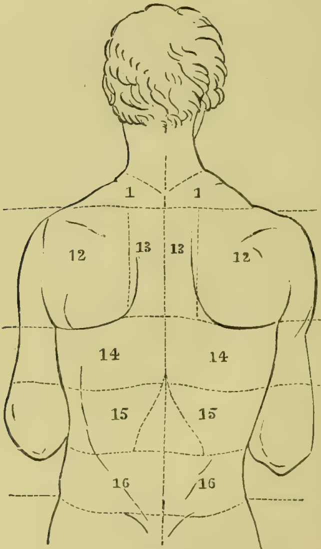

Fig. 23.

Within these limits is placed the upper lobe of the lung on both sides, close to the sternal border of the region. On the right side lie the superior *vena cava*, and a portion of the arch of the aorta. On the left side, close to the sternum, is the edge of the pulmonary artery. The inferior border of the region on the left side corresponds to a portion of the base of the heart; while part of the right auricle occupies the inferior corner of the region towards the sternum on the right side.

The Mammary Region (No. 3, Figs. 19, 20, and 22) has also a square-like form, and is bounded above by the line passing through the lower border of the *third* rib, where it joins the cartilages of the sternum below, by the line passing transversely on a level with the xyphoid cartilage (its upper border); outside by the vertical line passing to the outer tubercle of the pubes (on either side); and on the inner aspect by the edge of the sternum. The contents of the mammary regions differ on the two sides. *On the right side* the lung lies throughout immediately underneath the ribs. Its inferior border turns off almost at right angles from the sternum, at the cartilage of the sixth rib, whence its thin sharp border gently slopes outwards and downwards, so as to occupy the lower part of the region when the diaphragm is depressed. But when the diaphragm is elevated, the liver rises into this region up to the fourth interspace. The fissure between the upper and middle lobes of the right lung passes upwards and backwards obliquely across the region from about the *fourth* cartilage. The fissure between the middle and lower portion passes in the same direction from the *fifth* interspace. A portion of the right auricle, also a portion of the right and superior angle of the right ventricle, lie between the *third* and *fifth* ribs, close to the sternum, in the right mammary region. *On the left side*, at about the level of the *fourth* cartilage, the anterior edge of the left lung passes obliquely downwards, having abruptly left the edge of its fellow on the opposite side, so as to expose a free space of variable size for the heart (Fig. 24). The edge of the left lung thus reaches the *fifth* rib; whence it comes inwards and downwards to opposite the *sixth* rib or interspace, whence it finally passes nearly horizontally outwards and downwards into the lateral region. The anterior point of division of the lobes of the left lung lies about the fifth interspace, below the nipple. The left auricle and the left ventricle, with a small portion of the right ventricle about the apex, lie in the left mammary region, the apex of the heart generally lying immediately above the *sixth* costal cartilage (Figs. 22 and 24, the latter figure under the description of the heart, a little farther on).

The Infra-Mammary Region has a somewhat triangular form. Its boundary above is defined by the lower bounding line of the mammary region on each side; below, by the margins of the false ribs; inside, by the xyphoid cartilage or middle line; and outside, by the extension of the line from the acromial end of the clavicle to the outer tubercle of the pubes on either side. *On the right side* this region contains the liver, with the edge of the lung overlapping it in front, to a variable extent during full inspiration. *On the left side* the stomach and anterior edge of the spleen rise as high as the *sixth* rib in this region; and towards the inner portion of the region the left lobe of the liver lies in front of the stomach.

The Supra or Post-Sternal Region (or the jugular fossa) is a small hollow, bounded below by the notch of the sternum, and laterally by the *sterno-mastoid* muscles. The trachea almost completely fills this region; but on the right side the innominate artery lies at the lower angle; and in some persons the arch of the aorta reaches its lower border just at the notch of the sternum, where it may be felt pulsating. The region contains no lung.

The Upper or Superior Sternal Region comprehends that portion of the breast-bone which is superior to the lower border of the *third* rib. It covers the left and a small portion of the right innominate vein. The superior cava runs along its right edge; the ascending and transverse portions of the arch of the aorta; the pulmonary artery, from its origin to its bifurcation; the aortic valves near the lower border of the *third* left cartilage, at its junction with the sternum, or midway between the mesial line and the left edge of the sternum (the pulmonary valves being a little higher than these, more near the surface, and quite at the left of the sternum); and lastly, the trachea, with its bifurcation, on the level of the *second* ribs. The remains of the thymus gland, with areolar tissue and fat, lie in front of these parts, between the lateral pleural boundaries of the upper V-shaped portion of the anterior mediastinum, thus separating the edges of the lungs above towards their apices.

The Lower or Inferior Sternal Region comprehends the remainder of the sternum which lies below the level of the lower margin of the *third* rib. It contains the greater portion of the right ventricle, with the infundibulum of the pulmonary artery, and a small part of the left ventricle. The *mitral valve* is situated towards the upper end of this region, close to the left edge of the sternum, on a level with the *fourth* sterno-costal articulation; the *tricuspid valve* lies nearer the middle line, and more superficially. The edge of the right lung descends vertically along the middle line, and at the upper part of the region is a small portion of the left lung. Inferiorly, and deeper seated, is a portion of the liver, and sometimes of the stomach, while the line of union of the heart and liver corresponds with where the diaphragm intervenes.

The Axillary Region (Figs. 19 and 20, No. 7) extends from the apex of the armpit above to the line continued which marks the inferior border of the infra-clavicular region. In front it is bounded by the posterior border of the infra-clavicular region, and extends to the external edge of the scapulæ behind on either side. The region can only be brought into view by lifting the arm over the head, or by carrying it away from the side (as in Fig. 20). The region is hidden (as in Fig. 19) when the arm is at rest by the side. It comprehends portions of the upper lobes of the lungs, a great volume of lung-substance, and, more deeply seated, large bronchi.

The Infra-Axillary Region (Figs. 20 and 22, No. 8) is bounded above by the region already defined; anteriorly, by the mammary region; posteriorly, by the scapula; and below it extends to the margins of the ribs. It contains on both sides the lower edge of the lung, sloping downwards from before to behind. On the right side, also, is the liver, between which and the walls of the chest is interposed a thin layer of lung-substance during a full inspiration. On the left side is the spleen and stomach.

The Supra-Spinous Regions have the same boundaries as the superior fossæ of the scapulæ, and correspond to the posterior surfaces of the apices of the lungs (Figs. 21 and 23, No. 1).

The Infra-Spinous Region, sometimes called the Scapular (Figs 21 and 23, No. 12) is identical with the lower fossa of the scapula. It covers lung-substance.

The Inter-Scapular Region (right and left) lies between the inner margin of the scapulæ, divided into a right and a left region by the vertebral column, from the second to the sixth dorsal vertebra. It covers lung-substance on each side of the mesian line, the main bronchi, and bronchial glands. On the left is the œsophagus; and from the *third* or *fourth* vertebra downwards is the descending aorta. The bifurcation of the trachea takes place at the middle line between the two regions, but inclining rather to the right side (Figs. 21 and 23, No. 13).

The Infra-Scapular or Lower Dorsal Region is bounded by the continuity of the transverse line, which forms the inferior boundary of the infra-clavicular region, and which, being continued behind, crosses the inferior angles of the scapulæ and seventh dorsal vertebra. It extends below as far as the twelfth rib, corresponding to the transverse line, carried round, which formed the lower boundary of the *infra-mammary* region in front. As low as the eleventh rib lie the lungs. On the right side, from the level of the eleventh rib, extending downwards, is the liver. On the left is the spleen, occupying some of the outer portion of the region; while the intestines occupy some of the inner part of the region. Close to the spine, on the left side, is the descending aorta; and on both sides, close to the spine, is a small portion of kidney (Figs. 21 and 23, No. 14).

(B.) *Situation of the Organs in the Thorax.*

The *outer boundary* of each lung is marked by a line passing obliquely downwards and outwards from a little outside the centre of the clavicle towards the axilla, and vertically at a variable distance outside the nipple. Each lung rises from half an inch to an inch and a half above the clavicle, the relative height being unequal and variable. The inner margin of each lung passes downwards and inwards from the apex, and meets with the inner margin of the other lung at the middle line, at a point between the first and second cartilages, or at the second. The *inner margin of the right lung* continues vertically downwards along the centre of the sternum, or inclining a little to the left side, as far as the attachment of the xyphoid cartilage (Fig. 24). The *inner margin of the left lung* leaves the right at a point between the *fourth cartilages*, or a little higher or lower than this, and passes nearly transversely outwards for a short distance in the direction of the *fourth costal cartilage*. It then runs obliquely downwards and backwards in the course of a line drawn from the centre of the *fourth costal cartilage*, half an inch to one inch inside the left nipple, as low as the seventh rib (Fig. 24). The *lower boundary of the right lung* passes somewhat obliquely and then transversely from the attachment of the xyphoid to the sternum, across the cartilages of the sixth and seventh ribs backwards to the spine, which it touches on a level with the *tenth*, *eleventh*, or *twelfth* dorsal vertebræ (Fig. 23). The *lower boundary of the left lung* is a little lower than that of the right, and passes backwards from the point indicated on the seventh rib, to strike on the spine at a point usually a little lower than that on the right side. The *apex of each lung* lies beneath the anterior scalenus muscle and the subclavian artery. The apices are separated from each other by the œsophagus, the trachea, and the projection anteriorly of the bodies of the *first* and *second* dorsal vertebræ. The *base of the right lung* is hollowed by the projection upwards of the liver, which in the centre of the thorax ascends as high as the *fifth* rib or *fourth* interspace. The liver is also separated from the ribs by the expansion of the lungs between it and the thoracic walls. The *base of the left lung* may be also pressed upon by the left lobe of the liver; and it is always hollowed out, though to a less degree than the right lung, for the accommodation of the stomach and spleen, and, to some extent also, the left lobe of the liver.

The *heart* lies between the two lungs. The right auricle and a part of the right ventricle are covered by the right lung, the rest of the right

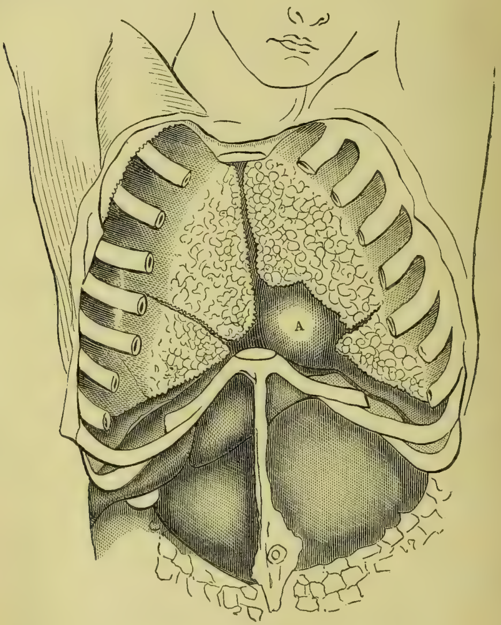

Fig. 24.*

ventricle being left bare by the divergence of the left lung from the middle line. The left auricle is covered by the right auricle and by the left lung. The left ventricle lies behind the right ventricle, but projects a little towards the left side, where it is uncovered for a short distance, beyond which its left border is covered by the left lung. The region corresponding to the uncovered portion of the heart (Fig. 24) is sometimes called the *præcordial* region or space. The *upper boundary* of this space is where the inner margins of the two lungs separate—namely, at the spot between the *fourth cartilages.* The *outer* boundary of the *præcordial* space is indicated by the diverging line of the inner margin of the left lung passing along the *fourth cartilage,* and then obliquely downwards, inside the left nipple. The *inner* margin of the space corresponds to the nearly straight inner margin of the right lung, behind the sternum, near the middle line. The *lower* boundary of the *præcordial* space is indicated by a line passing from the junction of the sternum to the xyphoid cartilage, directly to the left, or with a slight inclination downwards. Above this line is the right ventricle, and farther out is the apex of the left. Below it is the left lobe of the liver, and the stomach, separated only from the heart by the diaphragm and the pericardium. The *præcordial* region is thus slightly pyramidal in shape, its base being about two and a half inches long, and nearly horizontal. Its inner margin is about two inches long, and nearly straight; its outer margin is from three to three and a half inches long, and has a sloping direction from the apex to the pyramid (at the middle line between the *fourth* cartilages), to the outer extremity of the line indicating the base. This *præcordial space* corresponds to the left half of the lower portion of the sternum, and to portions of the cartilages of the *fifth* and *sixth* ribs. It may reach even to the junction of the cartilages with their ribs. Its inner and outer boundaries can be marked out only by light percussion; and the lower boundary can only be defined with difficulty by the same means.

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* Relative positions of the margins of the lungs to each other, to the thoracic walls, and to A, the *præcordial* region, comprehending the right ventricle of the heart, covered by its pericardium (after Dr. FULLER. See his work *On Diseases of the Lung and Heart*).

Below the boundary of the right lung the liver extends to the margins of the right false ribs, or a little below them. On the left side the space between the lower border of the lung and the false ribs, is occupied by the left lobe of the liver, the stomach, the spleen, and by a portion of intestine—the colon principally. The left lobe of the liver stretches across beneath the xyphoid cartilage, and below this to a variable extent towards the left side.

(C.) *Movements of Respiration and Changes in the Position of the Lungs.*

1. *In Health.*—(*a.*) During inspiration the lungs enlarge in all directions; the apices rise higher up, and the bases descend lower down; the points of union and of division between the inner margins are, in the one case, raised towards the apices, in the other case lowered down. The *præcordial* space is thus lessened in size by the advance of the anterior margin of the lungs. (*b.*) During expiration the state of things is reversed. The lungs fall from each other; but the point of division between the inner margins may be raised to a level with the *third* rib. The area of the præcordial space is increased. (*c.*) The difference between extreme inspiration and extreme expiration is considerable. In extreme inspiration the inferior boundaries of the lung are often from an inch to an inch and a half lower than they are in extreme expiration. (*d.*) The action of the heart causes

a slight difference. Each impulse presses aside the sloping inner margin of the left lung; but this is so instantaneous that it causes no appreciable alteration when the *præcordial* region is mapped out by percussion. (*e.*) During respiration the thorax enlarges in all directions by the movements outwards and upwards of the superior ribs and sternum, and by the movements downwards and outwards of the inferior ribs. *In women* the movements of the upper ribs are much greater than in men, while the abdominal movements are less. The difference is increased by the use of stays; but it does not appear to be altogether owing to these. In boys the costal movements are often considerable: in old age they are diminished.

2. *Changes by Age.*—In children the still considerably developed thymus gland separates the inner border of the lungs at the top of the sternum; and the point where they come in contact (converging from the apex) is lower than in adults. The lungs are also comparatively longer than in adults, and the inferior boundaries are lower down. In old people the lungs often alter considerably in shape, and produce corresponding alterations in position; the lower lobe, particularly in the left lung, becomes more posterior, and the upper lobes or lobe anterior. The lungs at last, in the old, become even larger above than below; and when mapped out by percussion, their several boundaries are found to have no certain and constant direction.

3. *Changes by Disease.*—One lung being incapacitated, the other lung undergoes supplementary enlargement; the inferior boundary is lowered, its inner margin is pushed to a variable extent across the median line over the heart, diminishing the *præcordial* space from the right or left side, according as the right or left lung is affected. So, also, if one lobe be affected, as by *pneumonia*, *tubercle*, or *cancer*, the other lobe, either upper or lower, as the case may be, is enlarged, and changes its position. In some diseases, as in emphysema, the lung enlarges; and if the emphysema is general, the lungs may meet each other almost at the top of the sternum; may not separate till on a level with the *sixth* rib; may leave the inferior margins at the *seventh*, or even the *eighth* rib; and may give a pulmonary percussion note in the posterior lumbar regions, below the ribs altogether. In cases of enlarged heart, or distended pericardium (unless there be coincident emphysema, or unless the lungs are floated forwards by *hydrothorax*), the lungs anteriorly are pushed aside, the point of separation of the inner margin is raised (especially in pericardial effusion), the inner margin of the right lung is thrown to the right side, and the inner margin of the left lung is thrown to the left side. Aneurism of the arch of the aorta, or tumors in the mediastinum, displace the upper portions of the lung; and tumors may even thrust aside and displace the heart. Abdominal diseases, as hepatic and splenic affections, peritoneal effusions, ovarian, uterine, or other tumors, also press up the thoracic organs, and alter their position. By such morbid states the inferior borders of the lungs may be not lower than the *third* intercostal space, or the *fourth* rib; the heart may be thrust upwards and outwards above and outside the left nipple. The student must also be prepared to meet with cases of more or less complete transposition of the viscera. (See a paper on this subject by Professor ALLEN

THOMSON, in the *Glasgow Medical Journal*, Vol. I.; also in the *Lancet* for August 8, 1863, by my colleague, Professor MACLEAN).

SECTION II.—SIGNS OF DISEASE FROM THE SHAPE OF THE THORAX.

The two halves of the thorax are seldom symmetrical. The right side in the most healthy persons often measures from *half an inch to an inch* MORE than the left; the right *infra*-clavicular space (particularly in right-handed persons engaged in laborious occupations) is apt to be more prominent than the left. The *fourth*, *fifth*, and *sixth* left cartilages often project more than the right; the *infra*-mammary and *infra*-axillary regions may be larger on the left than on the right side; the *infra*-scapular region, on the contrary, is usually larger on the right side, or it may be markedly prominent on both. The nipples (which in adult males are above the upper margin of the *fourth* rib, near its junction with the cartilages) are always equidistant from the middle line, but the left is somewhat lower than the right. Any marked changes beyond those now noted indicate disease either of the spine, as in *curvature;* or of the spine and ribs, as in *rickets;* or of the organs in the thorax. If *spinal curvature* and *rickets* are determined not to be present, any general expansions of one side as compared to the other, or general contraction, or (what is much more common) local expansion or bulging, or local contraction, depression, or excavation, become important signs. The existence of such conditions is ascertained by "*inspection;*" and accurately, by measurement with callipers or with tapes, or by ingeniously devised instruments which have been named "*stethometers*" or "*chest-measurers;*" and by the "*cyrtometer*"—an instrument which may be improvised by two pieces of common composition (bendable) gas pipe, or by two bands of copper or soft tin, joined at the ends by a piece of india-rubber tubing as a joint, which is to be applied over the spinous processes, and held firmly in position, while the two pieces of pipe, copper, or tin bands are brought round and accurately moulded to the sides of the chest (FINLAYSON, l. c., p. 462). The joint allows the arrangement to be removed without destroying the "set;" and being adjusted on a sheet of paper, a tracing of the form can be made with pen or pencil. General expansion of the thorax is usually produced by effusions of fluid into the pleura, or by extreme cancerous infiltration, or by emphysematous lungs. Much more rarely chronic consolidation will cause general or partial *bulging*. Tubercular deposition, at its earliest period, has been said to cause some degree of local prominence; but *retraction* usually follows more or less complete impairment of the functions of the lung, or of a part of it, as in softened tubercle or cancer, and in the period of absorption of pleuritic fluid which has firmly compressed the lung. Heart diseases and aneurisms of the aorta may cause local bulging, while tumors may produce both bulgings and retractions according to circumstances (*MS. Notes* of the late Dr. PARKES' Clinical Course at University College).

SECTION III.—PHYSICAL EXAMINATION OF THE CHEST.

I. By Simple Inspection of the Form of the Thorax.—It is necessary to note,—(*a.*) Its general shape; and especially as to the condition of the *supra-* and *infra-*clavicular spaces in respect of flatness, fulness, retraction, or bulging in these regions; the condition of the hollow above the notch of the sternum (the supra-sternal space); the form of the clavicles and their curvature; the height and breadth of the shoulders; the form of the sternum, as to whether or not it bends outwards or inwards; the curves of the spine; the position of the scapulæ; prominence of their inferior angles, and the firmness or laxity of the *latissimus dorsi* muscle; the distance of the scapulæ from each other and from the middle line; the contracted contour or expanded form of the lateral regions; the width, depression, or bulging of the intercostal spaces; and distance of the nipples from the middle line. (*b.*) It is necessary to obtain a *general notion as to capacity or size of the thorax, relative to the height, weight, and age of the individual, allowing for fatness or emaciation* (Spirometry; see previous pages, under "*Scrofula,*" Vol. I., p. 1000). The object of spirometry is to measure the amount of air received into the lungs, so as to learn what the lungs can do. Practically, and independently of all instruments devised for this purpose, one of the best modes to determine what the patient can do with his lungs is to make him count *one, two, three, four,* &c., on till he has no more breath left in him to count with, and so requires "*to fetch a breath.*" By practice, especially if a singer, he may now and then count more than he did on the first attempt; but every one has a number which can be reached normal to one's self, when the lungs are efficient in capacity. (*c.*) Observe the thoracic movements, and estimate in seconds the time taken to complete the inspiratory and expiratory acts; compare the movements of the two sides of the thorax, and also the abdominal respiratory movements with those of the thorax, so as to notice if either takes an undue share in the work of respiration. If the ribs scarcely move, and the parietes generally of the thorax remain at rest, while the surface of the belly rises and falls alternately with the respirations, the act is called *abdominal respiration,* because the abdominal muscles take the larger share in its performance. If, on the other hand, no motion of the abdomen is visible, the act of respiration is *thoracic.* Observe whether the whole acts of respiration are quicker or slower than natural—*i.e.,* more or less than *eighteen* to *twenty* per minute, or one to every four arterial beats. Note their frequency per minute. Observe whether they are calm, easy, and fully drawn, or short, forced, hurried, and incomplete attended by indications of pain, checked or partially arrested by cough Notice whether respiration is performed through the mouth or nose, o both; and whether the *nares* dilate and contract at each respiratory effor with any constrained movement of forcible expansion. Note any sensib odour or vapour of the breath, and its temperature. The movements a the apex of the lungs should be closely observed, by a combination inspection and palpation. "The observer stands behind the patient wi a thumb on each scapular spine, while the tips of his fingers lie over t apices in front immediately beneath the clavicles. The patient is th directed to inspire fully and quietly, when by watching the rise and fall of the fingers, a very accurate comparison of the two sides can be made" (FINLAYSON, l. c., p. 467). Deficient expansion at either apex indicates lung disease.

II. **By Measurement.**—The use of tape or callipers in deep and medium inspiration and expiration will detect any differences between the two sides, or undue differences in the size of the chest at different times. There is about one inch of an average difference in favour of the right side of the chest compared with the left, which is consistent with a normal state. A convenient plan is the double tape, suggested by Dr. Hare. It is formed by joining two common measuring tapes together, so that the beginning of each may be in the centre of the tape when joined. By putting the line of junction of the two tapes upon the spine, and holding it there, the ends of the tape can be carried round the body at any point, and the circumference of each side read off simultaneously. By taking the size at full expiration and full inspiration, the extent of *expansion* is determined, as well as the absolute and relative size of each half of the thorax. The level most commonly selected is about two inches below the nipple, in a line with the sterno-xyphoid joint—marking the middle line of sternum with ink and corresponding vertebra behind. The *general expansion* of a side of the chest is best learned by the use of the double tape, or by the use of some of the "*chest-measurers*" about to be noticed. The difference in the measurement between the fullest inspiration and the fullest expiration gives the general expansion of the lung. In health both lungs expand nearly equally from *three-quarters of an inch to an inch and a half*, or the right may expand a little more than the left. If there is any deficiency of expansion, there must be disease of some kind, but the nature of the disease cannot be known without additional signs. *Local expansion* is most accurately determined by the eye, the hand, and the chest-measurers of Drs. Quain, Sibson, or Leared; the exact levels of the measurements being always noted. All these instruments essentially consist of dials, with indices, moved by mechanism, connected with tapes passing round one or both sides of the chest—so many revolutions of the index indicating on the dial so many tenths and hundredths of an inch of expansion of the chest. Dr. Leared's instrument indicates differences on the two sides (*Med. Times and Gazette*, August 2, 1862); and Mr. Henry Thomson, of University College, has also suggested a simple addition to the tape measure, by which it is made differential. With the "*spirometer*" of Dr. Hutchinson, the "vital capacity" of the lungs may be determined; and with the "*stethograph*" of Dr. Ransome, tracings may be taken of the movements of the chest walls. His "*chest-rule*" notes accurately the exact position of any pulsation or dulness. The measurements (besides the circular) which are most useful are the distance of the nipples from the middle line; their distance from the sterno-clavicular articulations of each side; and the distance of the centre of the clavicle from the lowest point of the false ribs in the vertical line. The following numbers denote the movements of various parts of the thorax in health:—(1.) The sternum and the first *seven* ribs in tranquil breathing advance forward imperceptibly. The left *fourth, fifth,* and *sixth* cartilages, and the *sixth* rib, move less than on the right side, on account of the position of the heart. In forced inspiration the movement forwards is from *half an inch* to *two inches.* (2.) The expansion of the *eighth* and *tenth* ribs during deep respiration is increased, but is less than that of the first five ribs. (3.) The abdomen moves forwards in tranquil inspiration from ·25 to ·3 inch. In deep inspiration the movement amounts to about *one inch.* There are, however, very great variations in different persons; and "*chest-measurers*" must all be submitted (as *percussion* and *auscultation* are) *to the test of comparison between the two sides*—the difference which the heart causes between the two sides being remembered (PARKES, *MS. Notes*). It is necessary in all these observations so to divert the patient's attention as to cause him to look away from the instruments, otherwise the movements of the chest may become so affected as to vitiate the results.

III. **By Palpation**, or the application of the hand, more extended information is obtained than by simple inspection—such as of the intercostal distances, lateral and antero-posterior; expansion of the chest in the acts of respiration; appreciation of *vibrations* communicated through the walls of the thorax. The palm of the hand applied to the chest of a *healthy* person while he is speaking will appreciate a delicate *vibratile tremor* (commonly called *fremitus*), more marked according to the graveness, coarseness, and loudness of the speaking voice. The intensity is generally greater in front than behind, on the right side than on the left, and stronger towards the sternal than the humeral halves of the region below the clavicles. In *disease* the *fremitus* on the two sides must be compared. It is usually *increased* by whatever consolidates the fine vesicular texture of the lung, without obliterating the bronchial tubes. It is *diminished* by the intervention of liquid or air between the lung and the thoracic walls; also by such extensive consolidation of the lung-substance as to fill up the smaller bronchial ramifications leading to air-vesicles. *Vocal fremitus,* or *vocal vibration,* is observed by placing the hand over the surface of the chest of a person speaking. It is a delicate vibration, easily deadened by too forcible pressure of the hand. It is more marked in adults than in children, and in males than in females. It is more intense in long-chested than in short-chested persons; markedly so in thin than in fat people; and stronger in recumbency than in the sitting posture. A *rhonchial fremitus* may be heard, when certain *rhonchi* throw the bronchial tubes into vibrations sufficiently strong to be felt on the surface of the chest. The sibilant, sonorous, and mucous rhonchi have all this property. *Friction fremitus* may be felt by the hand, when the gliding motion of the pleural surfaces upon each other is attended by a perceptible vibration, by the collision and friction of plastic matter upon the surfaces.

Ordinary fluctuation may sometimes be detected by the hands, combined with shaking or suddenly altering the position of the chest, as by *succussion.* The hand is also used to appreciate the action of the heart. After full *expiration*, it is felt to beat between the cartilages of the third and fifth ribs, and at the neighbouring part of the sternum, generally immediately below and to the outside of the left nipple. After a full inspiration it may be felt as low as the sixth rib. The frequency of the respirations may be ascertained by the hand, applied to the surface *below the clavicles* in the female, and *below the epigastrium* in the male (WALSHE).

IV. **By Percussion.**—The position of the organs is ascertained during life principally by the signs derived from *percussion;* that is, from the nature of the sound produced by striking over them either "immediately," as when the chest or abdomen is gently tapped with the finger or a light hammer; or "mediately," as when a flat body is placed upon the chest or abdomen, which is then struck by the finger or the hammer. The main object of percussion is to determine the comparative density of subjacent parts. *Mediate* percussion is employed almost to the exclusion of *immediate.* The body which is interposed between the hammer and the skin is called a "*pleximeter,*" and consists simply of a flat piece of ivory or wood, or india-rubber, or the index finger of the left hand laid flat upon the chest. The hammer is made of a piece of whalebone, or of light steel, armed at its head with a piece of india-rubber; or the flat edge of the stethoscope encircled by a piece of india-rubber; or it consists of a light thimble covered with vulcanised india-rubber; or the points of the index or middle fingers of the right hand may be used for the purpose of a hammer, the nails being cut close. The fingers give the most delicate results.

Mode of Percussing.—The pleximeter is to be placed flat upon the chest, and held close to the walls. It is then to be struck perpendicularly, and with gentle or moderate force. If the fingers of the right hand be used as the hammer, the blow should be given from the wrist only, the elbow and shoulder being quite motionless. The force of the blow must vary according to circumstances. In the supra-clavicular space the finger, if it be used as a pleximeter, should be pressed closely into the space, and the blow should fall in the direction of a line passing to the centre of the chest—namely, downwards, forwards, and inwards. If the blow be directed too much forward or backward, it strikes on the clavicle, or parts at the back of the neck, and the "pulmonary percussion note" is not obtained. The clavicles are best percussed *immediately,* first at the sternal, and then at the acromial end. In the *supra-spinous* spaces the blow should fall downwards, inwards, and forwards. Over the rest of the chest the pleximeter may be placed in any direction, provided that corresponding points shall be struck on the two sides, as a slightly different note is given out, especially in thin people, by the intercostal *spaces* and the *ribs.* If the *spaces* are struck on one side, they should also be similarly, or with like force, struck on the other. The "*pulmonary percussion note*" is the sound given forth by striking on a part of the chest-wall below which lies a considerable depth of lung. The sound produced does not depend upon the proper tissue of the lungs, but upon the vibrations of the air they contain, and also upon the vibrations of the walls of the chest. The pulmonary substance vibrates but little; it is non-sonorous, and acts rather as a damper of sound. The qualities of sound to be noted are—(1.) *Amount in intensity of resonance,* commonly called *clearness;* (2.) *duration of the sound;* (3.) *pitch;* (4.) *volume of sound.* The intensity of sound is diminished by the lessened vibration of the walls of the chest, as in pleuritic effusions; or by the lessened vibration of the air, the air being lessened in amount; or by increase in volume of the lung-substance, as by exudation. The physical conditions which lessen the quantity of air are—(1.) Compression of lung-substance from any cause, such as exudation, tubercle, great congestion, or œdema; (2.) compression of the lung from fluid in the pleura, or by tumors. When the pulmonary percussion note is exaggerated, it may become what is termed "*tympanitic*," that is, a very clear sound, of less volume, but of higher pitch than the pulmonary percussion note. It is similar to the sound obtained by striking the abdomen when the intestines contain air, or a bladder moderately distended with air. It is generally caused by an increased quantity of air in the lung-tissue or in the pleura, as in pulmonary emphysema, or when cavities exist in the lungs, or in pneumo-thorax. A very thin and flexible wall will often give a tympanitic character to the pulmonary percussion note. There are some exceptions to these statements, namely—(1.) When the air is not apparently in excess, a thin layer of sound lung full of air, lying over hepatised lung, will give forth the sound; (2.) the upper lobes of the lung when hepatised in the lower lobes will also give forth the sound; and, (3.) when the lung floats on the surface of pleuritic fluid. *Tubular* sounds are produced when the lung is consolidated, covering bronchial tubes. In pleurisy the intensity of sound is diminished from both causes. The sound is *intensified or made more clear* by whatever increases the vibrations of the walls of the chest, as in thin children, or in persons becoming thin, and by increased vibrations of air in the lungs. The sound elicited by percussion of the chest varies with the region explored. The clearest sounds are obtained from the anterior regions, where the walls are thin; the least clear from the posterior, on account of the thickness of the walls. The degree of clearness also varies in the several *anterior* regions of the chest. *In the supra-clavicular region* the resonance ought to be very clear, approaching a tympanitic sound over the clavicle, where the sound ought to be clearest in the middle third; of high pitch in the inner third; but duller towards the scapular end. *In the infra-clavicular region*—between the clavicle and superior margin of the fourth rib—the sound ought to be very clear, and typically pulmonary; less clear and shorter, but of higher pitch, on the right than on the left side. *On the right side*, from the fourth rib downwards to the sixth or eighth (where the liver is reached), the sound on strong percussion is less clear, till it abruptly becomes flat. *On the left side*, from the fourth to the sixth rib, and from the nipple to the sternum, the sound is dull, from the presence of the heart; except in cases where the lung overlaps the heart in inspiration: where the heart is small, and where emphysema of the lungs exists. Below these points, on the left side, the sounds of the chest anteriorly are variously modified by the encroachment of the left lobe of the liver, or by the spleen, or by the distension of the stomach, over which the sound may be tympanic, from gaseous distension; and dull on the inner and outer parts of the region over the more solid organs just named. *Laterally*, when the arms are raised, the axillary regions on both sides are highly sonorous. *On the right side*, the right infra-axillary region is clear to near the sixth or eighth rib, when it becomes suddenly dull over the liver, the line of hepatic dulness varying during inspiration and expiration. *On the left side*, the sound may be more or less dull, from enlargement of the spleen; but when the stomach is distended with gas, it may be tympanitic. *Posteriorly*, the scapular regions give a clear sound, though much less clear than the corresponding anterior and lateral regions. The *infra-spinous* region is more sonorous than the supra-spinous. The *infra-scapular* region (especially over a portion of the bend of the ribs uncovered by muscle when the arm is extended) gives a very clear percussion sound. *On the right side* BEHIND, the line of hepatic dulness is at or near the eleventh rib; but strong percussion will elicit a flat sound an inch or more higher up. *On he left side* BEHIND, there may be tympanitic resonance, from gas in the stomach, about the seventh rib; and, about the ninth rib, dulness from the spleen. In the *interscapular region* clearness and duration of sound are lessened from the increased resistance of the mass of muscles.

V. **By Auscultation** is denoted "the act of listening," and is pulmonary or cardiac according as the sounds listened to relate to the lungs or to the heart. For this purpose the ear may be applied to the surface directly, merely interposing (for obvious reasons) a fine towel or thin piece of inen or calico between the chest of the patient and the ear of the observer. This is *immediate* auscultation. When a conducting medium—as an instrument called the *stethoscope*—is placed between the ear of the istener and the chest, through which the sounds produced within the chest walls are transmitted, the auscultation is then said to be *mediate. Mediate auscultation* is especially necessary when the sounds to be heard are limited to a small and definite region, as in listening to the sounds of the heart. The ear-piece of the stethoscope ought to fit the ear of the observer; or the ear-piece may be removed, so that the end of the stethoscope may be introduced into the orifice of the ear. It is important that the ear be well fitted. The extremity applied to the patient should be about an inch and a quarter in diameter, or less in some places.

Two stethoscopes joined together by flexible stems leading separately to each ear (double or bin-aural stethoscope) is an arrangement of great value in the differential diagnosis of sounds associated with lesions in the thoracic organs, especially when the sounds are obscure from their lowness. The ear-pieces are small, and fit into the ears. These double instruments seem to be efficient magnifiers of low sounds; and extremely useful in cardiac diagnosis when the action of the heart is rapid or irregular. When the first sound is indistinct at the apex, or cannot be identified with the apex beat, or when the second sound is indistinct, or when it is audible only at the base, the first sound being audible only at the apex, the difficulty of recognising the two sounds is very considerable. It is in such circumstances that the differential stethoscope devised by Dr. Scott Alison is of great value. It enables the sounds to be identified at the points where they can be heard best; and brings them into accurate relations with each other, because of the two ears receiving at the same ime the sounds from two points of the *præcordial* region (GAIRDNER). Another form of a differential stethoscope has been devised by Dr. Spencer, which he calls the "compound" stethoscope, in which the sounds rom each tube are communicated to *both* ears (*Brit. Med. Journal*, March 8, 1874). In the original *bin-aural stethoscope* the stem, arising from the ell end, or conical end, divides into two branches, the ends of which espectively fit into either ear. Both ears are thus engaged in listening o the sounds emanating from one and the same spot or area, which re thereby intensified. In the *differential stethoscope* of Scott Alison the tubes are separate throughout, with a separate ear-piece for each ear; and each terminating in a bell expansion, each of which can be applied to different parts of the chest. Dr. Henry Hilliard has also devised such a stethoscope (*Med. Times and Gazette*, 1875, p. 543). In the "compound" stethoscope of Dr. Spencer, the sounds from both bell expansions are communicated to both ears.

The forms of single and double stethoscopes are now very numerous, and all of them require much practical study and experience to use them with success. The ear-piece should fit the ear exactly in all forms of stethoscope; "and therefore, when a well-made stethoscope has been selected, and an ear-piece chosen which fits the ear comfortably, the student should keep to that one, and familiarise himself with its use" (Fuller).

The main object of the stethoscope is to *circumscribe* and *localise* the sounds; the chest end of the instrument should therefore be as small as possible, in order the better to appreciate the precise seat of their greatest intensity. To ascertain this, the instrument should be "worked towards the sound," and then "from the sound," right and left, up and down, till its end is on the exact spot whence the sound proceeds in its greatest intensity. By working the stethoscope round and round, and gradually contracting the circle, the *area* of the sound can be ascertained: and hence so far the lesion producing it can be localised (Hyde Salter). Several circumstances affect the quality of a stethoscope:—(1.) *The material which allows the least amount of sound to be lost, and least of all perverts or modifies the sound*, is the best for a stethoscope—namely, some *porous* wood which is a good conductor of sound. Cedar and deal are the best woods for the purpose. The denser the wood the more are the sounds apt to be modified; therefore, ebony is to be condemned. (2.) *The stethoscope should be of one piece of wood*—turned out of a solid block. It ought not to be part ivory and part cedar. (3.) *As to length and thickness*, the length most convenient is that which permits the instrument to be carried in the crown of one's hat. In military life, where hats are not accommodating in this respect, and in the regulation absence of pockets, Dr. Veale, the Assistant to the Professor of Medicine at Netley, has devised a convenient form of stethoscope suitable for the necessities of his military medical brethren (*Lancet*, Nov. 29, 1879). It may be solid if the wood is very porous; but generally it is hollow. Solid stethoscopes carry sharp sounds and musical notes with great intensity; but they do not transmit the respiratory rustles and other feeble and unmusical sounds so distinctly as the hollow stethoscope, which is the preferable instrument for common use. The length of the instrument is important, as differences in length cause certain sounds to be more or less distinctly audible; as the hollow tubes consonate, according to their length, with certain definite notes and their harmonics (Bristowe). (4.) *The chest end* should be small, not larger than *one inch and a quarter* in diameter; because the smaller it is, the greater is its localising power, and the narrower are the limits within which the seat of any particular sound can be determined. The chest end of the stethoscope should also be narrow, and smoothly rounded over the edge. (5.) *The ear-piece* should be *large* and *flat*, or slightly concave to receive the concha, to secure apposition and occlusion; but in this respect each man must fit his own ear (HYDE SALTER, "On the Stethoscope," *Brit. Med. Journal,* January 31, 1863). By stretching evenly and tensely over the pectoral extremity or bell of the stethoscope a piece of india-rubber, the friction caused by much hair developed on the chest may be avoided (FINLAYSON); and if a piece of parchment is similarly placed, so as to be in contact with the skin, and sufficiently firm and elastic to compress the tissues and transmit sonorous vibrations, it intensifies the sounds, and their source is more circumscribed. In the double stethoscopes it lessens the roaring. To auscultation of the larynx and trachea it is peculiarly adapted, as well as to that of the heart (HOGEBOOM, *New York Med. Journ.,* Vol. III., 1866).

Immediate auscultation ought to be learned and practised by the student in every case where it is practicable; although, for reasons of delicacy, fastidiousness, or filth, on the part of the patient, it may be necessary to have recourse to the stethoscope. In many suitable cases the chest can be more quickly and quite as satisfactorily explored by *immediate* as by *mediate* auscultation. *Auscultation in children* (who are apt to be frightened by the appearance of an instrument) is in general better effected by the ear directly applied to the chest. In such *immediate auscultation* the ear should be firmly and accurately applied to the surface, the pressure not being too light or too strong. Very young children must be held in the nurse's arms, so that the chest may become prominent, and the ear of the examiner be conveniently applied. The manner of breathing should be first noted, whether by the mouth or nose, abdominal or thoracic. Vocal resonance can best be estimated when the child is crying —the cry replacing the voice. The maximum intensity of respiratory murmur is, *anteriorly,* from the clavicle to below the nipple, on the right side, and not quite so far down on the left; *posteriorly,* the inferior part of the interscapular region; the minimum as in adults.

In the practice of auscultation, whether mediate or immediate, both patient and physician should be placed in positions of least restraint for both. The chest should be free of all clothing, except it may be a thin cotton towel or chemise. The anterior regions are best explored while the patient sits in a chair, the arms hanging loosely at the sides. To explore the posterior regions, the body should be slightly bent forward, with the arms slightly crossed. For auscultation of the lateral region, the arms ought to be raised and crossed over the head. If the patient cannot leave the bed, he may sit up in bed, or be turned over from side to side, according to the part to be explored. The examiner must not stoop nor bend his head too much; the stethoscope being evenly and closely applied to the surface of those parts of the chest to be examined, and not with too much pressure. Special care must be taken that it is not tilted up, so that air may not enter and move from below upwards; and also that a fold of dress, or any such thing, may not rub against its stem.

To become familiar with the knowledge to be acquired by means of auscultation, much time and labour must be devoted to its practice, alike on persons in health and in disease. It is necessary that the student should hear well. The "*Audiometer,*" of Professor Hughes, for the measurement of hearing, will accurately test his powers in this respect (*Proceed.*

Royal Soc., No. 196, 1879). A verbal description of the sounds to be learned is difficult, because the impressions made on the senses of one person cannot be communicated exactly by language to another; and the distinctions which subsist between the sounds heard in health and those in disease are not yet regarded as similarly significant by all, nor is their individual importance yet clearly determined. The examiner must learn, first, to appreciate the *natural respiratory and vocal sounds,* so that he may be able to detect such *modifications* or *changes* of them which amount to physical signs of disease. The *healthy vesicular murmur* of the lungs is "a diffused, soft, breezy, sighing sound, not to be described, whose probable site of production is the air-sacs of the lungs" (HYDE SALTER). It is divisible into two sounds, inappreciably separated from each other—the *sound or murmur of* IN-*spiration,* and the sound or *murmur of* EX-*piration.* The EX-*piratory murmur* in some is shorter, less intense, of lower pitch, than the IN-spiration on the *left* side. In some it is said to be altogether absent on that side. The duration is increased in old people, while its intensity is less and more prolonged in the right clavicular region than in the left (GERHARD, LOUIS). The *intensity of the vesicular murmur* varies in different healthy persons of the same age, sex, conformation, and apparently similar condition. It is loud and well-marked in infancy and childhood—so loud as to be distinguished by the distinctive name of "*puerile respiration.*" It becomes more subdued in adult age; and in old age it is frequently very feeble, and known as "*senile respiration.*" Generally, it is greater in the female than in the male in the upper regions of the chest. When the stethoscope is placed over the suprasternal fossa, a sound like that produced by air driven forcibly through a tube is heard, and may be divided into two times, one coincident with inspiration, the other with expiration, and separated by a brief interval. The quality of both sounds is peculiar and characteristic, and is said to be *tubular;* the *inspiratory* sound is of higher pitch than that of vesicular respiration, and the *expiratory* is intenser, longer, and higher in pitch than the inspiratory; this is *tracheal respiration. Laryngeal respiration* is said by many writers to differ greatly from tracheal respiration; but the difference is limited, as a general rule, to intensity; in other respects they are essentially the same (FLINT, *loc. cit.*, p. 131).

Auscultation of the Voice.—It is important to attend to the sounds of the voice as they are transmitted through the chest. Modified by the size of the tubes, and the nature of the substance through which they pass, they become signs of the condition of the organs transmitting them. In vocal auscultation the bin-aural stethoscope or the naked ear should be used, and the patient instructed to count *one, two, three,* slowly, distinctly, and moderately loud, and to repeat these numbers as often as is necessary. Over the trachea the voice seems concentrated and coming into the ear of the explorer: it is more or less distinct, resonant, and gives the sensation of a peculiar shock and fremitus; this is called *tracheophony.* The phenomenon of the direct entrance of articulated words into the ear at the point auscultated is called *pectoriloquy.* When the stethoscope is placed over the thyroid cartilage, and the patient is directed to speak, the voice will be found to be transmitted generally with less intensity, shock, and vibration, than from the trachea; this is named *laryngophony*

On listening over the chest, either *immediately* or *mediately*, to the sound of the voice, articulated words are not heard, and the resonance is less intense than over the trachea, is more diffused, seems farther from the ear, and usually the shock is wanting. Over certain parts of the chest, in many instances, there is no fremitus; but differences exist in different persons, and in the several regions, and in the corresponding regions of the two sides. Over the first divisions and subsequent larger subdivisions of the trachea—the larger bronchial tubes—as on each side of the upper part of the sternum, at and between the scapulæ, and in the axillæ, there is still considerable resonance, though diffused and distant, and frequently some fremitus; this is natural *bronchophony* or bronchial resonance (CLYMER, *Physical Exploration of the Chest*, 2nd Ed., p. 147).

Auscultation in Disease.—The modifications of the *respiratory murmur* in diseased conditions of the lungs are changes in its *duration* and *intensity*, *rhythm*, and *special character*. Its *duration* and *intensity* may be—(1.) Exaggerated or puerile; (2.) weak or senile; (3.) suppressed. Its *rhythm* may be altered to—(1.) Incomplete; (2.) jerking; (3.) divided; (4.) prolonged in the expiration. Its *character* may be—(1.) Harsh; (2.) bronchial or blowing; (3.) cavernous; (4.) amphoric.

Duration and intensity may be, (1.) *Exaggerated* (called also *supplementary* (ANDRAL), *puerile* (LAENNEC), and *hypervesicular respiration*), is simply increased intensity or loudness, without change in *quality*, *pitch*, or *rhythm*. It has been stated that the degree of the respiratory murmur varies in different persons, and in the same regions of the two sides of the chest; and this should be remembered in estimating its value as a sign of disease.* When accompanied by a greater number of respirations than natural, decidedly heightened in the anterior and superior regions, or diffused over one side, it is indicative of disease in a portion of the lung more or less distant, or of the opposite side. It is most frequently met with in solidification of the lung, as in pneumonia, large tubercular deposits, carcinoma, pulmonary apoplexy, or where a considerable portion of lung is deprived of air from a mechanical cause, as the pressure of an enlarged bronchial gland or air-tube, blocking up of the bronchi from plastic exudation, and in pleurisy. *Duration and intensity may be*, (2.) *Weak*, and known as *senile respiration* (indicated by diminished intensity or feebleness of the respiratory murmur, its other characters remaining the same), happens in a number of pulmonary disorders, and is a sign of much value, often marking the site of the lesion. It may be close to the ear (*superficial*), or more or less distant (*deep-seated*). It is generally persistent, though it may be intermittent. The expiratory sound is rarely heard, except in emphysema, where, along with feebleness of the murmur, it is often distinctly prolonged. The murmur may be actually diminished in degree from imperfect production, or the effect may be caused from its reaching the ear through some solid or liquid between the lung-surface and the chest-walls. It is caused by—(1.) An obstruction in the air-tubes, as in laryngeal disorders or foreign bodies in a primary bronchus, narrowing of the bronchi from inflammation and its products, spasms or permanent contraction of the air-tubes, and their compression by tumors, enlarged bronchial or lymphatic glands; (2.) Obstruction of the air-cells, in pulmonary tuberculosis, œdema, pneumonia, hæmorrhage; (3.) Over-distension of the air-cells, in emphysema; (4.) Gaseous, liquid, or solid effusions into the pleural sac; (5.) Impaired thoracic movement caused by pain, as in acute pleurisy and intercostal neuralgia, or from paralysis of the respiratory muscles, as in general paresis and hemiplegia. When weak respiration is limited to the apices of the lungs, and is accompanied by diminished resonance, it generally denotes tubercles; heard in the anterior, superior, and middle regions, with increased resonance, it is a sign of emphysema; and at the base of one or both lungs, and remote, with more or less dulness on percussion, it indicates pleuritic effusion. *Duration and intensity of respiration may be,* (3.) *Suppressed,* as in the entire absence of the respiratory murmur. It may be permanent or intermittent; and is met with in the same pulmonary conditions as weak respiration. It is chiefly valuable as a sign of pleuritic effusion, single or double.

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* According to Walshe and Fournet, when exaggerated respiration is a sign of disease, the excess of intensity is on the side of expiration, whilst in health it is on the side of inspiration.

The rhythm of respiration may be altered by being—(1.) *Incomplete,* when there is lessened duration of the inspiratory murmur, and is of two kinds: (*a.*) When its beginning is abridged (*deferred inspiration*), and it is not heard until an appreciable interval elapses after the commencement of the inspiratory act and the air-cells are fully distended, and then as a short wheeze, as in emphysema, bronchitis, and pleuritic effusions. (*b.*) It may be initially evolved, but abruptly stopped before the inspiratory act is ended, with a sort of hitch (*unfinished inspiration*). Its site is in the bronchioles. *The rhythm of respiration may be altered to,* (2.) *Jerking, interrupted, wavy, cogged-wheel respiration* (*inspiration entrecoupée, respiration saccadée*), when the respiratory murmur, instead of being continuous, is broken and whiffy. When partial, it is the result of incomplete dilatation of the air-cells from some cause or other. Its site is almost always the apex of the lung anteriorly, and oftener on the right than on the left side. It is thought to be a valuable sign of incipient phthisis. When general it is due to sudden arrest in the dilatation of the chest walls from pain or deficient innervation, as in dyspnœa, Cheyne-Stokes respiration, asthma, pleurisy, pleurodynia, palsy. It is also met with in pleuritic adhesions. *The rhythm of respiration may be altered by being,* (3.) *Divided,* when a distinct interval elapses between the inspiratory and the expiratory murmurs. It is caused by over and permanent dilatation of the cells hindering the expulsion of the air, and is usually heard in the middle regions anteriorly, as in emphysema. *The rhythm may be altered, by* (4.) *The expiration being prolonged,* "when it is the only or chief alteration in the respiratory murmur, and there is no change in degree or pitch: it is a diagnostic sign of much significance. Natural prolonged expiration happens in a certain number of persons, but only on the right side. Its value as a morbid sign is measurably coincident with its site. When heard over a limited space in the upper chest regions, it may indicate tuberculosis; when more general, and heard in the middle regions, emphysema; or it may indicate only temporary obstruction or compression of the air-cells" (Clymer).

The character of the respiration may be, (1.) *Harsh, rude, broncho-vesicular* (Flint), *vesiculo-bronchial* (Da Costa) *respiration,* "when it has less of the vesicular quality than the healthy respiratory murmur, being less soft, of higher pitch, more blowing, and not so equable. *In inspiration* the vesicular and tubular qualities are united, the duration short, the pitch more elevated, and the intensity variable; *expiration*, sometimes wanting, is, when present, always prolonged, of higher pitch, and sometimes of greater intensity than inspiration, to which it succeeds after an appreciable interval (FLINT). It denotes a certain amount of pulmonary condensation, from deposition or compression, and happens in phthisis, pneumonia, pulmonary apoplexy, cancer, fibroid degeneration, melanosis, œdema, and in pleural effusions of a certain amount. For its presence, the degree of condensation of the lung should not be sufficient to abolish vesicular respiration, which must to some degree, however slight, be heard in inspiration." *The character of respiration may be*, (2.) *Bronchial* or *blowing*, "when it is in all respects the same as natural laryngo-tracheal respiration, and in certain pulmonary disorders takes the place of vesicular respiration. *Inspiration* is tubal, of short duration, incomplete, and of high pitch. *Expiration* is nearly or quite as long as inspiration, and sometimes longer, of greater intensity, and higher pitch. From the sudden interruption of the inspiratory murmur, a distinct interval occurs between it and the beginning of the expiratory. It is always associated with condensation of the lung-tissue, and is heard in phthisis, pneumonia, pleurisy with effusion, uniform dilatation of the bronchi, with induration of surrounding tissue, &c. It may be *diffused* or *tubular*." *The character of respiration may be*, (3.) *Cavernous*, "when it resembles the sound produced by blowing into a hollow space; *inspiration* is of slow production, of low pitch, and not tubular in quality; *expiration* is of lower pitch than *inspiration*. Its common site is the superior regions. It betokens a pulmonary cavity or globular bronchial dilatation." *The character of respiration may be*, (4.) *Amphoric*, "when it gives the sensation of blowing into a large cavity with thick walls filled with air, and is imitated by blowing gently into a narrow-necked glass bottle. It is distinctly metallic and musical. It is caused by the air in the bronchial tubes acting on the air in the cavity. It may accompany both respiratory sounds, but is most often heard in inspiration; it is generally circumscribed, or only partially diffused. It is heard in pneumothorax with pulmonary fistula and large tubercular cavities."

"**Rhonchi, Râles, or Rattles** are the names given to certain accidental sounds met with in pulmonary disorders, caused by more or less disturbance of the natural respiratory process, and produced either in the air-tubes, the air-sacs, or in cavities formed in the lung-tissue. These endo-pulmonary sounds may be dry, moist, or of an indeterminate character. They have been classed as follows:—

(1.) Dry or Vibrating Rhonchi,	High pitch, . . .	Sibilant.
	Low pitch, ...	Sonorous.
(2.) Mucous Rhonchi, . . .	Crepitant, ...	Very fine.
	Moist crackling, . .	Less fine.
	Mucous, ...	Unequal, fine and coarse.
	Gurgling or cavernous,	Very coarse.
(3.) Intermediate Rhonchi, .	Clinking, ...	First dry, then thickly moist (gummy)."
	Crumpling, . . .	

Exo- or peri-pulmonary sounds happen in disease of the investing membrane, and these, from their site and genesis, are called *pleural friction-sounds.* These sounds are described as grazing, rubbing, grating, creaking, crumpling (Clymer).

"**Changes of Voice.**—The modifications of the voice which occur in disorders of the lungs and pleura are valuable signs. The natural tracheal or laryngeal voice may be heard in unnatural sites, or natural bronchial resonance may be materially altered in intensity, pitch, concentration, and apparent proximity to the listening ear, or it may be diminished or suppressed. The *whispering voice* may also undergo changes (Flint). These unnatural changes of the voice may be ranged under the following heads" (Clymer):—

Intensity, ...	Exaggerated resonance.	
	Bronchophony.	
	Exaggerated bronchial whisper and whispering bronchophony.	
	Diminished or suppressed resonance.	
Character and pitch, . .	Pectoriloquy, ...	Cavernous. Amphoric.
	Cavernous whisper.	
	Ægophony.	

The annexed tables may assist the student in comparing the sounds heard—(1.) in health, and, (2.) in disease, pp. 553 to 556.

Section IV.—Relation of the Parts of the Heart and Great Blood-vessels to the Walls of the Thorax.

A knowledge of the exact position of the several parts of the heart, particularly of its valves and orifices, and of their relation to fixed points on the surface of the chest, is essential to accurate diagnosis, and is acquired by the same methods as in the case of the lungs—namely, by *inspection, palpation, percussion,* and *auscultation.* The size of the heart, and of its several parts, may thus be relatively determined in the living subject; so also its relative position, and any amount of displacement it may have undergone, and whether or not its valves or orifices are diseased. The apex and base of the heart (being the most fixed parts) are the most convenient from which to trace the outline of the heart, and to determine what parts correspond to certain fixed points upon the surface of the chest.

Relative Position of Base and Apex.—The heart is situated obliquely in the cavity of the thorax, from above downwards, from before backwards, and from right to left. It lies behind the middle and lower bone of the sternum, also behind the cartilages of the third, fourth, and fifth right ribs, near the sternum, and the cartilages of the third, fourth, fifth, and sixth ribs on the left side, in front of the bodies of the sixth, seventh, and eighth dorsal vertebræ. It rests immediately above the diaphragm, upon its cordiform tendon, the serous layer of the pericardium only being interposed. Owing to the obliquity of its position, the *line of the base of the heart* looks upwards and backwards towards the right shoulder. The *apex* points downwards and forwards towards the space

TABLE I.—THORACIC *SOUNDS* OF *RESPIRATION* AND OF THE *VOICE* HEARD IN HEALTH.

Sounds of Respiration.	Synonym.	Character of the Sounds.	Common Site of Production.
Vesicular.	Respiratory murmur of inspiration and expiration.	*Inspiration.*—A soft diffused murmur of a gentle *breezy* character, increased in intensity with rapidity and force of respiration, and prolonged by a full inspiration. *Expiration.*—Slightly harsher and more hollow, weaker and shorter, usually not above one-fourth the length of inspiration.	Entrance of the air and its expulsion from the air-cells and terminal portions of the bronchi —vibrations of tissue therewith connected.
Puerile.	Loud vesicular murmur.	The respiratory murmur of children and women louder than that of adults, but with characters as above.	
Bronchial or Tubal.	Tracheal.	*Blowing* as of air passing quickly through a tube; higher in pitch than the vesicular sounds; more rapidly evolved. *Expiration* as long, or nearly so, as *inspiration;* generally a perceptible interval between *inspiration* and *expiration.*	Sites corresponding to the bifurcation of the trachea, the upper part of sternum, and between the scapulæ.
Sounds of the voice through the lungs, **Natural Bronchophony.**	Vocal resonance. Pectoral vocal resonance.	Obscure, thrilling sound of the voice, diffused, and conveying the idea of distant origin. Articulation sometimes appreciable. Resonance more marked in males than females, and in adults than children; is only markedly present over the first bone of the sternum and in the interscapular regions.	The voice in articulation, passing down the trachea and bronchi, is obscured, intercepted, weakened, and diffused, by passing through the spongy pulmonary vesicular tissue to reach the surface of the chest.

TABLE II.—THORACIC *SOUNDS* OF A MORBID TYPE, SOMETIMES CALLED *RALES* BY FRENCH, AND *RHONCHI* OR *RATTLES* BY ENGLISH AUTHORS, EVOLVED DURING THE ACTS OF *RESPIRATION*.

A.—IN THE PULMONARY SUBSTANCE.

English Name of Sound (THOMSON).	Synonym.	Character of Sound conveyed by	Relation to Inspiration and Expiration.	How and Where Produced.	Diseases with which it is most usually associated.
I. Bubbling.					
a. *Bubbling rhonchi or rattles.*	Mucous rhonchus or râle.	The bursting of bubbles of some size, unequal and varying in number, modified by coughing and expectoration.	Co-existing with both.	Bubbling of air through liquid (mucus, blood, or pus) in bronchial tubes, of the size of a crowquill, and heard in the central or middle part of the lungs.	Bronchitis after secretion has become established.
b. *Small bubbling rhonchus or rattle.*	Subcrepitant or submucous râles or rhonchi.	The bursting of more minute bubbles, producing weaker sounds.	Co-existing with both movements, but predominating during inspiration.	Bubbling of air through more or less viscid fluid in minute bronchial tubes, as at their peripheral distribution.	Capillary bronchitis of both bases of lungs, tubercular bronchitis of apex, resolution of pneumonia.
c. *Gurgling rattles.*	Cavernous rhonchi or râles.	The bursting of bubbles obviously of large size, with a hollow gurgling sound, or a metallic sound, if the bubbles be small.	Co-existing with both acts.	Caused by the bursting of bubbles in a hollow space, inclosed by more or less dense and smooth walls—the more so the more perfect the gurgling.	Excavations from tubercle or other causes, dilatations of bronchi, pus in the pleura, with a bronchial fistula.
II. **Clicking,**	Humid crackling, humid crepitation, humid crackling rhonchus.	Successive clicks, few in number, and tending to pass into the bubbling rhonchi, especially during expiration.	Co-existing with both acts, but more regular and distinct during inspiration.	Mechanism of its production obscure. It probably originates in the interior of softened tubercles, which have just commenced to communicate with the minute bronchi.	Generally in *direct* connection with tuberculous exudation, which has commenced to soften.
or **Crackling.**	Dry crackling rhonchus, dry crepitation.	A succession of three or four minute dry, short, sharp, crackling sounds, permanent in many cases when once established, and tending to pass into the clicking sound. At first it may disappear for a day or two, and again recur.	Co-existing exclusively *with inspiration.*	Mechanism of their production undetermined. Conveys the impression of being evolved at a distance from the surface, and in the great majority of cases is found in the *infra-clavicular* and *supra-clavicular* regions.	Generally observed on the eve of the softening process in tubercles.

III. Crepitation. a. *Primary.*	Crepitant râle or rhonchus.	The idea of crepitation, like that produced by rubbing slowly and firmly between the finger and thumb a lock of one's hair near the ear (WILLIAMS). An immense number of sharp sounds, conveying the notion of minute size and dryness.	Co-existing exclusively with *inspiration*, and at first towards its close only.	Probably due to the sudden and forcible expansion of delicate tissue, altered in its physical properties by the inflammatory state, and which probably undergoes minute ruptures.	Primary idiopathic pneumonia, or the pneumonic state established round tubercles. The crepitation of early pneumonia.
b. *Secondary.*	*Rhonchus crepitans redux.*	Crepiti of a bubbling nature, slowly evolved, few in number, and unequal or dissimilar and irregular in occurrence.	Audible in expiration as well as inspiration, but pertaining specially to the latter.	Probably due to the bubbling of air through fluid contained in the minute bronchi.	Co-existing with the resolution of pneumonia.
IV. Vibration. a. *Sonorous rhonchus.*	Sonorous râle.	A musical sound of a vibratory, deep, or grave tone, attended with fremitus of the walls of the thorax over a variable surface. *Snoring*, *humming*, *cooing*, and *bass* notes are its varieties.	Co-exists with inspiration and expiration, but especially marked in the latter, to which it may be limited.	Arises in the larger bronchi, and suggests the idea of vibrations.	Essentially associated with bronchitis.
b. *Sibilant rhonchus.*	Sibilant râle.	A high-pitched whistling sound of variable intensity and duration, and irregular recurrence. *Clicking*, *whistling*, and *hissing* varieties are described.	Co-existent with inspiration and expiration, especially marked in the former, but occasionally limited to either.	The influence of the passage of air on a local accumulation of viscid mucus.	Pulmonary emphysema and bronchitis.

B.—ASSOCIATED WITH THE MOTIONS OF THE PLEURA.

V. Friction.	Grazing, rubbing, grating, creaking, are varieties of this sound.	The sensation of friction by a series of abrupt jerking sounds, rhythmical with respiration, few in number and superficial in seat, limited in extent, attended with fremitus, palpable to the hand, and perceptible to the patient.	Invariably heard in inspiration, or in both respiratory acts.	The rubbing of two opposed serous surfaces together, attended by inflammation.	Pleurisy, or any cause of roughness on the surface of the pleura.

TABLE III.—THORACIC *SOUNDS* OF A MORBID TYPE EVOLVED DURING THE ACT OF *ARTICULATION* OF THE *VOICE*.

Name of the Sound.	Character of the Sound.	Physical Conditions under which it is supposed to be produced.	Diseases with which it is commonly associated.
I. Bronchophony.	Exaggerated resonance of the voice, unattended with articulation, diffuse or concentrated, and rarely producing any tactile sensation to the ear.	Increased density of the pulmonary tissue surrounding pervious bronchi, with enlarged calibre and hypertrophy of the substance of the bronchi. The more homogeneous the consolidation the better probably is the sound transmitted.	Hepatisation of lung; dilated bronchi; pleurisy, with effusion, when hepatisation co-exists.
II. Pectoriloquy, or Pectoriloquous Bronchophony.	Complete transmission of articulated words from the walls of the chest into the ear; the resonance being generally circumscribed and limited in extent.	When solid masses of lung lie between a large bronchus and the parietes; when a moderate-sized excavation exists, with smooth and dense internal surface.	Various morbid states in which such physical conditions may exist.
III. Amphoric resonance.	A ringing metallic sound reverberating through a cavity, and resembling that produced by speaking into a broad-mouthed empty pitcher.	The voice reverberating in a large cavity, communicating with a bronchus by a small aperture.	Chiefly in phthisis.
IV. Ægophony.	A sound—vibratory, tremulous, cracked, and irregular, limited in its seat, comparable to the bleating of a goat, or to the voice of the exhibitors of Punch; synchronous with the articulation of each word, or following like a feebly whispered echo from a distant source. The sound appears to flutter tremulously about the applied end of the stethoscope.	When a stratum of fluid contained in the pleura compresses the lung, the voice in the bronchial tubes is thus rendered more distinct by the compression of the pulmonary texture, and is thrown into vibration by the layer of fluid.	A tendency to it during the early period of pleuritic effusion, equally diffused and small in amount.

between the cartilages of the fifth and sixth ribs on the left side, slightly inside the vertical line of the nipple, where its impulse may be felt during life; that is, where the most superficial part of the left ventricle strikes the chest wall. *The base of the heart* is on a line with the interval between the cartilages of the second and third ribs.

THE PRÆCORDIAL REGION is *the region of the heart's superficial dulness.* Its limits, as already defined, correspond to a vertical line through the centre of the sternum; and about the middle of the bone, nearly on a line with the cartilage of the fourth rib, the edge of the left lung separates from this middle line, and passes obliquely to the left side, thus exposing a small portion of the pericardium (Fig. 24, p. 536), which is uncovered by lung. The exposed surface has a triangular shape; the apex above, the base below. The parts of the heart thus exposed beneath the pericardium are a part of the left ventricle, near its apex, and a portion of the apex of the right ventricle. This triangular *præcordial* space is on a plane below the nipple and the fourth rib (Fig. 24, A). Its base is on a line with the cartilage of the sixth rib; its right boundary nearly a vertical line through the centre of the sternum; its left boundary is an oblique line through the cartilages of the fifth and sixth ribs on the left side. Within these limits the heart is in contact with the parietes of the chest, yielding a characteristic sound to percussion. *The sac which incloses the heart* has a pyriform shape, the base below, the apex above, exactly the reverse of that of the heart. Thus, the *base of the pericardium* is on a line with the upper part of the xyphoid cartilage; *its apex* is a short distance above the origin of the large vessels, and generally on a line with the articulations of the cartilage of the second ribs with the sternum; but may extend as high as the level of the articulation of the first ribs with the sternum. The sac is wider at the centre (corresponding to the greatest transverse diameter of the heart) than it is at the base; and towards its centre it extends more towards the left side.

The line of the base of the ventricular portion of the heart is from three to three and a half inches below the clavicles (left and right respectively), and on a line with the junction of the cartilage of the third left rib and fourth right rib with the sternum. *The line of the base of the left ventricle* rises as high as a line drawn across the junction of the cartilage of the *third left* rib with the sternum—*i.e.*, about three inches below the clavicle on that side. *The line of the base of the right ventricle* corresponds to a line across the upper margin of the junction of the cartilage of the *fourth right* rib with the sternum—*i.e.*, about three inches and a half below the clavicle on that side.

The impulse of the apex of the organ is to be felt between the fifth and sixth left ribs, near where the body of these ribs joins the cartilage. The apex is a little below the fifth left rib, slightly to the left of its junction with its cartilage, and on a line with the articulation of the xyphoid cartilage with the sternum. The nipple in the male has been considered to be a useful guide. It is said to be upon the fourth rib, or over its upper or lower edge, a little more than an inch to the left of the junction of the rib with its cartilage. The edge of the left ventricle reaches the nipple on the left side. *The length of the ventricular* portion of the heart is determined by the length of a line drawn from the middle of the sternum, between the cartilages of the third ribs, to below the fifth left rib, slightly to the left of its junction with the cartilage. The greater part of the *right ventricle* lies behind the sternum: at its upper part it extends slightly to the right of this bone, a small portion extending under the cartilages of the fourth and fifth right ribs close to the sternum. Its apex is to the left of the sternum, a little above the apex of the heart; and a part of the right ventricle extends under the cartilages of the fourth and fifth right ribs, close to the sternum. The inferior margin of the right ventricle is nearly on a line with the junction of the xyphoid cartilage and the sternum. The anterior wall lies immediately under the sternum. It is overlapped at its upper portion by both the right and the left lung. The *left ventricle* is covered by the left lung; and all its anterior surface is to the left of the sternum, extending from the cartilage of the third left rib to the interspace between the fifth and sixth left ribs, near where the cartilage joins the body of these ribs. It lies between the sternum and the nipple on the left side, to which its left margin reaches. The *right auricle* lies to the right of the sternum, entirely covered by the right lung. Its appendix lies behind the cartilage of the third right rib, its tip rests against the right side of the ascending portion of the arch of the aorta, and is on a line with the pulmonary valves. The *left auricle* is entirely covered by the left lung. Its appendix is the only portion seen when the pericardium is laid open. It lies behind the cartilage of the third left rib, close to the sternum, resting against the left side of the commencement of the pulmonary artery. The *line of the base of the auricles* is on a line with the interval between the junction of the second and third ribs with the sternum, the greater portion of it being under the sternum.

RELATIVE POSITION OF THE ORIFICES OF THE HEART.—The *right auriculo-ventricular orifice* lies behind the centre of the sternum, on a line with the lower margin of the articulation of the cartilages of the fourth rib with the sternum. The *left auriculo-ventricular orifice* is on the same level, but on a plane posterior to the right. It lies behind the cartilage of the fourth left rib, near to or behind the sternum. The *valves of the pulmonary artery* are on a line with the space between the cartilages of the second and third ribs to the left of the sternum, and very close to this bone. In some instances they may lie a little lower down—namely, on a line with the junction of the cartilage of the third left rib with the sternum, and immediately under it. The *aortic valves* lie behind the sternum, on a line with the junction of the cartilages of the third rib with the sternum, and towards the left edge of this bone. When the valves of the *pulmonary artery* are situated lower down, the *semilunar valves* of the aorta will be lower also, and on a line with the interval between the insertion of the cartilages of the third and fourth ribs. A line drawn across the inferior margin of the third ribs corresponds to the *base of the valves of the pulmonary artery*, and to the *free border of the aortic valves.* The *right ventricle* ascending higher than the left, *the orifice of the pulmonary artery* is on a plane higher than that of the aorta; hence the *pulmonary orifice* is the highest up, as well as the most anterior, of all the orifices of the heart. The *aortic orifice* lies behind the pulmonary orifice, but on a lower plane. The *left auriculo-ventricular orifice* is immediately behind the aortic orifice, but on a lower plane. The *right auriculo-ventricular orifice* is nearly on the same plane as the left, but more anterior, about three quarters of an inch lower than the pulmonary orifice.

POSITION OF ARCH OF THE AORTA.—The *ascending* portion of the arch of the aorta curves to the right of the sternum, between the cartilages of the second and third ribs. In this part of its course it is still within the pericardial sac, and (in the dead subject) lies at the depth of an inch and a half from the surface, the margin of the right lung and the pericardium being between it and the parietes of the chest. The *transverse portion of the arch of the aorta* crosses the trachea at the centre of the first bone of the sternum on a line with the lower margin of the articulation of the cartilages of the first ribs with the sternum, and at a still greater depth from the surface. The *arch of the aorta* approaches most closely to the parietes of the chest at the point at which the *arteria innominata* comes off—*i.e., on a line with the junction of the cartilage of the second right rib with the sternum.*

THE ORIGIN OF THE PULMONARY ARTERY is on a line with the junction of the cartilages of the third ribs with the sternum. The tip of the left auricle rests against its left side. The pulmonary artery ascends about two inches before it divides; and at that point a portion of the margin of the vessel comes to the left of the sternum between the cartilages of the second and third ribs. The division of the artery is on a line with the upper edge of the cartilage of the second ribs where they join the sternum.

The *ascending vena cava* passes through the diaphragm by an opening, which corresponds to the upper part of the xyphoid cartilage. In various morbid states the apex of the heart is formed by the *right ventricle* alone, or by the right and left together; in hypertrophy of the right side, for example, associated with *bronchitis* or with *emphysema.* When the right ventricle chiefly enters into the formation of the apex of the heart, the apex is then broad and rounded, rather than of the normal conical form, and is evidence of long-standing pulmonary obstruction (BELLINGHAM, BOUILLAUD, FULLER, PEACOCK, WALSHE, WILKS).

SECTION V.—DIMENSIONS OF THE ORIFICES OF THE HEART.

The arterial orifices progressively increase equally till the meridian of life; after which, as age advances, the aortic orifice enlarges more rapidly; so much so that in old age it sometimes is larger than the pulmonary artery orifice. Beyond fifty years this is apt to occur. In children, up to the sixth or to the tenth year, the orifices (arterial) are both the same; after this the pulmonary orifice increases more rapidly, and becomes larger than the aortic. The two auriculo-ventricular orifices progressively grow or enlarge at a uniform rate, till the right side acquires the start of the left at an early age. The healthy working of the heart is consistent rather with its relative size and the relative dimensions of the orifices to each other, compared with the size and weight of the body generally, the condition of other organs, such as the lungs, the liver, the stomach, the spleen, and the kidneys, than with the absolute size of the heart itself, or of its orifices. In health, there is found to be a certain determinate relative magnitude of the four orifices of the heart to each other, and therefore a relative magnitude of the areas of those orifices, as shown by Dr. Herbert Davis, in *Proceedings of Royal Society*, No. 118, 1870. Measurements of the four orifices of the heart have now been recorded with great exactness by several anatomists, and the mutual relationship of the *areas* of these orifices is of practical importance in connection with cardiac diseases.

The mean circumference of the four orifices of the heart, expressed in English inches, is as follows :—

CIRCUMFERENCE* MEASUREMENTS (DR. REID).

Orifices.	Male.	Female.
Tricuspid, . . .	5·3	4·9
Pulmonic, . . .	3·7	3·5
Mitral, ...	4·6	4·2
Aortic, ...	3·2	3

CIRCUMFERENCE MEASUREMENTS (DR. PEACOCK).

Orifices.	Male.	Female.
Tricuspid, . .	4·74	4·562
Pulmonic, . .	3·552	3·493
Mitral, . . .	4	3·996
Aortic, . . .	3·14	3·019

The mean areas* of the four orifices are thus found to be :—

AREA IN SQUARE INCHES (DR. PEACOCK).

Orifices.	Male.	Female.	
Tricuspid,	1·78	1·6	= 1¾ sq. in. nearly.
Pulmonic	1·00	·97	= 1 ,,
Mitral,	1·27	1·27	= 1¼ ,,
Aortic,	·78	·67	= ¾ ,,

AREA IN SQUARE INCHES (DR. REID).

Orifices.	Male.	Female.
Tricuspid, . .	2·24	1·9
Pulmonic, . .	1·01	1
Mitral, . .	1·7	1·4
Aortic, . .	·8	·71

Thus the *tricuspid*, having the largest area, is more than double the size of the aortic opening; and the law which Dr. Davis believes to be distinct and constant, regulating their relative magnitude in health, is found by comparing the *ratios* of the areas of the corresponding orifices, expressed as below:—

$$\frac{\text{Area of Tricuspid,}}{\text{Area of Mitral,}} = \frac{1·78}{1·27} = 1·4 \text{ nearly.}$$

$$\frac{\text{Area of Pulmonic,}}{\text{Area of Arotic,}} = \frac{1}{·78} = 1·3 \text{ nearly.}$$

Or the area of the *tricuspid* bears nearly the same ratio to the area of the *mitral* which the ratio of the area of the *pulmonic orifice* does to that of the *aortic* orifice—*i.e.*, if the *tricuspid orifice* were twice the size of the *mitral orifice* in area, the *pulmonic orifice* ought to be twice the size of the *aortic orifice* in area. The two *ratios* differ only by about *one-tenth*. In man, the horse, donkey, dog, ox, calf, sheep, and pig, the same result has been found; and, generally, it is observed that the area of the *tricuspid orifice* is larger than the area of the *mitral orifice* by ·51 inch nearly; and so also is the area of the *pulmonic orifice* larger than the *aortic orifice* by the same amount. One healthy orifice being known, the area of the corresponding opening on the other side can thus be calculated. Any deviation may be so determined, and the amount of contraction or dilatatio fairly estimated. So, also, if the areas of any three of the openings b known, the area of the fourth can be estimated.

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* The area is got by multiplying the square of the circumference by ·07958.

Generally speaking, the *tricuspid orifice* ought to be larger than the *mitral orifice,* and *half as large again,* or more than double that of the *aortic orifice.*

The *mitral orifice* ought to be larger than the *orifice of the pulmonary artery,* and larger than the *orifice of the aorta* by more than *one-fourth.*

The *orifice of the pulmonary artery* ought to be larger than the orifice of the *aorta* by about *one-fifth* to *one-eighth.*

The *mitral orifice* tends to diminish under all forms of disease which diminish the supply of blood coming from the lungs to the left side of the heart—*e.g.,* smallness of the chest and lungs compared with the size of the person, and imperfect respiratory acts. Such forms of contraction are independent of organic disease of the part, unlike induration, which implies increased development of tissue round the orifice, and leading to its contraction. Under this latter condition ulceration may ensue, when the anterior curtain of the valve is generally partially destroyed; large soft vegetations or concrete fibrine are deposited from the blood, and are found adhering to the diseased parts. If the *mitral orifice* is relatively small and contracted, the left auricle may become surcharged with blood. Its walls are then apt to become thick, tough, and hypertrophied, in place of being uniformly thin. Pulmonary obstruction is associated with this condition, and enlargement of the right side is a subsequent effect. *Aortic obstruction* or contraction enlarges relatively all other parts of the heart. The orifices of the right side of the heart exceed in size those of the left, and more especially the *auriculo-ventricular orifices,* compared with the arterial orifices; but in advanced life the *aortic orifice* tends to exceed the *pulmonary.*

The researches of Bizot, Peacock, and Reid, all show that the size of all the orifices of the heart is relatively greater in the male than in the female, and that their size progressively increases as life advances in both sexes. The following are the average dimensions of the orifices of the heart given by Bizot:—

	MALE.	FEMALE.
Right auriculo-ventricular orifice, . . .	54·12 lines.	48·25 lines.
Left auriculo-ventricular orifice, . . .	45·17 ,,	41·3 ,,
Pulmonary orifice, ...	32·2 ,,	30·7 ,,
Aortic orifice, ...	31·15 ,,	28·4 ,,

SECTION VI.—RELATIVE BULK AND WEIGHT OF THE LUNGS AND HEART.

BULK OF THE HEART.—From birth to extreme age the volume of the heart is continually increasing, very obviously up to the twenty-ninth year. Such growth implies—(1.) Progressive enlargement of its orifices; (2.) Increasing thickness of walls of ventricles, and especially of the left; (3.) Progressive increase of weight and bulk—the bulk varying from 12·5 to 19·8 cubic inches in normal adult hearts. The thickness of the parietes of the heart varies also with age; those of the *left* ventricle are thicker in the male than in the female at every age, and the thickness increases as age advances. The parietes of the right side increase, but in a much less ratio. The *thickest part* of the parietes of the *left ventricle* is at the centre, next at the base, and it is thinnest at the apex (the mean thickness is under *six* lines). The *thickest part* of the parietes of the *right ventricle* is at the base, where it is above *two* lines. The *thickest* part of the *septum ventriculorum* is at its centre.

There are certain diseases which tend to increase the weight of the heart, without the valves or aorta being necessarily diseased. For example, *chronic bronchitis* tends considerably to increase the weight, and so does *Bright's disease of the kidneys*. The weights (abnormal) of the heart arrange themselves in the following order, beginning with the highest:—(1.) Without material disease of valves, aorta, or lungs, Dr. Peacock records a weight of 40 ounces 12 drachms. (2.) With adhesion of pericardium alone. (3.) With *aortic* disease the enlargement is considerable. (4.) With *aortic valve* disease there is greater enlargement. (5.) With mitral valve disease, enlargement is not so considerable as in either aortic disease or aortic valve disease.

Weight of Lungs and of Heart increases as life advances, and is greater in the male than in the female at all ages (Bizot, Boyd, Peacock). Generally, it may be stated that the weight of the healthy heart is greater in persons who die after short periods of illness, and less in those who have suffered from emaciating or exhausting diseases. Taking this difference into account, the inference is that in adult males who have died from acute diseases, or from the effects of accidents, the ordinary weight of the heart is from nine to eleven ounces avoirdupois; and in those who have died from chronic wasting diseases, eight to ten ounces. Occasionally in excessive wasting and several fevers, the heart's weight is as low as five or six ounces; and, on the other hand, in very powerful men over six feet it has been noted as high as twelve ounces.

Section VII.—Mode of Examining the Heart.

To determine the nature, situation, and extent of morbid changes in the heart, it is necessary to recognise readily any alteration in the heart's impulse, either as regards its strength or the situation in which it is felt it is necessary to be able to detect any difference in the extent and degre of the heart's superficial dulness, or any change in the character of it sounds different from those which are normal. The inspection of th external surface of the thorax, and the application of the hand to th *præcordial* region should never be omitted. Positive information obtained by these two methods of examination, which mutually assist o another. The exact point at which the apex of the heart comes contact with the parietes of the chest may be determined simply inspection and position. The *strength* or *feebleness* of the impulse of t heart is to be determined by the application of the hand. *By inspect* it is ascertained whether the two sides of the thorax are symmetric and (in connection with cardiac diagnosis) whether there is any bulg in the *præcordial* region, or any unusual pulsation at any part of parietes in the large arteries which come off from the arch of the ao as well as in the jugular veins or epigastric region. By *the applicatio the hand*—palpation—the force or impulse of pulsation is determi the frequency or slowness of the heart's action is judged of, and the regularity or irregularity of its movements. We may likewise appreciate by this means any *tremors* or *frictions* which accompany its action in the pericardium. To determine the impulse of the heart, the hand must be placed directly over the cardiac surface; but *mediate* palpation may be used by placing one end of the stethoscope over the part where the impulse is, when the extent to which the instrument is elevated, and the force with which this is accomplished, will give an idea of the strength of the heart's impulse, especially in hypertrophy, or in hypertrophy with dilatation of the ventricles. In healthy persons with well-formed chests the impulse of the heart is so slight as not to be perceptible to the individual himself; and it is felt only at one spot—namely, between the cartilages of the fifth and sixth ribs on the left side—*i.e.*, from one to two inches below the nipple, and to its sternal side. When the parietes of the chest are much loaded with fat, the impulse is scarcely perceptible to the hand; while in thin persons it is evident to the eye. The impulse is somewhat stronger in the erect than in the recumbent posture. A forced inspiration diminishes it, and causes it to be felt lower down than usual; while a deep inspiration elevates the ribs, without raising the heart in the same degree. In some cases it may be necessary to turn the patient from the recumbent position on to his left side and well round on his face before the apex beat is felt. Its *area* should then be marked with ink. In a forced expiration the impulse is more perceptible, and is felt higher up. In examining the heart it is therefore necessary to make the patient vary his position, and to examine the heart both during inspiration and expiration. Calmness and tranquillity on the part of the patient must be obtained, because mental excitement, as well as exercise or exertion, increase the impulse of the heart. A diminished impulse, circumscribed or weak, may be due to feebleness of the action of the heart from general debility of the system; or alteration of its muscular tissue, as in softening or fatty degeneration; or owing to disease in the lungs or pericardium, as from effusion into the sac, when the apex of the organ may be prevented from coming in contact with the parietes of the chest. The impulse is also diminished in cases of attenuation of the walls of the ventricles, with dilatation of their cavity. Emphysematous lungs may overlap the heart, and prevent its impulse from being felt. Increased impulse is generally due to some morbid state of the heart itself. It is stronger than natural in hypertrophy of the walls of the left ventricle, and is greatest in hypertrophy with dilatation of the ventricles. In such cases the impulse is slow, gradual, heaving, double, and occasionally so violent as to shake the bed on which the patient rests. This slow, progressive, heaving impulse is produced by no other cause than hypertrophy with dilatation of the ventricles of the heart; and in such cases the extent of surface over which the impulse is felt is much increased, and the whole side of the chest is sometimes elevated by the action of the organ.

The situation of the impulse may be altered by displacement of the heart, as (in cases of empyema) towards the left side when the right pleural cavity is distended with fluid or air, and to the right side when the left pleural cavity is distended with fluid or air. The impulse may then be felt on the right of the sternum. In cases of ascites the heart may be pushed upwards, and its impulse felt on a plane higher than natural; so also in cases of ovarian and other abdominal tumors, hysterical tympanites, or advanced stages of pregnancy. In emphysema of both lungs the heart is displaced downwards. The impulse is then felt sometimes as low down as the epigastric region. When fluid is effused into the pericardium, the site of the impulse is somewhat elevated; and as the amount of fluid increases, the impulse becomes weaker, unequal, undulatory, or irregular; and when the effusion is very considerable the impulse will be altogether absent. In hypertrophy of the *left ventricle*, with dilatation of its cavity, the impulse is felt lower down than natural, more to the left side, and occasionally on a line vertically from the axilla. In hypertrophy with dilatation of the *right ventricle*, the impulse is felt lower, more to the right side than natural, and not unfrequently on a line with the xyphoid cartilage. In the former case (hypertrophy with dilatation of the *left* ventricle), the impulse is progressive, heaving, and strong, elevating the hand or stethoscope of the observer, and felt over a very much larger surface than natural. In the latter case (hypertrophy with dilatation of the *right* ventricle), the impulse is felt over a more circumscribed space, and is neither heaving, prolonged, nor very strong. But downward displacement is not due *only* to hypertrophy. Three causes conspire to produce it—one is the hypertrophy of the organ; another is dyspnœal inflation of the lungs; and another is flattening of the diaphragm, which always exists where there is an abiding source of dyspnœa. This descent of the diaphragm is due to the instinctive efforts made in all forms of dyspnœa to obtain more air. The large heart is thus pushed downwards by the dyspnœal lung inflation, and pulled downwards by the diaphragm (HYDE SALTER, *Brit. Medical Journal*, February 8, 1862). *Turgescence and pulsations in the jugular veins* are appreciated by inspection and palpation. They are signs which accompany advanced stages of some cardiac diseases. Turgescence is the most common, and occurs when an impediment exists to the free passage of the blood through the right side of the heart; and when the tricuspid valve imperfectly closes the right auriculo-ventricular orifice, and so permits regurgitation into the auricle at each systole of the right ventricle, we have pulsation of the jugular veins as well as turgescence. Pulsation is always most evident immediately above the clavicles, and may extend half-way up the neck. When the *mitral valve* permits regurgitation, a "purring tremor" may be felt when the hand is placed over the region of the *mitral valve;* and a similar tremor may be felt over a dilated aorta when the valves are incompetent, or when a communication exists between a vein and an artery. The jarring pulse in the radial artery, in cases of aortic valve incompetency, is an instance of the "purring tremor" felt in an artery of small calibre.

Percussion.—The whole of that portion of the anterior wall of the chest behind which the heart is situated is sometimes called the *præcordial region;* but *anatomically* it is a region more limited, corresponding to that part of the pericardium uncovered by lungs (Fig. 24, p. 536 *ante*). Both regions, however, are definable by the limits of a dull sound on percussion and more slight percussion. The portion of heart *uncovered by lung* seldom exceeds *two inches* in any direction. It has a triangular shape, the base below, the apex above. It consists of a portion of the apex of the right ventricle, and of part of the left ventricle near its apex, on a plane below the nipple and the *fourth* rib. Its base is on a line with the cartilage of the sixth rib. Its apex is at the point where the margins of the opposite lung begin to separate from one another—*i.e.*, immediately below the *fourth* rib. The triangular boundaries of this anatomically true *præcordial space* are constituted—(1.) On the right side of the thin edge of the right lung, by nearly a vertical line through the centre of the sternum; (2.) on the left side of the thin margin of the left lung, by a more or less oblique line through the cartilages of the *fifth* and *sixth* left ribs; (3.) below it is bounded by the diaphragm. Over this region, a sound less dull than that yielded by the liver is elicited to slight percussion, and on a stronger percussion a difference of sound can be detected where the thin margin of the lungs covers the heart. The mode of percussing this region, so as to mark the lung part and the heart part, is to lay one finger over the decidedly dull part, and another over the slightly resonant edge of the lung, when, by striking the two fingers alternately, the arched line along which the organ lies in contact with the walls of the chest, may be traced with accuracy, unless fat obscures the resonance (Hope). Generally, the region of this *deep-seated dulness* extends transversely from the left nipple to a little beyond the right of the sternum, and vertically from the third to the sixth ribs. The true anatomical *præcordial space* may be diminished if the lungs are largely developed, and may disappear if the anterior margins of the lungs are emphysematous, so that their edges meet in front of the organ. Another means of estimating the size of the heart is by *auscultatory percussion.* This requires two competent persons to determine the result, and is managed as follows:—"A solid cedar cylinder, six inches in length and one inch in diameter, cut in the direction of the fibres, and with an ear-piece attached, is applied to the centre of the *præcordial* region, while the ear is applied to the other end: percussion is then made by another person from the point near where the cylinder is applied towards the limits of the heart in every direction. So long as percussion is made over the body of the heart, a distinct sharp shock is felt directly in the ear; but as soon as the limits of the heart are passed, this sharp shock immediately ceases, even in passing from one solid organ to another in contact with it, as from the heart to the liver" (Drs. Cammann and Clark). Practice will enable a discrimination to be made between the characteristic sound of the heart and the diffused shock produced by striking the ribs.

The mean diameter of the healthy adult cardiac region (not the anatomical *præcordial* region only) has been found to be as follows:—

		Inches.	Lines.
Vertical diameter,	Male,	4	0
	Female,	3	7
Transverse diameter,	Male,	4	4
	Female,	4	1
Right oblique diameter,	Male,	4	10
	Female,	4	10
Left oblique diameter,	Male,	3	10
	Female,	3	7

The region of the heart's superficial dulness is increased whenever the heart is enlarged, or whenever fluid to any amount is effused into the sac of the pericardium. If the walls of the ventricles are hypertrophied, or if their cavities are dilated, the dulness will extend over a wider surface, and its extent is in some degree a measure of the increased size which the organ has attained. The enlarged heart pushes aside the lungs, and a larger portion of it comes in contact with the parietes of the chest. In hypertrophy with dilatation, the heart attains the largest size that it is capable of, and the præcordial region may yield a dull sound over a square surface of from two to six inches. When hypertrophy predominates over dilatation, the space which yields a dull sound is wider from above downwards. When dilatation predominates over hypertrophy, the region in which a dull sound is yielded transversely is wider. When there is fluid in the pericardium, a larger surface than natural in the *præcordial* region yields a dull sound; the degree of dulness is more pronounced, and the sensation of resistance considerably greater than in the former case. The situation of the dulness, its amount and degree, are the guides as to its probable cause. When it is caused by enlargement of the heart, the site of the dulness is lower down and more to the left side, than when it depends upon liquid effused into the pericardium. If a large amount of fluid is contained in this sac, a dull sound may be elicited by percussion as high as the articulation of the second rib with the sternum, and even in some cases as high as the first rib. The degree of dulness over fluid is also much more marked than over the heart itself, and the resistance to the finger is greater. Solidification of lung in the immediate vicinity of the heart will cause an apparent extension of the cardiac dulness.

Auscultation and the Sounds Associated with the Action of the Heart.—Auscultation of the heart, like that of the lungs, may be either *mediate* or *immediate*; but generally there are many objections to immediate auscultation. In the case of females it is indelicate; in dirty persons it is disagreeable; while in contagious diseases it is not without risk; besides, there are some situations in which either the ear cannot be applied or in which the stethoscope is much more convenient. In many cases o valvular disease of the heart, the stethoscope, and especially the forms o the double stethoscope already noticed, are absolutely necessary.

Each complete revolution of the heart is accompanied by two successiv sounds, separated from each other by two intervals of silence. These tw sounds *are unlike*; and the two periods of silence *differ in duration*. Withi the limits of health these sounds have a *variable duration*, and *limit of surfa* over which they are heard, as well as of *intensity*, and of a certain *rhyth* The *rhythm* of the heart is maintained when its two sounds succeed ea other, and are followed by an interval of repose, which varies in leng according to the duration of the previous systole, and according to t rapidity with which the sounds succeed each other.

The first sound is coincident with the systole of the ventricles, impulse of the apex against the side of the chest, and the pulse of carotids. It is sometimes also called the *systolic* or *inferior* sound of heart. It should be listened to over the apex of the heart, and the *ca* *pulse felt* at the same time.

The second sound is synchronous with the diastole of the ventricles, the recedence of the heart from the side, and the pulseless state of the carotids. It is sometimes also called the *diastolic* or *superior* sound. It should be listened to at *mid-sternum,* near its left edge, and on the level of the second interspace. A very short silence (which only becomes obvious when the pulse does not exceed sixty in a minute) succeeds the first sound, but a distinctly appreciable period of silence and complete repose—a pause of some duration—succeeds the second sound. During an entire cardiac pulsation (arranged as to time into periods of tenths), the periods of *sounds* and of *silence* bear the following proportion to each other:—*Systolic sound,* $\frac{4}{10}$; *first interval of silence,* $\frac{1}{10}$; *diastolic sound,* $\frac{2}{10}$; *second interval of silence,* $\frac{3}{10}$; to be followed by the systolic sound commencing another pulsation. The *first sound* is of a prolonged and dull character compared with the *second sound,* which is more quick, short, and clear, and bears a close resemblance to that produced by *lightly tapping, near the ear, the knuckle of a bent finger with the soft extremity of a finger of the other hand* (HOPE).

The Line of Transmission of these sounds respectively is of the greatest practical importance to be observed, because, being so constant in health, any variation indicates some modifying cause. The *first sound* passes slantingly upwards to the left acromial angle, growing weaker and weaker on the way. Its intensity diminishes on the way to, and at, the right acromial angle. The propagation backwards of the first sound is thus clearest and fullest to the left; so that, while audible at the left back, it may be inaudible at the right. The *second sound* has the region of the base for its centre; and in nine people out of ten is heard more clearly at mid-sternum, *on the level of the second interspace,* than at any point of the præcordial region (WALSHE). It radiates towards the right and left acromial angles, with greater clearness to the left than the right, while posteriorly it is heard at the surface with less clearness and distinctness on the right side than on the left.

Next to the *lines of propagation of the sounds* being determined, it is necessary to analyse the *sounds* themselves, and compare them—(1.) At both sides of the apex and region of the base; (2.) at base and apex on the same sides of the organ; (3.) at base and apex of opposite sides. In any one of these regions, compared with the other, the sounds in health are found to vary so materially in positive and relative properties that any single description of them cannot be given.

The natural sounds of the heart thus indicated may be of abnormal character, as regards *intensity, pitch, duration, quality, rhythm, reduplication,* and *apparent distance,* at each of the following points:—namely, at the third interspace, and along the third rib for the distance of three inches from the left edge of the sternum, *for all sounds;* at the point where the apex beats, *for mitral sounds;* at the left of the sternum, and over the ensiform cartilage (if the apex be not in this position), *for tricuspid sounds;* at the third left costal cartilage and adjoining part of the sternum, *for both aortic and pulmonary sounds;* at the second right and second left costal cartilages, *for the sounds of aortic and pulmonary orifices respectively* (*What to Observe,* p. 43).

It is important to observe the relationship of the sounds of the heart to the pulse. The first sound anticipates, by a very short but appreciable interval, the pulse at the wrist. An interval consistent with health is undetermined; but it may be stated generally, that if the diastole of the most distant arteries, such as the posterior tibial, behind the inner ankle, or the arteries on the dorsum of the foot, is so much retarded as to become synchronous with the *second sound*, the state indicates disease. It is a frequent attendant on insufficiency of the aortic valves. When the sounds of the heart, after a certain number of perfectly regular beats, are suspended during the time usually taken to perform an entire revolution of the cardiac functions, the sounds of the heart are said to *intermit*. A sudden pause or silence then occurs, and such an intermission sometimes happens more or less regularly, or after a fixed number of regular beats. This constitutes *intermittence* or *intermission* of the heart's action. It may occur in individuals otherwise in perfect health; but it is common in diseased states of the valves or orifices of the heart, where some impediment exists to the direct passage of the blood, or where regurgitation is permitted.

Morbid Sounds of the Heart, or Murmurs.—The term "*murmur*" is applied to a sound superadded to the normal sound of the heart. It was first employed by the late Sir John Forbes, and has been very generally adopted since. The natural sounds may be so obscured, or even obliterated, that the *murmur* or *morbid sound* is alone heard. According to their supposed seat of production these *murmurs* are—(1.) *Endocardiac*, sometimes called *valvular*; (2.) *Pericardiac*, also called *exocardiac*. All endocardiac or valvular murmurs yield a "*blowing*," "*rough*," "*rasping*," "*sawing*," "*booming*," or "*bellows*" *sound*, as in the whispered expressions of the words "*who*" or "*awe*," the double letter "*ss*," or the single letter "*r*;" and there are certain spots where they may be heard in their greatest *maximum force*; namely,—(1.) A few lines above the left apex; (2.) just above the ensiform cartilage at mid-sternum; (3.) on the level of the third interspace; and, (4.) at the junction of the third left cartilage with the sternum. The physical causes which may explain the *mechanism* of these murmurs are due either—(1.) To pure constrictions of natural orifices; (2.) to pure widenings of natural orifices; (3.) to roughness of surfaces; or, (4.) to the association of the latter condition with either of the two former. When murmurs are due to such single or combined mechanism, they are said to be *organic murmurs*, t distinguish them from *inorganic* murmurs due to certain morbid cause not yet well understood, but which are connected—(1.) With certai states of the blood, as in spanæmia; or, (2.) with dynamic or functiona action of the heart itself. It is of the greatest importance to determin therefore, the nature of a *murmur*, especially as to whether it is reall *organic* or *functional*. In this investigation the essential points to b inquired into, to guide the diagnosis, are:—(1.) Observe the relationsh of the murmur to the systole or diastole; that is, the *rhythm* of a murmu or the position, in point of time, which it holds during the differe physiological acts which constitute a complete cardiac pulsation—name the *contraction*, the *dilatation*, and the *period of rest* of each of the caviti (2.) the spot of its maximum intensity on the surface of the chest; (the direction in which the murmur is transmitted; (4.) its quality a pitch; (5.) state of the natural sounds of the heart which may remain; (6.) presence or absence of any audible phenomena in the arteries or veins, or both; and (lastly) the duration and clinical progress of the case. Each orifice of the heart may be the seat of two murmurs, *constrictive* and *regurgitant*, *with* or *against* the current; and thus *eight murmurs* are the total number the occurrence of which is possible. The essential characters of these murmurs are condensed as follows, from Dr. Walshe's *Practical Treatise on Diseases of the Lungs and Heart;* and from Professor W. T. Gairdner's paper, entitled "A Short Account of Cardiac Murmurs," *Edin. Med. Journal*, Nov., 1861. In determining the attributes of a cardiac murmur, the first step in the inquiry is to determine which is the *second* sound of the heart, and which is the *first* (see p. 566). In the *rhythmical* succession of the heart's actions the phenomena which we can appreciate externally are a little later than the commencement of the heart's action. *Before there is either sound or impulse the peristaltic contraction has already taken place;* and whatever the pathological origin or seat of the murmur may be, those which immediately succeed the first sound and the impulse correspond to the period of ventricular contraction; and those which succeed the second sound correspond to the period of ventricular dilatation.

Dr. Gairdner gives the following classification and account of cardiac murmurs:—(1.) An *auricular systolic murmur*, or *presystolic bruit*, is one which *precedes and runs up to the* FIRST SOUND of the heart, and which is in all probability produced in one or other of the auriculo-ventricular orifices, inasmuch as it coincides with the forcible emptying of the auricles into the ventricles through these orifices. Its reasonable interpretation, therefore, is *obstruction to the current of the blood* ENTERING *a ventricle.* If the left auriculo-ventricular orifice is affected, the murmur will be found to have the character of a *mitral murmur*, and to have its area at A (Fig. 25). If, on the contrary, the *tricuspid* orifice be obstructed, the murmur will occupy the triangular area C (Fig. 25). That the *presystolic bruit* is due to the prolonged contraction of the auricles occurring during the ventricular diastole and time of rest has been demonstrated by cardiograph and sphygmograph (MAREY, MAHOMED, WILKS, FAGGE, see MAHOMED in *Med. Times and Gazette*, May 18, 1872). (2.) A *ventricular systolic* murmur *succeeds and runs off from the* FIRST SOUND; and it may be produced either in the *auriculo-ventricular* or in the arterial orifices. In either case it coincides with the *emptying* of the ventricles; and, therefore, if *auriculo-ventricular* in origin, it is *a murmur of regurgitation;* if, on the other hand, it is of *arterial origin*, it is *a murmur of obstruction.* A *ventricular systolic murmur* may thus have four distinct solutions among organic valvular diseases. If the area be *mitral*, it is *a murmur of mitral regurgitation.* If the area be *aortic*, it is *a murmur of aortic obstruction.* If the area be *tricuspid*, it is *a murmur of tricuspid regurgitation.* If the area is that of the origin of the *pulmonary artery*, it is *a murmur which indicates pulmonic obstruction.* (3.) A *ventricular diastolic murmur* succeeds and runs off from the second sound, and may be produced either in the *auriculo-ventricular*, or in the *arterial* orifices. In either case it coincides with the *filling* of the ventricles; and, therefore, if *auriculo-ventricular* in origin, it is a murmur of obstruction; and if *arterial*, it is *a murmur of regurgitation.* A *ventricular diastolic murmur* may thus have four distinct solutions among organic valvular diseases. If its area is *mitral,* it is *a murmur of mitral obstruction;* if its area is *aortic,* it is *a murmur of aortic regurgitation;* if its area is *tricuspid,* it is *a murmur of tricuspid obstruction;* if its area is

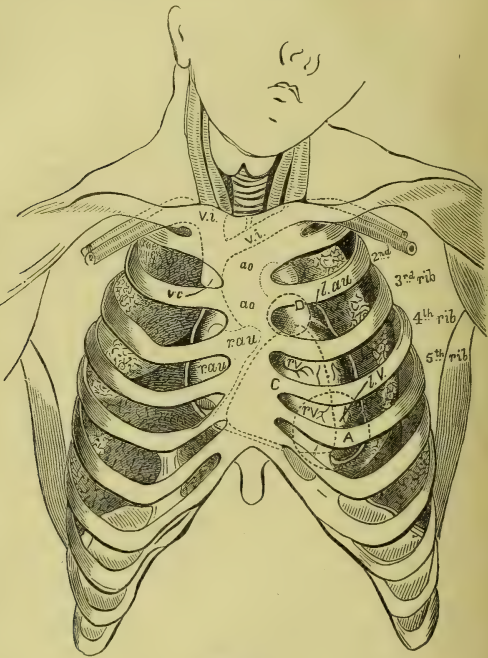

Fig. 25.*

* The heart: its several parts and great vessels in relation to the front of the thorax. The lungs are collapsed to their normal amount, as after death, exposing the heart. The outlines of the several parts of the heart are indicated by very fine dotted lines. The area of propagation of valvular murmurs is marked out by more visible dotted lines. A, the circle of mitral murmur, corresponds to the left apex. The broad and somewhat *diffused area,* roughly triangular, is the region of tricuspid murmurs, and corresponds generally with the right ventricle where it is least covered by lung. The letter C is in its centre. The circumscribed *circular area,* D, over which the pulmonic arterial murmurs are commonly heard loudest. In many cases it is an inch, or even more, lower down, corresponding to the *conus arteriosus* of the right ventricle, where it touches the walls of the thorax. The internal organs and parts of organs are indicated by letters as follows:—*r.au,* right auricle, traced in fine dotting; *ao,* arch of aorta, seen in the first intercostal space, and traced in fine dotting on the sternum; *vi,* the two innominate veins; *rv,* right ventricle; *lv,* left ventricle.

of the origin of the *pulmonary artery,* the *murmur denotes regurgitation from the pulmonary artery.*

The most frequent combinations of these murmurs are those which denote:—(1.) Combined aortic obstruction with regurgitation, indicated by ventricular systolic and ventricular diastolic murmurs. (2.) Mitral contraction, indicated by *auricular systolic* murmurs or *presystolic bruit.* (3.) Various combinations of the two preceding forms, the aortic and mitral valves being both diseased. (4.) Mitral obstruction with dilated right ventricle, and consequently tricuspid regurgitation, indicated by *auricular systolic murmur,* heard over area A (Fig. 25); and ventricular systolic murmur, heard over area C.

The rarest of all *murmurs* are those which denote *obstruction of the pulmonary artery,* and those of *tricuspid obstruction.* These murmurs are still more rarely observed singly, being usually in combination with diseases causing murmur on the left side of the heart.

In an able communication to the Hunterian Medical Society of Edinburgh, the late Dr. J. Warburton Begbie called attention to "the diagnostic value of an accentuated cardiac second sound" (*Edin. Med. Journal,* June, 1863). This accentuated second sound—equivalent to an intensified or greatly pronounced sound—is heard in instances of aortic aneurism and aortic dilatation, associated with atheromatous degeneration, as well as in some cases of hypertrophy and dilatation of the left ventricle. In a case of aortic aneurism the second sound of the heart has been observed so intensified or accentuated over the base of the heart as at once to be recognised even by *tyros* in the art of auscultation. When this sound occurs (which is of a *booming* or *ringing* character), it is to be presumed that the aortic valves are competent. If they were insufficient, a diastolic murmur would be the result, and would be apt to cause the most skilful physicians to overlook the existence of aneurism.

Modern diagnosis localises murmurs, chiefly from the observation of the areas of transmitted sounds already indicated. There are four distinctive areas to which murmurs arising at these orifices may be propagated. The accompanying woodcut (modified from those of Professors W. T. Gairdner, of Glasgow, and Luschka, of Tubingen) (Fig. 25, p. 570), indicates the areas.

1. **Murmur connected with the Mitral Valve, Orifice, or neighbouring portion of the Left Ventricle,** may be the result of inefficiency of the valve, by changes in its structure, or from roughness of its edges, as by vegetations, shortening of the *chordæ tendiniæ,* or fibrinous coagula amongst them causing obstruction. It may also result from simple roughness or deposit on the under surface of the valve without positive insufficiency. It is a *ventricular systolic murmur,* of maximum force, heard at and immediately above, or to the outside *of the left apex,* and which may completely or partially cover the first sound of the heart at the left apex, but which may also preserve its natural characters towards the base; and especially is this *systolic murmur* faintly or wholly inaudible at the right apex, the mid-sternal base, the pulmonary and aortic cartilages. It is more or less clearly audible about or within the inferior angle of the left scapula, and beside the dorsal vertebræ from the sixth to the ninth. This *murmur* is rarely of high pitch; and once established it is permanent.

The pulse is generally small, weak, irregular, *intermittent*, and *unequal*. *To find the area of this murmur* it is requisite to determine the exact seat of the apex beat, the patient lying a little to the left side, or even on the face. If there is no distinct apex beat, find the most remote point downwards and leftwards at which the impulse of the heart is discernible; test this point by percussion, to observe if it corresponds with the margin of the cardiac dulness; test it also by auscultation, to hear if the *first sound* is conveyed thither with special distinctness. If a murmur concurs in position with the seat of these different phenomena, and if its seat of diffusion is *round this point nearly in a circle* (Fig. 25), *it is probably of mitral origin*.

2. **Murmur associated with the Tricuspid Valve** may be due to regurgitation, or to the sharp collision of blood among thickened and roughened *chordæ tendiniæ*. It too is a *ventricular systolic murmur*, heard of maximum force *immediately above or at the ensiform* cartilage; inaudible, or nearly so, at the left apex, and very faintly, if at all perceptible, in the left vertebral groove opposite the lower angle of the scapula. It originates in the right ventricle; and when due to regurgitation, there is distension and pulsation of the auricle, vena cava, innominate and jugular veins, the distension of the latter being visible. It is generally a soft *murmur*, of low pitch, and rarely masks the systolic sound completely. It often escapes detection from two causes—namely, a powerful mitral *murmur*, with which it is usually associated, or a deep-seated venous *hum*. Professor W. T. Gairdner does not consider that *tricuspid murmurs* are rare, at least those of regurgitation. Their area is over the right ventricle, where it is uncovered by lung—*i.e.*, at the lower part of the sternum, and over the whole space between this and the seat of the mitral murmur. It is usually but little audible above the level of the *third* rib, and is thus distinguished both from the pulmonic, and still more from the aortic murmur. Its area is indicated by the triangular space in Fig. 25, but in cases of considerable hypertrophy and dilatation of the right side of the heart, especially in connection with emphysema (when the ventricle pulsates in the epigastrium), the murmur is heard loudest towards the xyphoid cartilage, and along the margin of the seventh left costal cartilage.

3. **Murmur connected with the Aortic Valve** habitually signifies a rough constriction of that orifice, and in rare cases has been traced to fibrinous coagula impeding the egress of the blood. It also is a *ventricular systolic murmur*, heard of maximum force at *mid-sternum opposite the third interspace, or upper part of the fourth rib*. It abruptl loses force between this point and the left apex, where it may be almos inaudible. Faintly perceptible at the second left cartilage, it is clearl audible at the second right cartilage, the notch of the sternum, and th left vertebral groove, opposite the second, third, and fourth dorsal vert bræ, thence rapidly losing strength downwards. It originates at th aortic orifice, and disappears about the sixth dorsal vertebra. It is pr pagated into the arteries of the neck. It is a high-pitched, harsh, lou and prolonged *murmur*. The concurrence of ventricular hypertrop increases its intensity and prolongs its duration the more contract the orifice is. The area of this murmur corresponds generally to regions of the sternum, and is often absolutely loudest close to xyphoid cartilage.

4. **Murmur connected with the Orifice of the Pulmonary Artery** may indicate obstruction or simple roughness in its valves, or pressure on the vessel by adventitious masses in the pericardium. It is also a *ventricular systolic murmur*, heard of maximum force at the *sternal edge of the third left cartilage,* or a little lower down, and imperceptible in the back. It is rarely met with.

5. **The Murmur indicative of Obstructive Narrowing of the Mitral Valve** is a *ventricular diastolic murmur,* heard in maximum force *immediately above and about the left apex.*

6. **The Murmur which indicates the Probability of Tricuspid Narrowing or Obstruction** is also a *ventricular diastolic murmur,* and is heard in maximum force at the *ensiform cartilage.*

7. **The Murmur which indicates Regurgitation at the Aortic Orifice** is likewise *ventricular diastolic,* and is heard of maximum force at mid-sternum, opposite the third interspace or fourth cartilage; and it is often carried down loudly to the left apex. It is usually of an inspired blowing character, sometimes almost hissing, rarely rough, and completely fills up the interval of repose and silence which ought to follow the second sound. It differs from *constrictive* aortic murmur in being heard with almost as much intensity *about the ensiform cartilage as opposite the third interspace.* When it covers completely the second sound of the heart at the point of its maximum intensity, the valves may be presumed to be utterly incompetent.

8. **The Diastolic Murmur connected with insufficient Pulmonary Valves** is so rare that it is only mentioned here to complete the notice of cardiac murmurs which may be heard.

The relative frequency of intra-cardiac organic murmurs may be stated in the following order, commencing with the most common—namely, *mitral regurgitant; aortic constrictive; aortic regurgitant; mitral constrictive; tricuspid regurgitant; pulmonary constrictive; pulmonary regurgitant; tricuspid constrictive* (WALSHE). These may, however, be variously associated together; and when they co-exist, they are to be distinguished by the *rhythm,* the *pitch,* and the *character* of the aspiration. The point at which a murmur is produced being in the majority of cases one of the four valvular orifices, all doubtful murmurs should be tested, in the first instance, on the supposition that they are valvular. With this object in view the most important practical points to be determined are,—(1.) The actual size and position of the heart, and the relation of its several parts to the thoracic walls (as described in the previous sections); (2.) The anatomical *præcordial* space must especially be accurately defined; (3.) The exact point of the apex beat is to be determined; (4.) The character of the impulse, both of the right and left ventricle, should be carefully studied; (5.) Determine by careful stethoscopic observation the exact seat and the limits of the diffusion of the murmur actually under observation.

The Pericardial Murmurs consist of *friction,* or rubbing sounds, analogous to those already described in the pleuræ (Table, p. 555), and result from the movements of two opposed surfaces on each other, having been rendered dry or rough by change of tissue or exudation. Pericardial murmurs are almost limited to cases of inflammation of the pericardium. These friction murmurs are generally double, and are sometimes louder during the diastole than the systole of the ventricles. They appear to be superficial or near, and are seldom audible beyond the limits of the *præcordial* region. They never replace the ordinary sounds of the heart, and are entirely independent of them. Their duration is usually short, frequently ceasing entirely after having been heard for a few days, and not unfrequently changing their character and seat within the period that they are audible. A peculiar vibratory thrill, sensible to the hand laid upon the parietes, frequently accompanies them.

SECTION VIII.—SIGNIFICANCE OF THE PULSE IN CARDIAC DISEASE.

Certain forms of cardiac disease are capable of impressing peculiar and well-marked characters upon the pulse. In health the pulse of the carotids ought to correspond with the ventricular systole, and with the first sound of the heart; and when the heart, the arterial system, and the blood are each in a normal condition, the force, the strength, the frequency, and the fulness of the radial pulse may be taken as a measure of the strength or feebleness of the systole of the left ventricle, of the rapidity with which the movements of the heart are performed, and of the amount of blood transmitted at each systole of the left ventricle. The beat of the pulse in the radial artery ought to be a little—a very little—later than the ventricular systole. The interval is almost imperceptible, unless the pulse is unusually slow. In the dorsum of the foot the interval is more easily appreciated. It is advisable to place one hand upon the præcordial region, or to auscultate the region of the heart, while the finger is on the carotid pulse, or the radial, to determine these points in all cases. The pulse at the wrist cannot be trusted to for determining the exact time of the heart's beat. *In softening of the heart* the pulse is sometimes much less frequent than the cardiac systole, because the impulse fails to be transmitted (FULLER, J. C. B. WILLIAMS, GAIRDNER). *In hypertrophy of the left ventricle,* when the parietes are increased in thickness, the systole is strong in proportion, the blood is propelled into the aorta with increased force, and the radial pulse is strong and hard. Its velocity may not be increased, but the systole takes a longer time to be completed, and the pulse will "dwell longer under the finger." When dilatation is combined with hypertrophy, so long as the circulation continues free the pulse will be full, or of larger volume, because the amount of bloo propelled at each systole will be larger; but when dilatation is combine with attenuation, or if dilatation simply prevails, the radial pulse wi have nearly opposite characters to those stated. It will be soft and wea *The pulse in aortic regurgitation* acquires a peculiar character. It is jerkin and receding, though regular; while the pulsations of the arteries of th upper extremities and of the neck are visible, as if "leaping." It h been named a "locomotive" pulse by Bellingham and Todd—*i.e.,* t arterial tubes are SEEN to move by elongation—"leaping forth at ea beat of the heart." This is sometimes termed the "pulse of unfill arteries." In well-marked examples it appears as if the blood v divided into separate little balls, which pass in rapid succession under the finger. The sensation is better *seen* and felt in a large artery, such as the brachial, and two or more fingers should be laid on the line of artery. The arteries (when tortuous especially) appear like worms under the skin, wriggling into tortuous lines at each pulsation (WILLIAMS). This kind of pulse is also sometimes observed in *aneurism of the ascending or transverse portion of the arch of the aorta*, as well as in cases of disease of the aorta itself, when it has become rigid, elastic, and dilated. It is a peculiarity brought out by raising the patient's arm vertically while feeling the radial pulse. *Intermission of the pulse* indicates the slightest degree of derangement of the heart's action. It is not uncommon in persons advanced in life, in gouty subjects, and in derangement of the digestive organs with flatulence. It is also met with in cases of disease of the valves, or of the muscular tissue of the heart. *An unequal pulse* is one in which some of the pulsations are strong, and others weak. *An irregular pulse* is one in which a few rapid beats are succeeded by one or more slower beats, and when the interval between them is different. *An unequal and irregular pulse* are much more unfavourable signs than a simply *intermittent pulse.* Both are met with in the same cases—in certain diseased states of the valves at the left side of the heart, or in morbid conditions of the muscular tissue. *In contraction of the left auriculo-ventricular orifice,* the pulse, in addition to being weak and intermittent, will become small, irregular, and unequal, although the heart's action continues to be strong. "The heart may often beat so violently as to shake the patient in his bed, while the pulse is small, weak, and irregular. It appears as if there were two *pulses;* one is slow and deliberate for two or three beats, succeeded by three or four rapid and indistinct pulsations" (ADAMS). *In mitral regurgitation,* when the closure of the valve is very imperfect, the pulse becomes weak and small, and will intermit if the circulation is hurried; and when a considerable quantity of blood is permitted to regurgitate into the auricle, the pulse will also become irregular and unequal. *In contraction of the aortic orifice* (when it becomes extreme only) the pulse becomes small and intermittent or irregular, resembling the pulse of considerable constriction of the mitral orifice. When the contraction is slight, it is neither weaker nor smaller than natural, and is perfectly regular. *In degeneration of the muscular tissue* the pulse in the advanced stages is small, weak, irregular, and unequal, sometimes slow, and the impulse of the systoles fails to be propelled. *In pericarditis with copious liquid effusion,* the pulse presents somewhat similar characters. *During the formation of fibrinous concretions within the cavities* of the heart the pulse suddenly becomes small, weak, intermittent, and irregular.

SECTION IX.—USE OF THE SPHYGMOGRAPH.

The idea of the instrument called the "*Sphygmograph*" first came to the mind of Galileo. Vierordt afterwards contributed a useless instrument laying claim to the name. The more perfect instrument was devised by M. Marey to determine various points in the physiology of the circulation of the blood; and, as an instrument of great accuracy, it could not fail to become of much value as an aid in determining the nature of diseases of the heart and arteries. The instrument and its practical use was first shown me in the autumn of 1863, by my teacher, the late John Goodsir, the distinguished Professor of Anatomy in Edinburgh, who predicted for it an extensive field of usefulness in diagnosis. The late Dr. Anstie (who was the first to show the instrument to a medical society in this country), and Dr. B. Foster (who was the first to publish an account of any considerable number of observations made with the instrument), Drs. B. Sanderson and Mahomed were afterwards most active exponents of the methods of appreciating the characteristics of the pulse by the sphygmograph. Mahomed, Grimshaw, Boileau, Gemmell, Clifford Allbutt, and Galabin have since added much to our knowledge on the subject. From the writings of these men the following account of the sphygmograph is given; but more especially from those of Dr. Foster, of Birmingham, who kindly lent me his MS. notes for the previous edition of this text-book, when he not less kindly revised this section, and added much valuable matter to it. Marey's instrument, with slight modifications, is the one usually employed; and it ought to have Mahomed's modification for estimating pressure. Pond's instrument may also be mentioned.

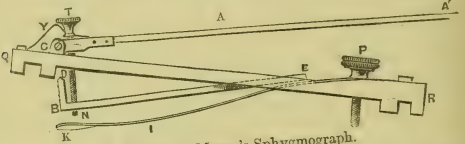

Fig. 26.*—Marey's Sphygmograph.

To "*feel the pulse*," and to "*look at the tongue*," all patients consider as most essential duties on the part of the physician. The sphygmograp does not aim at superseding either of these essentials. It aims at addin to our knowledge by writing down, for our inspection, the traces o *hardness* or *softness* of the pulse, thus measuring the *arterial tension*—tha is to say, the greater or lesser *pressure of the blood within the blood-vessel*

* The woodcut (Fig. 26) shows in the interior of the frame Q R the essential part the instrument, which consists of a flexible steel spring I, covered on its under surfa at its free extremity with a convex plate of ivory, K. This ivory plate rests up the artery to be examined, and, by virtue of the elasticity of the spring I, exe a certain pressure upon it. Each pulsation of the vessel raises the spring sligh at K, and the muliplication of this movement is obtained by means of a very li lever, A, which moves upon a pivot, C. The elevation of the spring is transmit to the lever, very near to its centre of movement, by means of a bar of metal, which moves round the point E; this bar terminates in a vertical plate, B D, is pierced by a screw, T. When the screw acts upon the spring, the connectio established between the spring and the bar, and the movements of the spring transmitted to the bar, and through its vertical plate to the lever. In orde insure the transmission of the movement, the plate B D must be in contact with under surface of the lever. By means of the screw T we can arrange this, and reg the interval between the point of the plate B D and the under surface of the l In order that the lever should not be projected too much upwards by sudden m ment, and also that it should overcome any slight friction experienced in the at its terminal point A, a small spring, Y, rests upon its fixed extremity, and pre over its descent. The screw, P, enables us to regulate the amount of pressure cised upon the artery by the spring, I.

The value of the instrument at present mainly rests on the exactitude and precision which it gives to our notions regarding "the *practical significance of various forms of pulse*"—a kind of knowledge only acquired by great and long experience. It is also a kind of knowledge very difficult to be conveyed to a pupil, but at the same time it merits quite as much attention as the urine or temperature. In the use of the instrument great care and much patience are required. The artery for which it is specially adapted is the radial; and the point at which it is most advantageously applied is just where the artery crosses the inner side of the styloid process of the radius. If the artery can be compressed against bone, it is a most important point for the production of a perfect tracing, and when the compressibility of the pulse is to be measured. The shape and development of the forearm, the position, size, and depth of the radial artery, all affect the ease of application and amount of pressure required. When the tactile spring, K (Fig. 26), has been placed accurately on the radial artery, the pressure must be varied until the maximum rendering of the pulse-movements is obtained—*i.e.*, till we obtain the greatest upstroke, when the spring follows most perfectly the expansion of the artery, and with which the apex is most sharply marked and free from roundness, and the various notches best defined (MAHOMED). This variation of pressure is a matter of much importance. All observers agree that a pressure *too great* will destroy the finer features of the pulse-trace, and manufacture a flat-headed curve. A pressure *too slight*, on the other hand, results in the record of a small and mean tracing, in which all the finer features are lost. The use of incorrect pressure is one of the most common sources of error in sphygmography, and the greatest care is necessary in deciding upon the amount of pressure to be employed. The amount, when ascertained in each case, should be carefully recorded. The amount of pressure required to totally extinguish the pulse, so that the lever remains perfectly motionless, should

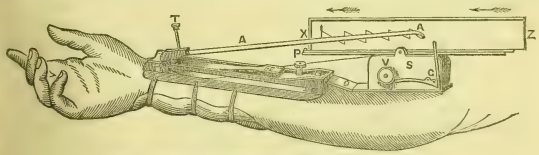

Fig. 27.*—Sphygmograph applied to the arm.

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* The woodcut (Fig. 27), modified from Marey, shows the instrument placed upon the arm over the radial artery in the position for use. The lever, A, is here seen to carry at its free extremity a little pen, which, filled with ink, registers its movements upon the paper which covers the plate X Z; this plate is moved at a uniform rate in the direction indicated by the arrows by means of watchwork placed beneath in the case, S. Ten seconds are occupied by the passage of the plate. The button, V, enables us to wind up the watchwork; and the small regulator, G, starts the plate, or stops its motion, as desired. The application of the instrument Dr. Foster has found much facilitated by the use of elastic bands, instead of a silk lace, as recommended by Marey. These bands embrace the arm, and are hooked on to the small projecting points on the metal framework, as seen in the diagram. The addition of a pad, suggested by Mr. Waters, to the under surface of the arm, renders the instrument more easy to the patient, and prevents any pressure from the bands.

also be recorded. It gives an estimate of the force with which the heart is contracting. Some of Marey's followers in France still dispense with any special means for adjusting the pressure on the artery, other than those furnished by the instrument in its original form, and contend that the maximum rendering of the pulse can always be obtained by careful manipulation (LORAIN). In this country several plans have been devised for measuring the pressure exerted on the pulse, viz.—(1.) By having a dial-plate described round the screw, P (Figs. 26 and 27), on which an index attached to the screw would point out the amount of pressure in grammes (FOSTER); (2.) by fixing weights on the head of the screw, T (Figs. 26 and 27), and so adding the extra pressure directly over the artery (FOSTER); (3.) by the addition of a screw and brass block to the carpal end of the sphygmograph, by means of which the distance between the under surface of the spring K (Fig. 26) and that of the frame at D can be increased or diminished, and the pressure exerted by the spring on the artery modified in either direction (SANDERSON);* (4.) by regulating the length of the radius of the eccentric in contact with the spring by turning a thumbscrew. The pressure thus applied to the artery is indicated by the figure engraved on a dial-plate, to which an index points. The plate is marked in ounces of troy weight: and the pressure may vary from 1 to 18 ounces—a range wide enough to meet any requirement (MAHOMED, *Medical Times and Gazette*, Jan. 20, 1872, p. 64). Each observer should graduate his own instrument, so as to be able to calculate the pressure exerted by the spring in each case. The instrument thus improved becomes a valuable though rough method of estimating arterial tension. It is true that the maximum rendering of the pulse can be obtained without any of these modifications, as the French observers contend; but it is only by some such methods as those described that the results obtained under different conditions, at different times, and by separate observers, can be made comparable, and full scientific value given to the record. Another practical point of importance consists in the reduction to a minimum of the friction between the receiving plate (if it be glass) and the point of the pen. Spurious tracings may result if this be not carefully attended to. The pen at the end of the lever should be made of very flexible metal, or of a needle like that used in microscopic manipulation; it can then be easily adjusted. For collecting the trace, smoked enamelled paper is the best; because (1.) the friction can be reduced more effectually; (2.) the pen scarcely ever fails to leave its record; and, (3.) because the tracings, when fixed (as they easily can be by photographers' varnish or a solution of *shellac* in rectified spirit, or of *gum benzoin* in methylated spirit, in the proportion of 1 to 6), are convenient for reproduction by photography, and for pasting into the daily record of the case in an hospital or private journal. It may be prepared by smoking over the flame of a paraffin lamp; and when the tracings are taken, the patient's name, date, and pressure employed, ought to be written with a sharp point on the tracing. In the selection of a sphygmograph the followin points must be attended to:—(1.) The instrument must be of full size (2.) the spring must be strong; (3.) the lever long enough to amplif the pulse movements fully; (4.) the clockwork should give a rapid rathe than a slow passage to the recording plate. The last point is very important, as a rapid transit gives a clear development to all the features of the line of descent. The sphygmograph ought to give an exact representation of the pulse-form, the frequency of the pulsations, and their regularity. It enables us to see at a glance any peculiarity in the entire series, or in any single pulsation. A trace, as below (Fig. 28), is composed of a series of curves, each of which (corresponding to a complete revolution of the heart) is called a *pulsation.*

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* Vide *Medical Times and Gazette*, March 21, 1871.

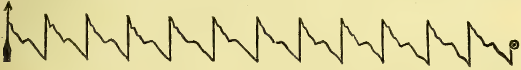

Fig. 28.

For purposes of description the pulse-curve (which corresponds with each pulsation of the radial artery) may be divided into three parts—the line of ascent, the summit, and the line of descent. It is important, however, to regard the pulse-curve as consisting of a systolic and a diastolic part, coinciding with the two periods of each cardiac revolution.

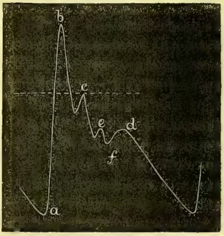

Fig. 29.

The woodcut (Fig. 29) represents a typical radial pulse-trace enlarged (after Dr. FOSTER); *a* to *b*, Line of ascent; *b*, Summit wave (primary or percussion wave); *c*, First secondary (or tidal) wave; *d*, Great secondary wave, or true dicrotic wave, or wave of distension; *e*, Third secondary wave, not generally seen. The notch preceding *e* corresponds to closure of the aortic valves, and is sometimes called the aortic notch. The first secondary wave, *c*, should be situated at the junction of the *upper third* with the *lower two-thirds* of the line of ascent, *a*, *b*, about the level of the dotted line. From *a* to *f* the ventricle is contracting or contracted, and *f* marks the closure of the aortic valves. The third wave, *e*, is seldom marked; but the three others can generally be distinguished, except when the tension is very high. The line of ascent corresponds to the ventricular systole, and marks the flow of blood into the aorta and great vessels. All these parts are represented in a healthy pulse-tracing, the pressure for which varies from one and a half to three ounces. The points to be noted in a tracing are:—(1.) The number of pulsations recorded in a certain time will give the exact frequency of the pulse. (2.) The length of the line of ascent, and whether vertical or more or less oblique. (3.) The shape of the summit, whether acute, round, or flat. (4.) The number, size, and position of the secondary waves. (5.) The direction and length of the line of descent beyond the aortic wave, and if there are any undulations in it. (6.) The relative height and depth of the curves, observing as to whether their summits and bases are in the same level or not. The *line of descent* is generally broken by the occurrence of several secondary waves, which correspond to the vibrations in the blood-column alternately to and from the heart. The summit of the trace is usually followed by a small notch, and then the trace rises again to form the first secondary wave, *c*. The relative position of this wave to the summit part is of most practical importance to observe. The deep notch in which the *third* secondary wave, *e*, is situated, which precedes the *second* secondary wave, *d*, is of great importance, as it marks the closure of the aortic valves. It corresponds with that reflux of blood towards the heart which forces together the lappets of the valves; and the *second* or great *secondary* wave (or dicrotism proper) corresponds to the vibrating motion towards the periphery given to the blood by the sudden flapping together of the aortic valves, and the prevention of regurgitation into the left ventricle. From *a* to the deep notch preceding *d*, corresponds to the period of the heart's systole, and measures the duration of each cardiac contraction. From the deep notch preceding *d* to the beginning of the next line of ascent marks the period of the heart's diastole (Dr. FOSTER'S *MS. Notes*). From the beginning of the line of ascent *a* (Fig. 29) to the notch *f*, in the line of descent, is the period of the ventricular systole. The notch, *f*, separates this systolic part of the curve from the remainder of the line of descent, which corresponds to the period of the ventricular diastole. Having made this distinction between the two parts of the pulse-curve, it is necessary to explain each of its finer features. These consist of elevations called waves, *b*, *c*, *d*, *e*, with intervening notches. The line of ascent, *a*, *b*, terminates in the first, percussive, or summit wave. This line is produced by the vibration imparted to the blood-column in the arteries, by the opening of the aortic valves at the very commencement of the ventricle's contraction. When the arterial tension is low, and the ventricle acting with its usual vigour, this line is lofty and nearly vertical, and ends in a pointed summit wave. When the arterial tension is high, or the ventricle weak, the vibratory element is not so well developed, and the line of ascent is consequently not so lofty nor so vertical, nor is the summit wave so sharp. The summit, *b*, wave is sometimes rounded and prolonged, but this only occurs when the first secondary wave, *c*, is blended with it. This first secondary wave, *c*, is due to the wave of distension, or pressure wave, which is produced by the passage of blood into the aorta from the heart. When the arterial tension is high, this wave is proportionally more developed, and is nearer to the summit of the trace; in cases of very high tension it is blended with the summit wave, and gives th summit of the trace a rounded form. This is perceived in the pulse b the finger in the qualities of fulness and hardness. There is anothe small secondary wave, *e*, which is not often seen except in pulses of lo tension; it is most probably vibratory in its origin. The two notche remain—the first precedes the wave, *c*, and represents the collapse of t arterial wall after the sudden vibration in the blood-column, conseque on the opening of the aortic valves. The great notch, *f*, which mar the end of the systolic part of the curve, is called the aortic notch, and due to the centripetal reflux, which effects the closure of the aortic valv The moment these valves are closed, the blood-column rebounds fr them as from a springboard, and sends towards the periphery the *g* secondary wave, *d*, or *true dicrotism*. When the arterial tension is hi and the aorta consequently very full, the aortic valves are closed quic and the reflux and the great secondary wave are faintly marked. On the other hand, when the tension is low, the reflux is more decided, and the aortic notch and the great secondary wave are more developed. After the great secondary wave or dicrotism, the line of descent falls gradually as the blood flows onwards, and by its obliquity marks the celerity of the fall of the pressure within the vessels, indicating the facility with which the blood passes on its course. The line varies in form; sometimes it is purely oblique, at others it forms a curve convex upwards, and occasionally one or more undulations may be seen in it in states of low tension. These are most probably vibratory in their origin. A state of low tension, with great elasticity of the arterial walls (such as we have in slight febrile conditions, when the capillaries are dilated and the passage of blood easy), favours the production of all these vibratory waves. When these waves are distinctly seen, the pulse-trace is sometimes called *polycrotous*, just as the normal pulse-trace is called *tricrotous*, from possessing three principal waves—the summit, the first secondary, and dicrotism, or great secondary waves. *Senile change* (Fig. 30) in the vessels, and consequent loss of elasticity (*senile pulse, or pulse of rigid arteries*) is indicated—(1.) By the diminution of *dicrotism;* (2.) by the great dimensions of the curve; (3.) by the closer proximity, not only of the first, but of all secondary ascensions to the apex of the curve; (4.) by the great development of the first secondary wave as compared with the dicrotism; by the substitution of a plateau for an apex; and by the unbroken character of the descent line—*i.e.*, the high positions of all the secondary waves in the line of descent.

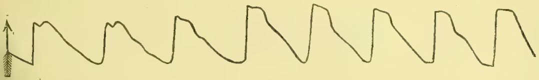
Fig. 30.*

In examining a pulse-trace, it should be noted in addition to the form of each pulsation, whether the summits of all of them can be joined by a straight line, and whether the bases can be connected by a similar line parallel to the former. In some instances this ceases to be the case, and a series of pulsations cannot be contained between such imaginary lines. The pulsations become irregular, and the line to join their summits or bases must cease to be horizontal. The line joining the summits of a series of pulsations is the line of the maxima of *arterial tension.* Its value as an indication is not absolute; it only tells us the variations that the arterial tension may undergo during the period of the observation; and it enables us to judge of the relative pressure within the vessels during any of the cardiac contractions registered. This *line of greatest tension* is of much value; and, with the corresponding *line of least tension,* should be observed in all cases. These lines generally undergo parallel deviations, so that a glance at either usually suffices to inform us of any change. Particular febrile diseases cannot be recognised by pulse-forms peculiar to them, but the changes which the pulse-curve undergoes are intimately connected with temperature variations. In the healthy pulse-curve the *tricrotous* form exists, but the febrile pulse ever tends to become *dicrotous*, and may become *monocrotous*. These changes are chiefly effected by the deepening of the aortic notch. When the notch has not sunk down to the level of the curve basis (the line of least tension), and has not quite annihilated the first secondary wave, but has retarded the great secondary wave or dicrotism, the pulse is called *hypo-dicrotous* (Fig. 31). With this form of pulse the temperature of the body rarely exceeds 100° Fahr.

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* *Senile pulse*, first half after Dr. S. GEMMELL: the second half after Surgeon-Major Dr. J. P. BOILEAU, from an officer, eighty years of age, and in vigorous health, who fought at Waterloo.

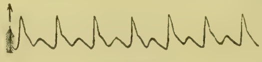

Fig. 31.*

When the aortic notch sinks to the level of the curve-basis, the first secondary wave having disappeared, or nearly so, and the dicrotism being still more retarded, the pulse is called *dicrotous* (Fig. 32). This form corresponds with a temperature of about 103° Fahr., and with a pulse-rate of over 100 per minute.

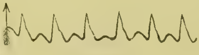

Fig. 32.†

When the aortic notch sinks below the level of the curve-basis, and the *dicrotism* appears partly blended with the line of ascent of the next pulsation, so that the aortic notch is below the level at which the upstroke begins, the pulse is called *hyper-dicrotous* (Fig. 33), and the temperature usually ranges above 104° Fahr. Other signs of importance also occur. A short and non-vertical ascension-line, with a square or blunt summit, indicates failing heart-action. The occurrence of irregularity in the pulse-curve at the height of the pyrexia is another grave sign. In its mildest form the irregularity consists in a want of exact similarity in the successive pulsations which affects the systolic portion more particularly, and tells of varying rigour of ventricular systole. When, however, there is an undulatory irregularity of the general line of the pulse-trace, we have a sign of still graver import, which informs us that the power of the ventricle

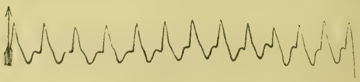

Fig. 33.‡

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* *Hypo-dicrotous pulse.*—The arrow-head indicates the top of the tracing.

† *Dicrotous pulse.*—The arrow-head indicates the top of the tracing.

‡ *Hyper-dicrotous pulse.*—The arrow-head indicates the top of the tracing.

is momentarily changing. When at an advanced stage of the fever the hyper-dicrotous pulse changes to the *monocrotous* or imperfect *monocrotous* form (Fig. 34), it is an almost certain indication of death (WOLFF).

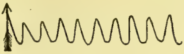

Fig. 34.*

Even if a pulse-trace fails to indicate any specific lesion, the sphygmograph is the most exact measurer of tension and index of a *hard* or *soft* pulse. When we find the pulse-trace without any well-marked notch *before* the great secondary wave, and the line of descent forming an almost unbroken oblique line, it indicates *high arterial tension*, with arteries unusually full, giving the incompressible hard pulse. On the other hand, when the line of descent falls suddenly, the arteries are insufficiently filled, and the pulse is known as the soft compressible pulse. The *force* of the pulse is indicated by the height of the pulsations. The greater the elevation of the lever, the greater the energy of the pulse-beat; and in many cases the strength of the ventricular contraction is expressed by the force of the pulse. But this law has many exceptions, and we find that the altitude of the pulse-trace depends on several other conditions. For example:—(1.) The volume of the artery greatly influences the amplitude of the trace. This can be seen in traces collected from old persons. In senile changes (Fig. 30) the volume of the vessels is increased considerably, and the trace betrays great fulness. Marey believes this to be due, not solely to the hypertrophy of the ventricle which exists in the old, but also to the dilatation of the artery. (2.) The state of arterial tension modifies greatly the force of the pulse; and, as the tension is dependent on the state of the capillary circulation, it may be said that in most cases "the force of the pulse is not in relation with the energy of the ventricular systole, but that it is regulated by the state of the circulation in the ultimate ramifications of the vascular system." By means of the manometer, in a great number of experiments, this law has been proved to hold good—a feeble state of arterial tension giving to the finger and the instrument the sensation of increased amplitude. The above diagrams (Figs. 35, 36) illustrate this (MAREY). Differences in the amplitude of the traces are very distinct. In the state of feeble

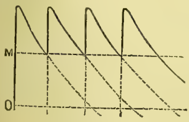

Fig. 35. †

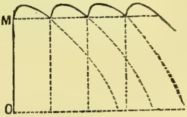

Fig. 36. ‡

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* *Monocrotous pulse.*—The arrow-head indicates the top of the tracing.

† The form of pulsation in a state of feeble tension.

‡ Under a state of strong tension.

tension, or easy passage of the blood onwards, the lever falls quickly to the point of least tension, and is elevated considerably at each pulsation. In the case of difficult passage of the blood through the capillaries (and consequently of great arterial tension), the lever descends slowly by a line convex upwards; and, long before it has reached a minimum tension equal to that in the former case, the lever is raised slightly by the next pulsation. While the lines of the *maxima* of arterial tension are the same in both cases, the lines of the *minima* are very different. On this depends the amplitude of the pulse-trace. (3.) The duration of the interval which separates the pulsations has also an effect on the amplitude of the trace. This is due to the fact that, during a long interval, the blood flowing continually onward lessens the pressure in the vessels, and thus favours the greater amplitude of the next pulsation. The discrimination of increased arterial resistance, as measured by the exaggeration of the systolic expansion, is the element of diseased action, whose detection and estimation are the most important purposes to which the sphygmograph can be put (Dr. SANDERSON). "Anatomical researches lead us to believe it probable that the earliest beginnings of what we may call degenerative disease consist in such structural alterations of the minutest arteries as, by rendering them less pervious to the circulating blood, must inevitably lead to increased arterial resistance estimable by the sphygmograph; and if by such an examination we can ascertain that the heart is overtaxed long before any change can be detected by auscultation or percussion, it is obvious that we have made a step forward in practical utility." Thus, he considers it likely that the *sphygmograph* is to be of greater use as an aid in forming an opinion as to the probable duration of life than in any other department of medical practice. There are many persons in whom, in the absence of any other trace of ailment, the pulse-curve indicates that the arterial resistance is excessive. The question is, "Are such persons sound?" Dr. Foster, and the late Dr. Anstie, on the other hand, state that the diseases in which the *sphygmograph* will prove of the greatest diagnostic value are—(*a.*) In aortic regurgitation, by estimating the amount of vavular imperfection; (*b.*) In discovering unsuspected commencing cardiac hypertrophy, senile disease of arteries, or capillary disease dependent on degenerative processes in the ultimate tissues; (*c.*) Above all, in discovering the existence of intrathoracic aneurisms, and in deciding the locality of an aneurism; (*d.*) In aiding prognosis and decisions as to treatment in the course of acute diseases.

A method has recently been invented by Dr. B. W. Richardson for making the movements of the pulse *audible* by the *telephone.* The instrument employed has been named "*the sphygmophone.*" A secondary sound is got from the movements of the pulse at the wrist by adding a *microphone* to a Pond's sphygmograph (See *Proceed. Royal Soc.*, p. 196, 1879).

SECTION X.—GENERAL SYMPTOMS OF THORACIC DISEASE.

The physician of the present day is a close observer of symptoms—*subjective* and *objective*—of constitutional states, and of physical signs; and with the many delicate instrumental appliances to appreciate those signs, he ought to form his judgment from the combined evidence of them all. The general symptoms which express derangement of the pulmonary organs and their functions are *dyspnœa, cough, expectoration, tenderness,* and *pain.* The general symptoms which indicate derangement of the cardiac apparatus are *palpitations, sinking,* and *fainting,* combined sometimes also with *dyspnœa, cough, pain,* and *tenderness.* The sensation of *dyspnœa* is brought about by an embarrassed or laborious breathing, amounting in severe cases to a sense of suffocation, expressed by the common English phrase, "*want of breath;*" or by the meaning conveyed by the Latin term "*anxietas,*" when the dyspnœa is at the point of greatest intensity. It is aggravated by exertion, some positions of the body, and a full stomach. The act of speaking is frequently arrested "*to fetch a breath;*" and the patient who suffers from dyspnœa cannot hold the breath, or refrain from the attempt to inspire, as a person in health can. The dyspnœa may occur in paroxysms, and the acts of respiration may be painful. The number of respirations performed in a given time is an important indication of dyspnœa. The number is greatly increased, and often unequally so, when the paroxysm is aggravated. In health, from eighteen to twenty acts of respiration are unconsciously completed in a minute, according as a person is lying, sitting, standing, or walking; and the ratio of the acts of respiration to the pulse varies in the proportion of one to four (WATSON), or one to nearly six (WILKS); that is, about one complete act of respiration for every *four* or *six* beats of the heart. The *altered rhythm of the breathing* may also indicate *dyspnœa,* which may be due to nervous or renal disorders. It may be "*suspirous*" or "*sighing*"—consisting of a few quick gasps or deep sighing inspirations, followed by a period of slow and shallow respirations, or a temporary suspension of it. It suggests cerebral disease and cerebral irritation, and fatty degeneration of the heart's fibres. In its most marked aspect it has been described as the "*Cheyne-Stokes respiration*" —which consists in "the occurrence of a series of inspirations increasing to a maximum, and then declining in force and length until a state of apparent apnœa is established. In this condition the patient may remain for such a length of time as to make his attendants believe that he is dead, when a low inspiration, followed by one more decided, marks the commencement of a new ascending and then descending series of inspirations" (STOKES). In children and in women the rate is more rapid. The following forms of dyspnœa are to be recognised :—(1.) *Cardiac dyspnœa* or *cardiac asthma*—the dyspnœa of cardiac disease and aortic aneurism; (2.) the dyspnœa of Bright's disease; (3.) the dyspnœa of asthma; (4.) the dyspnœa of bronchitis and emphysema; (5.) the dyspnœa of phthisis. All these will come to be noticed under the respective diseases of which they are characteristic symptoms. *Cough, expectoration,* and the nature of the *sputa,* furnish valuable indications of thoracic disease.

Microscopical Elements of Sputa consist of—(1.) Young epithelium-cells—*i.e.,* of mucous corpuscles; (2.) Of mature epithelium, in the form of pavement, cylindrical, or ciliated bodies; (3.) Cells containing granules, or bodies like cells made up of granules; (4.) Pus-cells; (5.) Coloured corpuscles of the blood; (6.) Fibrine, in the form of flake-like membranes, or as casts of the smaller bronchi and pulmonary air-cells, as in the expectoration of plastic bronchitis and in pneumonia from the third to the seventh day. They are sometimes seen as dichotomous cylinders with rounded enlargements, composed of fine filaments, generally covered with granules. (7.) Fat occurs in granules or globules. (8.) Tubercle matter; earthy, calcareous, amorphous, and crystalline particles also occur. (9.) Substances derived from the food. (10.) Carbon and true pigment, free or contained in cells. (11.) Fragments of pulmonary tissue.

Chemical Characters of Sputa.—The late Professor Laycock, of Edinburgh, had a chemical analysis made of very fetid expectoration in bronchitis, which demonstrated the existence in it of *butyric* and *acetic* acids. The odour was characteristic of the *butyrates of ethyl*, resembling the smell of Mayflower or apple blossoms, combined with an odour of fæces. Chemical investigation may thus demonstrate the cause of the excessive fetor in those cases which resemble gangrene of the lungs, if the smell alone is considered (*Med. Times and Gazette*, May, 1857, p. 480). A much more extended analysis has been recently made by Bamberger into the chemistry of the sputa—(1.) In chronic bronchial catarrh; (2.) in bronchial dilatation; (3.) in chronic pulmonary tuberculosis; (4.) in the infiltration of acute tuberculosis; and, (5.) in pneumonia. In sputa (chiefly catarrhal) the salts vary but little—the organic matters very considerably. The insoluble salts form about 4 to 5·5 per cent. of the whole saline contents; the chief amount consists of *chloride of sodium* and *phosphate of potash.* Puriform matter predominating causes the sputa to contain a greater quantity of organic and inorganic substances; there is considerably more *phosphoric acid* in the ash, considerably less *chlorine*, and less *sulphuric acid.* The ash of pneumonic sputa differs from that of catarrhal in several respects. The alkalies, combined with phosphoric acid (which amount to 10 and 14 per cent. of the saline constituents of catarrhal sputa), are almost entirely absent in pneumonia during the inflammatory period, but the sulphuric acid is remarkably increased. The quantity of chlorine (37 per cent.) is nearly the same as the average in the catarrhal (36 per cent.); and there is not much variation in the insoluble salts, except in the phosphate of iron derived from the blood. In the period of resolution the sputa of pneumonia become more similar to the catarrhal; the *phosphoric acid* increases, the *sulphuric* diminishes, and the chlorine reaches a very high amount, while the potash and soda are present in the same relative proportion as in the catarrhal, whereas during the inflammatory period this was inverted. Sugar has been detected during the height of the inflammation; and Dr. Beale has shown that an excess of the chloride of sodium is constantly present in pneumonic sputa. In the sputa of *bronchiëktasis*, sulphuretted hydrogen, acetic, butyric, and probably formic acids were detected (*New Syden. Society Year-Book*, 1860, p. 128; SCHMIDT'S *Jahrb*, band. 114, p. 3).

Sputa Typical of Pneumonia is characterised by its viscidity, semi-transparency, and tenacity, adhering strongly to the vessel containing it. So tenacious is it, that the vessel may be turned upside down without the sputa becoming detached from the sides. This rusty-coloured sputa consists of mucus intimately mixed with blood—not streaked with it, as in bronchitis, but thoroughly mixed and amalgamated with it; so that it acquires a yellowish, or reddish-yellow, or even a red colour, according to the quantity of the blood. If the disease be not very intense, the expectoration never attains the degree of viscidity or the depth of colour above referred to; but though still tenacious and adherent to the sides of the vessel, moves from one part to another as the vessel is tilted. If the disease progresses to a favourable termination, the sputa become more abundant, less adhesive, and less highly coloured, passing through the various shades of orange, until at length they become greenish or whitish, and resemble the expectoration of ordinary catarrh. If the disease be hastening to a fatal termination, the expectoration becomes scanty, less tenacious, and of a darker or dullish-brown hue, resembling the juice of prunes. If the type of inflammation be typhoid, or adynamic, or connected with tubercles in the lungs, the mucus may be tinged, or even streaked with blood; or it may consist throughout of nearly colourless, stringy, and more or less frothy mucus (FULLER, *On Diseases of the Chest*).

Sputa Typical of Gangrene of the Lung, at first of a muco-purulent character, sometimes tinged with blood, emits a very disagreeable odour; and as soon as a free communication is established between the air-passages and the sloughing tissue of the lung, they not only acquire an intensely fetid gangrenous odour, but assume an appearance more or less characteristic of the disease. They lose their muco-purulent character, becoming extremely liquid or sero-purulent, and of a dirty greenish or ash-grey colour. At the same time the breath acquires an offensive putrid odour, the pulse is feeble and rapid, with evidence of great and increasing prostration, (FULLER, l. c.)

Sputa Typical of Acute Bronchitis appear, after a few days, as a thin, saltish, frothy mucus, sometimes streaked with blood. They increase in quantity, and soon become glairy, semi-transparent, and of a faintly yellowish colour. Subsequently they assume a greyish or greenish-yellow tint, and become opaque and viscid. If the attack is severe, they become muco-purulent, and in some instances may even lose their glairiness, presenting the character of thoroughly opaque mummillated sputa. In *chronic bronchitis* the sputa may be either, (1.) The expectoration of a greyish, or greenish, or yellowish-white muco-purulent matter; or, (2.) The expectoration being difficult, the sputa are comparatively scanty, consisting of stringy, tenacious mucus, of a greyish or yellowish-white colour, occasionally streaked with blood; at another time, expectoration being easy, the sputa are more copious, muco-purulent in character, of a yellowish-green colour, having a faint unpleasant odour; at another time the sputa are profuse, almost wholly purulent, of a nauseous and sometimes a fetid odour, usually running together into one mass, but often remaining separated, and forming distinct mummillated masses; or, (3.) There is a profuse expectoration, sometimes to the extent of half a pint in an hour, of a thin, watery, ropy fluid, which varies in opacity, but is usually somewhat transparent, resembling gum-water (FULLER).

Sputa Typical of Plastic Bronchitis consist of ordinary bronchitic sputa, or blood-tinged mucus, with fragments of white fibrinous matter, or white fibrinous casts of the bronchi, which are ejected during violent paroxysms of cough. These concrete masses vary from mere fragments to large pieces of from *one* to *four* inches in length, and may be either tubular or solid, their ejection being preceded and often accompanied by spitting of fluid blood. These casts consist of concentric laminæ, found at different periods in successive layers, and consist of amorphous granular matter intermixed with mucous corpuscles, compound granular cells, oil-globules, and ovoid cells containing dark colouring matter, such as exists in ordinary bronchial mucus (FULLER).

Sputa Typical of Acute Phthisis consist—(1.) Of frothy mucus, often speckled with blood; and when the tubercle softens, the sputa become muco-purulent or purulent. (2.) Of little more than frothy mucus, the sputa being scanty. (3.) Of expectoration, at first scanty, thin, colourless, and transparent, somewhat resembling saliva or gum-water, of a greyish colour, and more or less frothy. After a time the thin colourless sputa lose some of their transparency, and are seen to contain specks of opaque matter, which gradually subside and form a deposit resembling the sediment in barley-water; or they remain suspended by the more ropy part of the secretion, and float in the transparent mucous fluid in the form of striæ. Gradually becoming less aërated, they become more glairy and more tenacious, lose their pearly-grey colour, and are seen to be mixed with specks or streaks of an opaque white or buff colour, and not unfrequently with specks or streaks of blood. (4.) As the malady progresses, the characters of the sputa change again. They become opaque, of a whitish or yellowish hue, and are coughed up in more distinct and homogeneous masses. Sometimes they form rugged pellets of a yellowish-white colour, resembling boiled rice, which sink or partially float in a colourless, semi-transparent, ropy, non-aërated mucous fluid; or, accompanied by little mucous fluid, the sputa form large masses of a buff or yellowish-green colour, flocculent in appearance, but perfectly smooth in outline, which do not coalesce, but remain distinct and separate from each other if expectorated into a vessel of water. All these forms of sputa occasionally occur in *chronic bronchitis* as well as in *phthisis* (FULLER).

Cough.—The severity, the frequency, or paroxysmal nature of the cough must be ascertained; also the circumstances which excite it most; and whether it is attended with pain, or followed by expectoration or vomiting. The ease or difficulty of the expectoration must be noted, and whether it is accompanied or not by pain. The quantity of the sputa ought to be measured in the day and night; and notice should be taken of the form of the masses spat up, their transparency or opacity, colour and viscidity, tenacity or adhesive property. The special characters of the sputa ought in every case to be closely observed, noting particularly its thin, serous, or frothy character; whether it contains any membranous or concrete exudation-masses or blood; and it should be examined microscopically.

Pain.—The exact locality of pain in the chest should be ascertained, its severity, and the direction it tends to take. What particular circumstances aggravate it, and the effects of breathing, coughing, pressure, and postures should be ascertained.

Palpitation.—When *palpitation* occurs, its severity ought to be estimated by laying the hand over the region of the heart of the patient. It is desirable to ascertain its constancy; the circumstances which aggravate its existence or produce it, such as the influence of exertion, going up a hill or up stairs, and the influence of mental emotion (*What to Observe*, pp. 39-44).

Expression of the Countenance in Thoracic Disease is often significant of heart disease. In acute inflammatory affections of the lining or investing membrane, it acquires an anxious and depressed character. An elevation and depression of the *alæ nasi* or *nares* are commonly observed with the respiratory acts; and the occurrence, in children, of these phenomena indicates a greater amount of disease than the general symptoms would lead us to suspect. In chronic cases, when the circulation is impeded, the venous system becomes congested, the face bloated and dusky, the eyelids puffed, the eyes staring, the conjunctivæ suffused, the lips and cheeks purple, respiration laborious, the air-passages loaded with mucus, and the jugular veins distended or pulsatile.

CHAPTER XVI.

DISEASES OF THE HEART AND ITS MEMBRANES.

THERE are few complaints which more surely tend to shorten life, and none give rise to greater suffering and discomfort, than diseases of the heart, pericardium, and great vessels. Alike in the young and in the old, they are the chief causes of sudden death, and if not suddenly fatal "they lay their own hard conditions on the continuance of a man's life, and almost settle beforehand the manner of his death" (Sir THOMAS WATSON). These diseases are to be described as affecting—(*a.*) The *pericardium;* (*b.*) the *endocardium;* (*c.*) the *muscular structure* of the heart; and, (*d.*) the *blood-vessels;* or, (*e.*) as combinations of these lesions.

(*a.*) *Diseases of the Pericardium.*

PERICARDITIS.

LATIN EQ., *Pericarditis;* FRENCH EQ., *Péricardite;* GERMAN EQ., *Pericarditis*—Syn., *Entzüngdung des Herzbeutels;* ITALIAN EQ., *Pericarditide.*

Definition.—*An inflammation of the fibro-serous membrane containing the heart, and investing it on its external aspect.*

Pathology.—(*a.*) *Causation.*—Pericarditis is generally a *secondary* local manifestation of some general disease. It has been described also as *primary* or *idiopathic*—the result of cold; but such cases are doubtful. There is scarcely a form of constitutional malady which may not be accompanied by this lesion; but those with which it is most apt to be associated are *rheumatic fever, Bright's disease, pyæmia* (KIRKES, *Med. Times and Gazette*, October 25, 1862, p. 432), *scarlatina, small-pox, puerperal fever, gout, scurvy, pneumonia, pleurisy, peritonitis, hydatid cysts, abscess in liver or* *mediastinum, inflammation of lungs or pleura, scrofula, tubercle, cancer.* These diseases are mentioned in the order of the frequency with which they give rise to pericarditis (Drs. TAYLOR, T. K. CHAMBERS, A. W. BARCLAY, and Dr. FULLER). In cases of *acute rheumatism,* pericarditis occurs once in every 5·97 cases (FULLER). Pyæmic cases of suppurative pericarditis are sometimes the results of accidents to the mouth and pharynx, giving rise to purulent infection by phlegmonous inflammation of the areolar tissue at the back of the mouth, descending between the pharynx and spine. Destruction of the subserous tissue of the pericardium and pleuræ takes place, and purulent pericarditis. I dissected such a case in Glasgow Infirmary many years ago, where the back of the mouth was injured by a dirty spoon; and Dr. W. T. Gairdner also records a case, p. 16 of his *Clinical Notes.* Similar *continuity of inflammation* by extension of other diseases from neighbouring parts—*e.g.*, cancer of œsophagus, and the like (J. W. BEGBIE); also *pleurisy, pneumonia, chronic cardiac disease, thoracic aneurisms, caries of ribs, tumors and abscesses in the vicinity,* may bring about pericarditis. It may also arise from injury to the pericardium from a wound, or a laceration from a fractured rib (*traumatic pericarditis*).

(*b.*) *Morbid Anatomy.*—The constant motion of the heart upon opposing serous aspects of the pericardium modifies the effects of inflammation, as the exact apposition of the opposing surfaces is constantly changing. In tracing the effects produced by the inflammatory process, the changes which take place both in the epithelium of serous membranes, and in the connective-tissue elements below, ought to be studied. The examination "should not be confined to an examination of the lymph coating, its free surface, or to the mingled liquid and solid products lying in the serous cavity; but the membrane itself should be carefully inspected, not only at its free surface, but for some distance into its substance" (TURNER, *Ed. Med. Journ.*, April, 1864). Like other serous membranes it is liable to inflammation, commencing in hyperæmia, with exudation of liquor sanguinis, migration of blood-corpuscles, and increased activity of growth of the epithelial layer. Effusion of a serous fluid is diffused over a large extent of surface, which sometimes becomes purulent—*suppurative pericarditis,* and the account here given of its morbid anatomy will, *cæteris paribus,* apply to other serous membranes. In the early stages the free surface is no longer smooth and glistening, but becomes covered with a soft "lymph," loaded with rudimentary colourless corpuscles (see p. 68, Vol. I.) These corpuscles are not unlike those which first occur in the fluid of herpes. The changes in the pericardium itself are very early shown in the epithelial cells on its free surface, which become swollen, opaque, and loosened, so as to fall off into the serous cavity. Examined individually, they are seen to be more granular and with multiple nuclei; and thus a rapid multiplication of the contained nuclei takes place (endogenous multiplication), which are set free by the breaking down of the cells themselves. In the corpuscles of the connective tissue beneath the layer of epithelium, germination also goes on; small groups of rudimentary corpuscles appear, formed by germinating changes in the nucleated cells; and these multiply with such rapidity that adjacent groups run together, and form masses of immature corpuscles so numerous in the focus of inflammation that the inflamed part seems to be altogether composed of these new products—all trace of its natural structure being lost. The more superficial layers of this corpuscular inflammatory lymph becomes detached, and falls into the liquid exudation (composed of emigrant blood-cells and the small round newly-formed elements of rapid growth), which pour with more or less abundance into the serous cavity, where they mingle with the fibrinous coagula found in that fluid. When the inflammatory lymph becomes vascular, vessels are seen to pass into it from the connective tissue beneath the epithelium (PAGET and TURNER). The pericardium is seen to be reddened by dense capillary congestion of vessels from the deeper textures, with here and there extravasations of blood. The tissues become infiltrated with serum, and, losing cohesion, are easily torn. As the inflammatory lymph increases, the surface assumes a shaggy appearance, so that fine villi, papillæ, elevations, folds, or masses of new formation are developed by the excessive germination of the young connective tissue corpuscles. More often the appearance of the surface is rough and shaggy, like the villi of a large animal's intestine, or the surface of a bullock's tongue; or the lymph may be smooth as a gall-bladder. These changes constitute the first step in the formation of the so-called false membrane, the adhesive lymph which glues the surfaces to each other. The serous fluid thus seems to coagulate upon the opposed surfaces, and to become vascular. It may be tough and adherent, or soft and granular.

The exudation, which more or less distends the cavity, is separable into a fluid and a more or less solid portion. The liquid part may be scanty, or it may amount to several ounces (8 to 10, but may range from 2 to 60). It first accumulates in the upper and anterior part of the sac, the heart occupying the lower portion. The adhesive inflammatory lymph also first begins to accumulate in this region, especially on the surface of the pericardium, which fits tightly round the heart, and most in the vicinity of the roots of the great vessels. *Post-mortem* evidence of *pericarditis* may be seen here when not found in any other part of the pericardium. When the serum is in great amount, however, the whole of the sac is distended, and the lungs, especially the left, are pressed backwards, and the thorax may be dilated over the region of the heart, whose cardiac dulness is increased. The inflammatory lymph and fluid is generally heavily charged with fibrine, forming reticulated and villous masses upon the walls of the pericardium; so that the aspect of the heart's surface on opening the pericardium is like that of the surface of a sponge, or a network-like lace, or of two surfaces which have been besmeared over with a soft, plastic, sticky substance like lard, which, being placed in contact, have been quickly pulled apart. It is this form which is most commonly met with in the pericarditis of acute rheumatism (NIEMEYER).

The *muscular substance of the heart* may not, in early stages and recent cases, suffer any material alteration; but when the inflammation has continued for some time, the muscular texture of the heart becomes infiltrated with serum, softened and flabby, when dilatation of its cavities may supervene. For a greater or less depth it is deeper in colour than normal, and the finger readily passes through it. The ultimate results upon the pericardium are thickening in proportion to the amount of excessive germination of new tissue, and the amount of inflammatory lymph which is not reabsorbed. If the products are small in quantity, the liquid portion may soon be absorbed; and the solids, undergoing fatty degeneration, or liquefactive degeneration of some kind, are absorbed also. The greater the amount of lymph, the more difficult is the absorption of the products. When thickened parts remain, they present the appearance of tendinous-like bands, or elongated white adhesions connect opposite surfaces together, or there may be more or less extensive gluing together of the two surfaces, which are not found to materially impede the action of the heart. Such terminations may be considered as recoveries for all practical purposes. But if the pericarditis continues for a long time chronic, the thickening of the membrane and new tissue may be very considerable all over the surface; so that permanent damage remains, and exercises an increasingly deleterious influence, even after the main bulk of inflammatory material has been absorbed. A fine fibrous mass of young connective tissue thickens the pericardium, and incloses the heart in a dense indurated capsule (*universal adhesions*).

Partial inflammations continued for a length of time are apt to result in what have been termed "*white spots*," or "*milk spots*," *maculæ albidæ*, or *fibroid granulations.* The *white* or *milk spot* is so often found in hearts which in other respects are perfectly healthy, that many pathologists doubt their morbid nature (BAILLIE, SŒMMERING, HODGKIN, J. REID). The anterior surface of the right ventricle is their most frequent seat. Occasionally these spots are observed upon the surface of the left ventricle, or upon the auricle, or upon the prominences of the coronary vessels. Their size varies from a fourpenny piece to a crown, or larger. They are more common in adult than in early life; but they have been observed in the infant under three months old. They increase after the age of eighteen, apparently progressively with age. About 33 per cent. *post-mortem* examinations, from the ages of eighteen to thirty-nine, show such *white spots;* and about 71 per cent. from ages between forty and eighty. There are two forms of the white spot—namely, (1.) A superficial, which may be peeled off; and, (2.) a deeper spot, which cannot be so detached (BIZOT, PAGET, KING, HODGKIN). Some difference of opinion prevails as to the cause of these spots. Some may be due to previous pericarditis, but all are not of this origin. The weight of evidence seems to be in favour of attrition being the cause of the greater number; yet all are not referable to this cause alone (HODGKIN, KING, JENNER, WILKS). The circumstances which favour the development of this white spot are those which would increase the rubbing of the part against the pericardium applied to the anterior wall of the thorax. Those circumstances are—(1.) Dilated heart; (2.) impeded action of the lungs (*a.*) from those diseases which, leading to augmented volume, tend to press the heart forwards; and (*b.*) from continuous pressure upon the chest in an antero-posterior direction, commenced at an early age, before the epiphysis of the ribs and the pieces of the sternum are fully grown and united, as in young soldiers who, during great exertions, carry a loaded pack and wear cross-belts. *White spots* due to inflammatory origin will generally be found associated with other *post-mortem* evidences of inflammation, such as adhesions by distinct filaments or bands of lymph between the heart and pericardium, especially about the roots of the great vessels.

Pericarditis may also terminate in purulent formation (*suppurative pericarditis*). There may also be multiple and scattered accumulations of pus in the subserous layers, as in cases of pyæmia. Such local pus-formations commence in the subserous filamentous tissue, beneath the cardiac fold of the pericardium, and occasionally assume the form of minute abscesses in the subserous tissue on the surface of the cardiac fibres (CRAIGIE, *Path. Anatomy*, p. 706). In the chronic forms of pericarditis, inflammation may express itself—(1.) By attacking the original membraniform exudation not yet removed; (2.) by thickening and pulpiness of the pericardium; (3.) by the formation of a thick, tough, universally unyielding case; (4.) by hæmorrhage; (5.) by purpura; (6.) by tuberculosis; (7.) by osseous concretions; (8.) by cysts from parasites and succulated conditions of the pericardium, containing pus-like fluid, the remains of old pericarditis—so-called abscess of pericardium (*Path. Trans.*, Vol. IX., p. 89).

Symptoms.—(*a.*) *General.*—The symptoms of pericarditis vary much as regards their expression, and especially in accordance with the coexisting malady with which it may be associated, thus modifying the symptoms of each. In some instances they are most insidious in their approach, as, for example, when pericarditis follows upon *pleuritis* or *pneumonia, scrofula with tubercle* (*tubercular pericarditis*), *Bright's disease, chronic disease of the heart,* or *aortic aneurism;* and participation of the pericardium in the inflammation often remains undiscovered till revealed by *post-mortem* examination. In other cases the symptoms appear to be violent and unmistakable from the commencement. In pericarditis, rheumatism has been found to assume a new and formidable aspect; and rheumatic pericarditis is generally attended with more violent symptoms than non-rheumatic pericarditis. The most marked characteristics are, decided evidence of local trouble in the chest, especially, (1.) pain more or less severe in the præcordial region. From this point it radiates over the whole of the sternum, sometimes extending to the brachial plexus and down the left arm. This pain is accompanied by disturbance of the heart's action, a sensation of constriction over the whole chest, tenderness in the corresponding intercostal spaces, urgent distress, and an incapacity to take a long breath, or to cough. The patient is, (2.) restless and anxious, and this anxious expression of the countenance is peculiar and striking from the first. When acute pericarditis is not the result of rheumatism, the patient may suffer no pain, and the symptoms are often most obscure, general as well as physical. The countenance is pallid, and assumes an aspect of distress. There is an incapacity or unwillingness on the part of the patient to lie on his left side. The pain in the region of the heart may be acute, severe, and shooting towards the shoulder, augmented by movement and increased by pressure upwards against the diaphragm. (3.) There may be febrile exacerbation, the pulse, varying from 90 to 110, full and strong, but often intermittent, or otherwise irregular. (4.) Dyspnœa may exist in proportion to the distension of the sac with fluid pressing on the lungs, and sometimes on the œsophagus, causing dysphagia, pain in the cardiac region, palpitation, and subsequent dyspnœa as the most common signs. This state of things having lasted from three or four days to a week, the patient may die suddenly. Yet all of the signs and symptoms described in books may be absent, as when it occurs in connection with acute rheumatism. The pulse may give forth no sign; the breathing may not be changed; and pains may not exist, and yet pericarditis may be present. The action of the heart may get feebler and feebler, weaker and weaker, the circulation becoming irregular. The pulse at the most may get intermittent, the veins of the neck prominent, the skin cold and pale; and, with œdema of the limbs, death may soon follow, with pericarditis latent, and unrecognised. Before death, and often throughout the more severe periods of the disease, there is *delirium* of a peculiar kind—sometimes quiet, but often wild and furious. This delirium is peculiar, and has been noticed particularly by Sir Thomas Watson, Dr. Burrows, and others.

(*b.*) *Physical Signs.*—When the pericardial effusion is great, it tends to impede, by its pressure, the action of the left lung; and hence the patient prefers in such a case to *lie on his left side*, so as to give more free play to the right lung; or he sits up, but bent forward, in his bed. Generally, the physical signs of pericarditis may be described as follow:—(1.) *Inspection* discloses a distinct bulging of the cardiac region, especially in children and the young. The thoracic walls must be yielding, and the effusions large to permit the production of this effect. Ossification of the costal cartilages, after adult age, is apt to hinder its production. (2.) *Palpation*, to feel the position of the heart's beat, as to whether it is in its proper place, and also as to the force of its impulse. The more copious the exudation the weaker is the impulse, unless in cases of hypertrophy, or violent palpitation. Different positions of the patient influence the results. Standing upright the impulse may be felt; lying down it may not, as the heart recedes behind the fluid which separates it from the thoracic walls. *Friction fremitus* also is said to have been felt by the hand laid over the region of the heart in some cases (NIEMEYER). (3.) *Percussion* may reveal nothing if the lung covers the pericardium, even although exudation may be present to the extent of half a pound. On the other hand, the *form* which the dulness assumes over the heart is one of the most characteristic and important signs. At first the increase of dulness is towards the root of the aorta and pulmonary vessels, and extends upwards to the second rib, or even higher, and passes beyond the right edge of the sternum. When the effusion is very copious, the dulness forms a triangle, with the base to the diaphragm and the obtuse apex alone. As it extends lower down it gets broader, and may pass beyond the right border of the sternum on the one side and the left mammary line on the other. Extension of the dulness in this direction to the left side, beyond the point at which the apex beats, is a positive sign of the existence of a collection of fluid in the pericardium (NIEMEYER). (4.) *Auscultation* brings out the remarkable disproportion between the extreme dulness and the extremely feeble impulse and the inaudibleness of the heart's sounds. Friction-sounds, like those of pleuritis, may be heard before the surfaces are separated by fluid, and may again return as the fluid disappears.

Dr. Stokes arranges cases of pericarditis into three classes, as in the following tabular statement given by the late Dr. F. Sibson:—

First Form.	Second Form.	Third Form.
Slight though general effusion of coagulable lymph:—Absence of pain or local suffering frequent. No sign of muscular excitement, nor any special character of pulse. No increase of dulness over the heart.	*Secretion of serum in abundance, causing distension of the pericardial sac:*—The local and general symptoms more decided, though often very trifling. Irregular action of the heart and pulse, often very manifest in the advanced periods. Remarkable increase of dulness over the heart.	*Muscular excitement, if not myocarditis:*—Local distress, often extreme, even at the outset. Tumultuous action of the heart. Irregularity of pulse. Dyspnœa, orthopnœa, œdematous swellings, syncope, death.

The first stage of pericarditis, *before exudation,* is not discoverable by physical signs (Stokes). This period rarely lasts longer than thirty-six hours. It is to Dr. Stokes, in 1833, that the Science of Medicine owes the description of the most characteristic physical sign of pericarditis—namely, a double *frottement* or *friction-sound.* Others also about the same time had noticed such a sound, and had correctly interpreted its meaning. Bouillaud and Collin, on the Continent, and Drs. Watson, Latham, and Mayne, in this country, had all, independently of each other, perceived and appreciated the symptom—a circumstance which (as Sir Thomas Watson justly remarks) gives to the symptom a greater amount of importance. This sound closely resembles a rasping murmur. It has been named a "*to and fro*" sound by Sir Thomas Watson. It is apt to disappear gradually from below upwards with the increase of effusion, and to return with its decrease. It may disappear from the apex to the base with the progressive formation of firm adhesions. It is usually limited to the region of the heart, but changes its character and its seat from day to day. It is sometimes remarkably modified by local bleeding, passing from a loud rough sound to a soft bellows murmur—most rough and intense during inspiration. The hand applied over the cardiac region will sometimes detect a rubbing sensation, which ceases with the cessation of pericarditis.

Diagnosis.—It is necessary to bear in mind that *friction-sound is not necessarily present in pericarditis.* During the progress of a case, friction-sound may be absent or present for long periods—its presence or its absence bearing no appreciable relation to the intensity of the disease. The amount of fluid effusion and position of the lymph have much to do with this. A really considerable effusion of fluid generally at first muffles, then renders barely audible, and finally removes the sound, becoming indistinct as the heart's sounds are gradually extinguished. Like the heart's sounds, the friction-sound continues audible longest, and is recovered soonest towards the region of the roots of the great vessels. When the lymph is rapidly condensed into firm granulations, and the parts of those granulations most exposed to attrition have become polished and rubbed away, so that the points, gradually receding from each other, present fewer and fewer points of contact, then the friction-sound may subside, although no further effusion of fluid takes place (W. T. Gairdner). Again, *the presence of friction-sound is not necessarily a proof of the existence of pericarditis.* There are permanent exocardial murmurs, probably associated with the well-known "*milk spots*" on the anterior aspect of the heart (Gairdner).

These are short and ill-defined murmurs, especially with the first sound of the heart, occurring in persons otherwise healthy, about the left border of the sternum, at the level of the third and fourth intercostal spaces, or lower. *It is only when the murmur arises for the first time under observation, or when it accurately coincides with the development of symptoms, or where it corroborates and explains the symptoms, and the other physical signs already existing, in such a manner as to leave no doubt of its nature, that we are justified in assuming that a friction-murmur over the heart is pathognomonic of acute pericarditis.* These pericardial or exocardial friction-sounds or murmurs may be, and are often, mistaken for endocardial murmurs (TAYLOR, STOKES, GRAVES, SKODA, SIBSON); and the distinction between exocardial and endocardial murmurs is not always easy, nor is it to be effected by the ear alone.

The exocardial may be distinguished from the endocardial sounds by the nature and nearness of the exocardial sound—by its existence with diastole as well as systole—its limitation to the region of the heart—its non-existence over the great vessels—its variations over different parts of the heart—its rapid and frequent change in character, or its disappearance from day to day—its want of correspondence with the rhythm of the heart, while it seems to follow upon its movements (SKODA), or to precede and follow the impulse (WUNDERLICH)—its co-existence with tactile vibration, and where there is much effusion, with an extensive cone-shaped region of cardiac dulness. The apex of this dull region points to the top of the sternum, its broad base extending downwards to the right, and far to the left of the epigastrium. In addition to these signs, which, when they exist, serve to establish the existence of pericarditis, and separate pericardial from endocardial murmurs, we have other very characteristic signs by which to distinguish every case of pericarditis, especially in the earlier stages. By the aid of pressure, applied gently over the region of the heart, we have a test decisive as to the cause of these sounds, when we are in doubt as to whether it is endocardial or exocardial. *If the noise is that of a valve-murmur, pressure from without does not increase or modify it, except in some anæmic persons, over the aorta. If, on the other hand, it is that of a friction-murmur, soft and bellows-like, of exocardial origin, pressure intensifies the noise, and converts the sound into a rustle or rub. By pressing gently on the costal cartilage or end of the sternum with the stethoscope, the intermediate fluid is displaced, and the roughened surfaces are brought into contact.* This method of diagnosis is most valuable, especially in the early stages, when it is of real importance to arrive at a correct diagnosis (Dr. F. SIBSON, *Provincial Med. Trans.*, Vol. XII., p. 540). "A really refined and intellectual diagnosis," not one founded on the mere *aural* recognition of acoustic characters, is necessary to guard against serious mistakes. In the majority of cases where the friction-sound of pericarditis is recognised, it is known to be such by the circumstances in which it occurs, rather than by the mere character of the sound itself (STOKES, BELLINGHAM, WALSHE, FULLER, GAIRDNER). Increased extent of dulness in percussion, and marked prominence over the cardiac region, are also two characteristic signs (SIBSON, *Med.-Chir. Review*, 1854). When extensive effusion takes place, the heart is pushed upwards to the fourth, third, or second intercostal spaces; consequently, the seat of the heart's impulse, of the rubbing sounds, and of the tactile vibrations, all are correspondingly raised (SIBSON, LATHAM, WALSHE).

A valuable distinctive sign of *pericardial effusion, when contrasted with pleuritic effusion,* is, that when the left side is dull in front and resonant behind, it is a pericardial, and not a pleuritic effusion (STOKES). Any large increase of fluid at once betrays itself, especially in the young, by the protrusion of the left cartilages and ribs, the widening of their interspaces, prominence of the ensiform cartilage, and in some extreme cases by an epigastric fulness or even tumor. When the fluid increases, the pulse becomes feebler, and more disposed to falter and to flutter. It becomes irregular and excited; and often the patient is so fixed in one position that he fears to move, lest he may aggravate by exertion the dyspnœa and action of the heart from which he suffers so intensely. The jugular veins not unfrequently become distended, and this distension does not lessen during inspiration when the effusion is great. A significant sign is thus furnished of the greatness of the obstruction which exists to the thoracic circulation. Œdema and great coldness of the extremities are also apt to supervene with such a state of things. When, however, the products of inflammation become solid, and little serum remains, the pericardium, by the opposed serous surfaces, becomes attached to the heart throughout, and the pulse then resumes its force and regularity; and, if the patient survives, this adhesion remains for life.

Prognosis.—While the consequence of pericarditis "is sometimes, though rarely, the speedy extinction of life, in nineteen cases out of twenty the disorder proves fatal at a remote period, destroying the subject of it more slowly, indeed, but almost as surely." Such is the opinion of Sir Thomas Watson; and again he writes, "I am certain it is a fertile, but often unsuspected source of chronic disease of the muscular substance of the heart, and of its consequences, asthma, dropsy, sudden death." The mode of death tends to be by *asthenia,* or by *syncope* induced by paralysis of the left ventricle. A fatal termination may occur, although rarely, in less than forty-eight or thirty hours. In ordinary cases which progress favourably, the disease generally begins to yield in the course of a week or ten days, and sometimes sooner under active treatment. It is not now, however, generally believed that when pericarditis terminates by adhesion, dangerous or fatal lesions of the heart always tend to supervene. It is consistent with the experience of Drs. Stokes, Sibson, Wood (of Pennsylvania), W. T. Gairdner, Smith, Wilks, Moxon, and other observers, that hypertrophy and dilatation of the heart do not commonly follow on pericardiac adhesion. Sir Thomas Watson, however, with many others, regards adhesion of the pericardium as a suspicious prognostic of future evil—"that other structural changes will soon or slowly develop themselves, and first render life burdensome and full of suffering, and then consign the patient to an earlier grave than might else have awaited him." Among the patients of a general hospital, from 6 to 8 per cent. die of pericarditis, while a proportion varying from 3 to 8 per cent. have had the disease at a previous period and recovered from it (W. T. GAIRDNER). Generally, it may be stated, that there are differences in the results of pericarditis in the healthy compared with the results in the unhealthy, sickly, weak, "worn out," pale, or scrofulous. A person otherwise in fair health may survive a first shock of such a disease, but with a damaged organ for life. The sickly, weak, and scrofulous linger on, victims to palpitation or dyspnœa, often approaching to suffocation, and after some weeks die suddenly. The worst class of cases are those in which pus exists with the serum. The more serum there is with lymph, the more unfavourable is the case, especially if no indication of absorption exists. Where there is only inflammatory lymph occurring in an otherwise robust person, the case is not unfavourable. When pericarditis accompanies *pneumonia*, *pleurisy*, or *acute articular rheumatism*, the result, so far as pericarditis is concerned, is generally favourable. *Rheumatic pericarditis* proves fatal in about one out of every six cases (FULLER). When it complicates *Bright's disease*, disease of *the heart*, *aneurism*, or *scrofula* with *tubercle*, or in the *purulent* form, as in *septicæmia*, it is generally fatal. But even in the cases that do appear to recover, remote effects may still result, especially chronic pericarditis—the inflammation sooner or later breaking out afresh—which rarely terminates in complete recovery, and which of itself does not directly prove fatal, but generally indirectly through œdema of the lungs and slow suffocation. The exudation in such chronic cases is generally extremely profuse, and the dyspnœa severe. Relapses of inflammation seem to follow relapses, and the substance of the heart itself becomes extremely soft, relaxed, and discoloured. The circulation then begins to indicate distress, the pulse becomes small and irregular, the venous system overloaded, and general dropsy sets in. But depression of the cardiac action is generally most intense in the cases of suppurative pericarditis, a result which may be not less due to the constitutional disease than to the local lesions. The most common sequelæ of pericarditis are as follow:—(1.) Adhesion of the opposed surfaces of the pericardium, with more or less complete obliteration of the pericardial sac. (2.) Dilatation of the heart, the more likely to result in proportion to the length of the attack, and the amount or extent of adhesion. (3.) Hypertrophy of the heart, if its texture be sound; or, (4.) Atrophy and fatty degeneration, from constant chronic infiltration of the heart's texture, as well as from the pressure of the surrounding morbid pericardium.

Treatment.—The results of recorded treatment are unsatisfactory in the highest degree. Sir Thomas Watson writes, that "in a large proportion of the cases, whether they be treated well or ill, or not treated at all, the patients will seem to recover." Although blood-letting and calomel have hitherto been regarded as two most efficient agents in accomplishing the indications of cure, yet Taylor's analysis of cases shows that pericarditis may come on during salivation. The late Dr. Parkes informed me that he has seen this occur two or three times. It is, then, very doubtful if calomel ever does any good in pericarditis, notwithstanding that its use, so as to affect the gums, is advised by Watson, Graves, Stokes, and Fuller. Professor W. T. Gairdner, Drs. John Taylor, of Huddersfield (*Med. Times and Gazette*, 1849), and J. Risdon Bennett, of London (*Lancet*, Dec. 6, 1851), are all unfavourable to such a use of mercury. In all the *constitutional diseases* in which pericarditis occurs as a complication, mercury is certainly contraindicated. In rheumatism and in Bright's disease, which furnish by far the larger number of cases of pericarditis, mercury is decidedly objectionable; and it is now well known to be productive of most injurious consequences in Bright's disease. Its use tends to weaken the heart and the system at large, and thus proves a source of additional irritation to the cardiac tissue, by undue reaction on the system.

Venesection is never to be practised. Treatment by bleeding may be carried out by leeches over the præcordial region, or by cupping there in exceptional cases. When the pain is very marked, where it has strongly the characters of angina, leeches are to be applied in moderate numbers over the præcordial space. From four to six leeches (W. T. GAIRDNER), ten to twenty (NIEMEYER), so applied, according to circumstances, followed by fomentations, very commonly relieve the pain, and rapid improvement follows. To judge as to the repetition of blood-letting, the force of the heart must be observed, as indicated by the pulse at the wrist, by the actual strength of its impulse, and by the character especially of the first sound. If the impulse continues vigorous, and the first sound is undiminished, blood-letting may be repeated by the further *local* abstraction of blood by leeches; but if the impulse has manifestly declined in force, while the first sound is lessened, great caution is required in the further abstraction of blood. *Fomentations*, sometimes plain and sometimes medicated with *opium*, are the chief local remedies to be afterwards relied on. In the second stage of the disease, when liquid effusion distends the pericardium, some reliance may be put in treatment by a blister of a large size over the præcordia. *Diuretics* and *iodide of potassium* are also to be given. "Stimulants are often imperatively called for. The weakened heart requires to be supported and invigorated. This may be effected by the use of *wine*, and by the use of *opium*." If depletion has been excessive —if signs of muscular weakness supervene—if there be evidence that the heart, previous to the attack, was in a weakened state—if a tendency to collapse or to a typhoid state manifests itself—"we must give wine, quite irrespective of the physical conditions of the heart" (STOKES). Niemeyer recommends *digitalis* as a suitable remedy in cases where the beat of the heart is very frequent and insufficient, causing cyanotic and dropsical symptoms. Its effect is then very markedly beneficial. *Opium*, in doses of one grain (*i.e.*, stimulant doses) every third hour, seems "to expend itself solely on the disease," and its beneficial effects are seen to result when it does not produce contraction of the pupil, nor headache, hot skin, furred tongue, nor constipation (CORRIGAN, STOKES, GRAVES, SIBSON). *Battley's solution* is perhaps the most useful preparation. This remedy must be watched closely. There are two more important circumstances to be attended to—namely, that our treatment of pericarditis must depend upon the stage of the disease in which it is first discovered to exist; and it is important to know that pericarditis from acute rheumatism calls for a totally different line of treatment from that associated with Bright's disease, or diffused inflammation of a low type (SIBSON). In the *rheumatic form*, *colchicum*, in the form of a draught, and the administration of *alkaline remedies*, are indicated by the constitutional state. Warmth, especially moist warmth, and flying blisters are the best agents to promote absorption. Niemeyer considers that the use of cold applications, such as ice-bladders, applied over the cardiac region, is deserving of great reliance; others condemn cold applications.

Paracentesis is to be recommended in all those cases in which the effusion is so great as to cause alarming distress, orthopnœa, obstruction to the venous circulation, and serious interference with the heart's action. In such cases a fine exploring trochar and canula is to be introduced (not plunged), so as to make a valvular opening below the heart, either to the left of the xyphoid cartilage, or through the fifth intercostal space, close to its anterior extremity, and the fluid drawn off by means of a syringe (SIBSON, in *Med.-Chir. Review*, July, 1854). The result may be merely palliative; but, as Niemeyer justly remarks, "even to afford the sufferer opportunity, after the operation, to pass the night in his bed (perhaps for the first time in a long period), and to enable him to sleep a little, is a great gain."

The general principles of safe treatment are summed up by Dr. W. T. Gairdner as follow:—(1.) To make large allowance for the insignificant and spontaneously healing class of cases revealed more by physical signs than by symptoms, and to regard them as demanding little active treatment; (2.) to consider rheumatic pericarditis in general as a disease susceptible, to a great extent, of cure under palliative local remedies and fitting constitutional treatment; (3.) to hold the general treatment as subordinate to the constitutional treatment of the disease with which the pericarditis is associated.

TUBERCULAR AND HÆMORRHAGIC PERICARDITIS.

LATIN EQ., *Pericarditis Tuberculosa;* FRENCH EQ., *Péricardite Tuberculeuse;* GERMAN EQ., *Tuberculöse Pericarditis;* ITALIAN EQ., *Pericarditide Tubercolase.*

Definition.—*Pericarditis in a scrofulous constitution, with miliary tubercles developed in the young membranous tissue of chronic pericarditis, and attended with repeated outbreaks of inflammation.*

Pathology.—In the scrofulous cachexia (Vol. I., p. 1000), where *acute miliary tuberculosis* of the lungs exists, pericarditis with minute tubercle on the pericardium may be also found. It is a common form of pericarditis in the army, and its invasion is very insidious, so that without physical examination its recognition and diagnosis may not be possible. It is also often rapid and acute in its results. The greyish granules of miliary tubercle on the pericardium do not undergo further change to softening, but the patient dies rapidly of the tubercular fever, often without betraying any symptoms of the existence of tubercles in the pericardium, or of pericarditis. It is also one of the forms of chronic pericarditis, so that inflammatory attacks are frequently repeated. There is generally a hæmorrhagic effusion in the sac in such cases, and where there is a disposition to purpura or scurvy, or during the progress of some malignant febrile disease, it occasionally happens that blood as well as serum or pus is effused within the sac (*Hæmorrhagic pericarditis*). The source of the blood is from the new-formed vessels of inflammatory exudation, which, being yet tender, delicate, and thin-walled, give way (see Vol. I., p. 103); or it may be from the highly congested vessels of the serous membrane, and the softened tissues which prevail in scorbutus. The lymph of such cases is of a spongy red colour and shaggy appearance, exactly similar to that which exudes from the large intestine into the small gut in cases of scorbutic dysentery. If the blood is small in amount, the serum of the pericarditis has a reddish colour, but if the flow be considerable, the effused mass may resemble a pure extravasation, and assume a blackish colour. The lesion sometimes occurs in recent pericarditis,—occurring in cachectic patients, such as those suffering from *chronic alcoholism*, *scrofula*, and *Bright's disease.* The walls are studded with translucent prominences of the pericardium, which may afterwards become yellow and cheesy. Blood in the pericardium may also be the result of rupture of cardiac or aortic aneurism, or from a diseased coronary vessel.

ADHERENT PERICARDIUM.

LATIN EQ., *Pericardium Adhærens;* FRENCH EQ., *Péricarde Adhérente;* GERMAN EQ., *Verwachsung des Pericardiums;* ITALIAN EQ., *Pericardio Aderente.*

Definition.—*More or less adhesion of the opposed surfaces of the pericardium by inflammatory exudation (adhesive lymph) becoming organised, and with more or less complete obliteration of the pericardial sac.*

Pathology.—When lymph has been effused, it is commonly found organised, so that the pericardium is partially or universally adherent all over the heart. In some instances the lymph effused, instead of forming adhesions, becomes converted into cartilaginous, and even osseous patches, which are readily detached from the surface of the heart by the scalpel. *Parasitic cysts* may also establish adhesions, as well as *fatty* and *fibroid growths.* The adhesions are thus sometimes partial and sometimes total: sometimes consisting in a firm agglutination of the opposed surfaces, or merely in a few long bands and fibres. In some cases the remaining pericardial tissue is thus transparent, atrophic, and dry, so that the heart appears as if it were almost naked; in other cases the pericardium is converted into a dense, indurated, unyielding case, or cartilaginous mass. The adhesion may be so indurated, that remains of effusions may still exist, in a sacculated condition, sometimes purulent (so-called abscesses of the pericardium).

Symptoms.—The subsequent degeneration of the heart, which an adherent pericardium entails, brings about functional difficulty in its action, which may be still further aggravated by valvular disease or other cardiac lesions. The more dense and unyielding the inclosing pericardium is, the greater is the functional difficulty of the heart. The pulse becomes extremely small and always very irregular; dyspnœa, cyanosis, and dropsy very soon appear. A sinking of the intercostal space with the heart's beats, and a rising again during diastole at the region of the apex impulse, is considered a sign of value, as indicating an adherent pericardium; but the symptom is often wanting (NIEMEYER), and no symptoms are distinctive of the lesion (see also WILKS in *Guy's Hospital Reports*, Vol. XVI., p. 196, 1871).

Treatment must be regulated by the functional difficulties of the circulation, and by the source to which those difficulties may be traced.

DROPSY OF THE PERICARDIUM.

LATIN EQ., *Hydrops-pericardii;* FRENCH EQ., *Hydro-péricarde;* GERMAN EQ., *Herzwassersucht*—Syn., *Hydro-pericardium;* ITALIAN EQ., *Idro-pericardio.*

Definition.—*An increase of the normal liquor pericardii, containing little albumen, and analogous to the fluid of chronic hydrocephalus.*

Pathology.—(*a.*) *Causation.*—Increased amount of fluid in the sac of the pericardium occurs in cases of wasting, with shrinking or diminution in the bulk of the heart (*Hydro-pericardium ex vacuo*). The veins of the heart being obstructed also lead to increase of fluid. It is thus seen in diseases of *mitral valves, emphysema,* cirrhosis of the lungs—diseases in which the right side of the heart is working under abnormal conditions. In other cases it occurs as a part of general dropsy, as in *Bright's disease, chronic affections of the spleen,* or *cancerous conditions* of the system.

(*b.*) *Morbid Anatomy.*—When the fluid is large in amount, the pericardium is dull, white, and without lustre; the fat has disappeared from the surface of the heart, and its connective tissue is œdematous. The quantity of serum effused varies from a few ounces to a few pints. It is simply serum. Louis has given one case in which the effusion amounted to four pounds, and Corvisart another in which the quantity was still more considerable. Walshe records sixty ounces; and when such great effusions occur, the diaphragm is depressed below the end of the sternum by the great amount of fluid contained in the sac of the pericardium.

Symptoms.—The condition is always secondary, and, therefore, symptoms beyond the physical signs of effusion are to be sought for in connection with those lesions of which it is a more or less constant result. Pyrexia is absent, and there is no marked disturbance of the heart's action. Friction-sounds are absent, and it generally follows or accompanies hydrothorax.

Treatment must be regarded by the attendant circumstances of the case, and in accordance with what has been already written on the subjects of pericarditis and dropsy. Paracentesis may be required.

(*b.*) *Diseases of the Endocardium.*

ENDOCARDITIS.

LATIN EQ., *Endocarditis;* FRENCH EQ., *Endocardite;* GERMAN EQ., *Endocarditis;* ITALIAN EQ., *Endocarditide.*

Definition.—*An inflammation of the lining serous membrane of the heart, covering the valves and lining the chambers of that organ.*

Pathology.—(*a.*) *Causation.*—The inner membrane of the heart is exposed to the action of substances which may be taken up by the absorbents and introduced into the circulation. Of all substances *alcohol* has the most striking effects on this tissue. It is not only proved to be absorbed and actually to circulate in the blood; but there are few drunkards the inner membrane of whose heart and large vessels is not more or less diseased; so that *alcohol* probably acts as a specific poison on the endocardium. The morbid conditions of the blood associated with *Bright's disease,* with *rheumatism* and *gout,* and with *syphilis,* appear also to act especially upon this tissue, and patients who suffer from these diseases may ultimately die of some form of endocarditis, sometimes extending along the lining membrane of the aorta. Diffuse inflammation of the endocardial membrane has also been known to follow the application of a ligature round an artery. Such cases of traumatic origin are rare. Bamberger records two. Infective processes (attributed to the presence of minute organisms (*mycosis*) by certain German observers) have also been set down as a cause of *ulcerative endocarditis.* The mixture of white fibrous and elastic tissue in great abundance at the valvular reduplications, renders those parts prone to disease in the constitutional states of the system in which the fibrous textures are more particularly involved, as in rheumatism; and the great majority of cases of *endocarditis* arises in the course of *acute articular rheumatism,* all the more readily the greater the number of joints attacked. About 20 per cent. of the cases of acute articular rheumatism are said to be complicated with *endocarditis* (BAMBERGER). *Bright's disease* is the morbid state next in frequency which is complicated with *endocarditis,* especially the acute form developed after scarlet fever. *Acute infectious disorders,* especially puerperal fever and measles, are also prone to be complicated with *endocarditis.* Wunderlich regards *measles* as the most prolific cause of endocarditis next to *rheumatism.* In all such cases it seems not improbable that the irritant which sets up the inflammation, acting mainly on the tissue of the narrow passages through the heart—its orifices and valves—is the super-heated blood of the fever-patient, which marks the intensity of the fever (BILLROTH, WEBER, NIEMEYER). Tear and wear may also be set down as a cause. The parts affected are the parts on which the great tear and wear on the action of the heart are expended, and thus they are probably the first to suffer, owing to the mutual friction of the valvular edges upon each other.

(*b.*) *Morbid Anatomy.*—By a species of selection (the reasons for which are in a great measure speculative), the coverings of the orifices and valvular structures of the heart are by far the most frequent seat of lesion in the inflammation of the internal lining membrane of that organ. The peculiarity of the minute structure of these parts, as influencing the arrangement assumed in the first instance by morbid deposits, seems to have been first definitely stated and illustrated by Sir Thomas Watson, (*Lect.* lxi., p. 275, 3rd Edit.) There is found to exist, inclosed between the reduplications of the endocardium, a quantity of fibrous tissue. An increase in its amount takes place at the centre of each aortic valve, forming the *corpora Arantii,* and at each of the extremities or angles of the segments. The minute exudations which are formed as the result of the inflammatory process in the endocardium may be seen to arrange themselves in double festoons, suspended as if from the *corpora Arantii,* often in a row, like a string of beads, along the line of union of the thick portion of the valve, with the inner convex margin of its two thinner crescentic portions, forming fringe-like or fibrinous warty outgrowths or excrescences. The repeated attrition of the opposed surfaces of the valves pushes aside the exudation from the thin crescentic portions of each valve as fast as it is deposited, and while yet plastic, and so heaps it up along those boundary lines of contact; "just as a thin layer of butter on a board would be displaced and heaped up in a little curvilinear ridge by the pressure of one's thumb." The inner *arterial tunics* and the *endocardium*, when they suffer from inflammation, suffer from the *parenchymatous form*, most important in pathology (see Vol. I., p. 65), namely, that form in which active disturbances of nutrition are provoked by an irritation; but which, instead of producing an exudation *between and amongst* the elements of the tissues, causes a *swelling* of the individual normal elements themselves, resulting in proliferation of their cells and increased volume of the part. Thus, the general result arrived at from the most recent observations on the inflammatory process by Virchow, Lister, Turner, Moxon, and Wilks, especially tend to confirm the statements of the late John Goodsir, that "*an explanation of its phenomena is not to be looked for by referring them to actions of the extreme vessels, but to a disturbance of the forces which naturally exist in the extravascular portions of the inflamed part*" (*Anat. and Path. Observations*, 1845, p. 43). The inflammation of endocarditis does not originate in the deep layers of the endocardium, but in its extravascular and superficial minute elements of tissue; hence silvery opacity is the first permanent change, marking after death the previous existence of parenchymatous inflammation. The minute elements of the tissue become swollen—enlarged individually, by the infiltration of a liquid, the chemical properties of which resemble *mucin*—a *mucinous* inflammation, the fluid of which coagulates into the form of threads upon the addition of acetic acid. A vast formation (excessive germination) of new cell-growth at once commences, each new growth, as the inflammation advances, rising up like a Phœnix out of the ashes of the old. These new cells become organised into connective tissue, which often develops into reddish or greyish-red delicate villi, as the disease advances, giving the endocardium a finely granular aspect, or covers it with coarse granular wart-like papillæ, firm and hard at their base, with round, bulbous, soft and gelatinous free ends. These constitute the valvular vegetations, to which deposits of fibrine from the blood adhere, which must be distinguished from the vegetations themselves. In some rare cases ulceration takes place—*ulcerative endocarditis*. Enlargement, cloudiness, and excessive germination of cells go on with such activity that the tissue softens and breaks up, producing loss of substance and ulceration of the endocardium—a condition of great danger. It is often the starting-point of *true metastasis* by *embolism*. The minuteness and friability of the parts favour the penetration of the softened and separated particles into the smallest capillary blood-vessels of distant parts, such as the *spleen*, *kidney*, *brain*, and *heart* (*capillary embolia*), and hæmorrhagic infarction, fibrinous accumulation (or metastatic abscess, which is rare) may be the result. It is rare for the blood-vessels of the *liver* or the *lungs* to be so affected on account of the direction and mode of the *arterial* connections. If particles of fibrinous clots, and such like *débris* of ulceration, pass into the *carotid* or *vertebral* arteries, then, according as the artery of the brain is totally or partially occluded, will be the formation of hæmorrhagic foci (*capillary apoplexy*), with their consequences, or partial anæmia may result, and consequent death of the anæmic part of brain (*yellow softening*). Similar occlusion of a large blood-vessel of the extremities may lead to gangrene of the fingers or toes.

The inflammation hardly ever affects the whole lining of the heart, but usually is limited to patches of varying size; proceeding, in the first instance, mainly from the valves and textures composing the orifices. At *post-mortem* examinations, reddening of the texture (the result of imbibition merely of the colouring matter of the blood) must not be mistaken for the redness of inflammation, which it is only possible to see in rare instances (ROKITANSKY). Redness from imbibition is quite superficial, and no individual vessels (*vasa vasorum*) are to be seen; but reddening from inflammation exists also in the deep as well as superficial parts, and the hand lens may show the larger capillaries filled with blood to bursting (FORESTER). The prolonged existence of the inflammatory state ultimately thickens and hardens, by interstitial deposit, the tissue inclosed between the folds of the serous membrane constituting the valves, so that their action is much impaired.

Laceration and fissures of the relaxed and softened endocardium sometimes occur. The *chordæ tendiniæ* may give way, thus obviously and suddenly interfering with the tension and function of the valve (see some cases of this kind, with drawings, in the records of the Naval Medical Department for 1870). Sometimes the lappet of a valve, or one surface of it, is torn, or the endocardium giving way over the muscular structure of the heart, blood may be forced continuously through the rupture, so as to tear asunder, more or less, the cardiac muscular fibres, and so produce a *true acute aneurism* of the heart. Some examples of this may be seen in the Museum of the Army Medical Department in the Royal Victoria Hospital at Netley. These aneurisms are circumscribed sacs, varying in size from a filbert to a walnut, bounded at its entrance by a torn and ragged endocardium, its walls formed by the forcibly separated fibres of the muscular substance compressed against the pericardium.

Symptoms.—(*a.*) *General.*—The patient prefers to lie on his back (*dorsal decubitus*), and he may perhaps incline to toss about with his arms. Pyrexia may prevail of a specific kind, as when *rheumatism, Bright's disease*, or *typhus fever* is present, or it may be *idiopathic inflammatory fever*, associated with the endocarditis. So long as the cardiac orifices are not seriously obstructed, and no obstruction exists in the lungs from pneumonia or bronchitis, no special sensation of dyspnœa is complained of. The pulse ranges in frequency between 80 and 120; and it has been stated by Dr. Taylor even to lose in frequency and energy at the outset of the affection, a result which may be due to the infiltration of the heart's texture with serous fluid. More or less headache may prevail. Generally, however, the patient may not complain, even when special inquiry is made regarding the condition of the heart.

(*b.*) *Local.*—A more extensive, forcible, and abrupt impulse of the heart than natural, combined with endocardial murmurs of a soft, low tone and blowing sound, in a febrile state of the system, and with cardiac uneasiness, are signs suggestive of the probability of endocarditis. A careful study of the development, order of occurrence, and combination of the general symptoms and physical signs, can alone convert that probability into a certainty. Discomfort and uneasiness at the heart, with more or less palpitation, may be present. The extent and power of the heart's impulse ought to be examined carefully and repeatedly; and the conditions which tend to subdue or to aggravate these phenomena ought to be ascertained. The areas of the heart's dulness, both superficial (in breadth) and deep-seated, undergo increase (BOUILLAUD, SKODA, WALSHE). Its impulse is almost always more extended and stronger than natural at the commencement, till infiltration of tissue takes place, when the pulse and force of the heart become small and soft. The heart's sounds undergo modifications as soon as the tissue of the valves become changed in texture, substance, and shape, by the products of inflammation. The murmurs which accompany purely acute endocarditis, arranged in the order of their frequency, are—(1.) Aortic obstructive; (2.) mitral regurgitant; (3.) aortic regurgitant; (4.) aortic obstructive and mitral regurgitant together (WALSHE). (The student is referred back to page 566, *et seq.*, for the account of the sites of maximum intensity where these murmurs may be listened to.)

The comparative frequency of aortic and mitral valve disease in civil life is not easy to estimate. It has been determined with more or less accuracy from the combined observations of Drs. Barclay, Chambers, and Ormerod, as tabulated by Dr. Sibson. The *mitral valves* are more subject to disease than the *aortic*. *Aortic valve disease with hypertrophy* is the commoner form in the army. The disease is often limited to one set of valves, but it is more often common to both valves than limited to either. When associated with *acute rheumatism,* the endocarditis affects both valves in the greater number of cases; and the *mitral* more frequently than the *aortic.* In the young, who are subject to *acute rheumatism,* disease of the *mitral* valve, and in the old, who are subject to *atheroma,* disease of the *aortic* valve predominates. In the more severe cases in which the valve disease is itself the cause of death, the *mitral* valve disease is shown to be the most prone to go on to a fatal issue (*Med.-Chir. Review,* Oct., 1854, p. 431). But *aortic regurgitation* is the most swiftly fatal.

Diagnosis.—As there is no difference in character between the murmur of endocarditis and that which attends established valvular disease, it is necessary, in order to appreciate the existence of endocarditis, that the *murmur should be developed under observation at the early period of an acute attack* (WALSHE): and if a mitral or aortic murmur supervene while a case of *acute rheumatism* is being watched, especially if there be congestion and an expression of anxiety in the face, with distress in the region of the heart (*not caused by pericarditis*), there is a strong probability of endocarditis (SIBSON). The substitution of an abnormal murmur at the apex for the *first* cardiac sound is the most frequent and important sign (NIEMEYER). But the symptoms are often exceedingly insidious in their origin and progress, and the disease is rarely simple, being generally combined with pericarditis. Moreover, as the *general* constitutional symptoms of these two diseases do not differ, the detection of endocarditis, *per se,* is one of the most difficult in practice. Like pericarditis, it is often latent (as in rheumatic fever), and the practitioner is surprised by his patient showing symptoms of valvular disease after an apparently perfect recovery from fever (STOKES). A murmur *per se* is no sufficient evidence of endocarditis. Stokes, Sibson, and Graves have each of them recorded cases where *mitral*, and still more often *aortic*, murmurs have been generated, when no valvular disease existed,—in cases of fever, in a case of fatty degeneration of the heart, and in a case of pericardial adhesion. These murmurs have also been recorded during life, when no trace of valve disease was observed after death (Drs. BARCLAY, MARKHAM, CHAMBERS, W. T. GAIRDNER, and SIBSON). As in pericarditis, it is important to recognise the *friction-sound* pathognomonic of its existence (apart from any endocardial murmur with which it might be confounded); so in endocarditis it is, if possible, still more important to detect endocardial murmurs when masked by pericarditis, for the grazing sounds of the latter disease may altogether mask those of the valve murmurs. The principles on which the diagnosis is to be effected are involved in the facts that friction-sounds of pericarditis are limited to the heart's region (STOKES); while the sounds of the heart, and the murmurs which attend the lesions of its valves, are propagated in certain determinate directions; and while they are heard in maximum intensity at certain points, more or less defined, they may be detected by following the line of propagation at points beyond the mere limits or region of the heart itself. Upon these grounds data are furnished by which to distinguish the murmurs of endocarditis. (See p. 566, *et seq., ante.*) They must be looked for in suspicious cases from day to day *beyond the region of the heart;* and if a *systolic mitral murmur* is heard extending an inch and a half beyond the nipple, it is most probably due to *mitral regurgitation* (SIBSON). The detection of an *aortic murmur* with pericarditis is much more difficult, because the *friction-sound, frottement,* or "to and fro" sound, often mounts to the top of the sternum. The *aortic murmurs* are therefore to be listened to in the line of the natural propagation of the aortic sounds; and if an *aortic murmur* exists, it can only be distinguished in the neck—the best point for examination being just above the sternum, a little to the right, over the *innominate artery.* If, after listening to the first sound, the second sound be observed to follow clearly and distinctly, the chances are that there is no affection of the aortic valves, even if there be a loud systolic murmur. If, however, the second sound be indistinct, inaudible, or prolonged, or replaced by a diastolic murmur, aortic endocarditis may be suspected or detected (SIBSON). (The student is requested to contrast p. 566, *et seq.*, with these statements.) As far as the immediate practical value of the information is concerned, it seems to be really unimportant where the exact seat of the murmur is. It is of no practical importance, for example, in the first instance, whether the murmur proceeds from a "mitral, a tricuspid, or a semilunar valve, or whether it may be due to a contraction, or a dilatation, an ossification, a permanent patency, or a warty excrescence." The practical points to be determined, in the first instance, reduce themselves to two—namely, *First,* "Do the murmurs proceed from an organic cause?" *Second,* "What is the vital and physical condition of the texture of the heart itself with which they are associated?" For subsequent practical purposes the limits of inquiry may be circumscribed to the recognition of the occurrence of contraction or of dilatation of the orifices, because both of these conditions are attended with a permanently open state and permanent valvular disease. While the occurrence of murmurs and their nature, and the circumstances under which they are developed, are of the utmost importance to establish the existence of an endocarditis, the condition of the muscular substance of the heart must be the great guide in prognosis and treatment. The vital and mechanical state of the heart's cavities must be ascertained. The action of the heart must be carefully noticed at different times, as to whether its force and vigour are above or below the natural standard—whether it is liable to excitement from slight causes—and whether it tends to regular or irregular action, as regards rhythm or frequency of revolutions. An extremely feeble pulse, with scanty filling of the arteries, implies implication of the texture of the heart. The duration and origin of the disease must be ascertained; and how far the brain, kidneys, or spleen suffer from the mechanical or vital effects of the lesion (STOKES).

Another class of symptoms and results are apt to be associated with endocarditis and valvular disease, as when the products of inflammation poison or spoil the blood, which are cast off in small fragments into the blood stream (*multiple embolism*). Rigors, heat of skin, profuse perspiration recurring irregularly, dull, earthy-yellow discoloration of the skin (not of the conjunctivæ), diarrhœa, more or less bilious, pinched anxious countenance, intense prostration, and muttering delirium, are the symptoms which announce this untoward occurrence (WALSHE.) Secondary deposits in spleen, kidney, or the brain are the records of its morbid anatomy. But these secondary deposits may not be the direct result of existing endocarditis. Virchow, Kirkes, Simpson, and Rokitansky have shown how the fibrinous coagula, which have become permanently attached to the valves, covering the vegetations upon them, may become worn away superficially, and taken into the blood in fine particles, thus leading to secondary coagula (as shown at p. 604) in the capillaries of the spleen and kidneys, to obliteration of these vessels. "Infarcts" or wedges of fibrine thus accumulate. In the capillaries of the brain they lead to softening, and sometimes to sudden death. Vegetation may be growing in rank luxuriance over the valves, and yet there may be no præcordial distress, no local symptom of any sort to awaken suspicion of mischief. But the physical signs are ever changing from day to day, in correspondence with increasing changes in the form, site, and volume of the vegetations. The systemic phenomena are, severe pyrexia, and if infection (mycosis or micrococci?) be present, then symptoms of septicæmia will follow, with delirium and petechiæ, the result of capillary embolism. The fever and temperature (105° and upwards) is of the hectic or remittent type.

Prognosis.—So far as endocarditis is concerned, the immediate prognosis is similar to that stated under pericarditis; but the future changes of life being prolonged depend upon the lesions which remain permanent. Lesions of the valves are the most common result (*valvular diseases of the heart*). After an attack of endocarditis, there may be no defect of valves capable of physical recognition. The valve-texture may be nevertheless damaged, so that shrinking, atrophy, and retraction of the damaged tissue, commencing gradually, progresses very slowly. No valvular disease may appear for months after an attack of endocarditis; and any pain in the region of the heart which may have attended such an attack may be quite forgotten. If valvular lesion is fully established, and remains persistent, *hypertrophy* is certain to follow, and the danger will be the greater in proportion as *dilatation* of the cavities of the heart predominates over hypertrophy. Under all circumstances *dilatation* is a most serious disease; increasing directly as the excess of the capacity of the cavities over the thickness of their walls; as the softness and flabbiness of the heart's tissue; and as the general deficiency of tone in the system and impoverishment of the blood. Once dropsy has supervened, life can with difficulty be prolonged beyond twelve or eighteen months. The usual termination of *endocarditis* is death from the resulting lesions of the valves; and it is rare for a patient to die suddenly from endocarditis alone, unless in the course of *Bright's disease.* The fatal results do not usually ensue until a lapse of years, when the valves, though acting normally and efficiently for a time, at last begin to undergo changes, and become so deformed that their functions are at last impaired, and valvular disease is confirmed. An extremely feeble pulse, with scanty filling of the arteries, is a bad sign, showing that the muscle-texture of the heart itself is implicated. Rigors are an unfavourable sign; also sudden swelling of spleen, pain in that region, vomiting and albuminuria, or hemiplegia—the signs of true metastasis. If neither aortic lesion, hypertrophy, nor dilatation results, the tissue of the heart itself may be so impaired as to lead to softening, like what occurs in typhoid and typhus fevers, scurvy, or purpura. Fatty degeneration of the cardiac tissue is also a result to be apprehended. The valuable medical reports of St. George's Hospital, London, prepared by Drs. A. W. Barclay and Rogers, contain the following statistics relative to the percentage of mortality *among hospital patients* from *diseases of the heart* during a period of six years, given in the order of increasing mortality—namely, from *hypertrophy,* 60·5; from *dilatation,* 52·1; from *pericarditis,* 34·8; from *valvular disease,* 24·5; from *endocarditis,* 9·19.

Treatment.—No defined line of treatment can be laid down suitable for all cases; because the circumstances under which endocarditis occurs are extremely varied. What has been written relative to *pericarditis* applies equally to *endocarditis;* but the management of cases in which the patients suffer from valvular lesions and their immediate consequences demands the adoption of various lines of treatment. *Stimulants* are generally called for in large quantities; and *digitalis* also may be necessary if the action of the heart is weak and failing. When endocarditis seems lapsing into the chronic stage, the use of *iodide of potassium* and *liquor potassæ,* combined with bitter tonics and abundant nutriment, are to be recommended. *Alkalies* and *carbonate of ammonia* are to be given with free stimulation by alcoholics in cases of obstruction from blood coagulation in the cavities of the heart. Cases of *malignant, infective,* or *ulcerative endocarditis* (attended with fever and pyæmic symptoms in which retinal hæmorrhages are almost invariable), are to be treated in the same way as cases of septicæmia. They constitute the *diphtheritic endocarditis* of the Germans, some septic source poisoning the blood (Vol. I., p. 799, *et seq.*)

CHRONIC DISEASE OF THE VALVES AND ORIFICES OF THE HEART.

LATIN EQ., *Morbis Valvarum;* FRENCH EQ., *Maladies des Valvules;* GERMAN EQ., *Klappenkrankheiten;* ITALIAN EQ., *Malattie delle Valvolve.*

Definition.—*Lesions of the valves of the heart, or of its orifices, coming on for the most part insidiously, and which, persisting, induce obstruction or regurgitation, tending to hypertrophy and dilatation of the heart, with congestion of the pulmonary and systemic capillaries, œdema, anasarca, and dropsy.*

Pathology.—(*a.*) *Causation.*—The term "chronic valvular disease" is sometimes used to distinguish chronic lesions of the valves of the heart from "chronic endocarditis;" for it has been already shown that the valves and orifices of the heart are often damaged by this affection. Chronic valvular disease may be very insidiously established as a local expression of chronic Bright's disease, or chronic rheumatism or gout, and in such forms of constitutional derangement of the system as are associated with imperfect nutrition of the body.

(*b.*) *Morbid Anatomy.*—The lesions to which valve disease may be referred are of the following varieties:—(1.) *Vegetations;* (2.) *Fibroid thickening;* (3.) *Atheromatous* and *calcareous degeneration;* (4.) *Aneurism;* (5.) *Laceration;* (6.) *Simple dilatation of orifice;* (7.) *Malformations.*

Symptoms.—The exact lesion which causes chronic disease of the valves or orifices must generally, however, remain a matter of conjecture; and it is not of so much practical importance to determine the lesion as it is to determine whether it is of such a nature as to cause obstruction to the flow of blood, or to permit of its regurgitation. One or other of these conditions is indicated by an endocardial murmur, the persistence of which points to a condition which must lead to cardiac hypertrophy and dilatation. Nevertheless, disease at the various orifices operates differently the one from the other in many particulars, a resumé of which is here given from the writings of Drs. Fuller, Foster, Gairdner, Hilton Fagge, and Fothergill.

Aortic obstruction (one of the most common forms of valvular disease) has little effect in producing engorgement of the pulmonary capillaries, or general systemic congestion and dropsy; except when it induces dilatation of the left ventricle, and so causes the *mitral valve* to become so inefficient as to permit of regurgitation. The dilatation of the left ventricle is the turning-point in these cases. So long as the hypertrophy is sufficient to overcome the obstruction at the aortic orifice all goes on well. When the obstruction is no longer compensated for in this way, the ventricle begins to dilate. Each systole leaves a little blood in the left ventricle; the left auricle is no longer able to empty itself completely, and dilates under the accumulated blood: gradually, pulmonary and systemic obstruction begin to appear. Aortic obstruction is the least rapidly fatal form of chronic valvular disease, because its mode of compensation—hypertrophy of left ventricle—is the simplest (FOSTER). It is a question of increased power; and as the contracting action of interstitial inflammation of the valves and orifice is very gradual, the muscular hypertrophy can keep pace with the changes; and is usually, therefore, the most perfectly compensatory, and lasts longest. The character of the pulse is not materially altered, except in well-marked cases, when it becomes slower; but when the action of the heart is forcible, and the obstruction is rough, excessive eddying of the blood may be produced, causing *a thrill* at the base of the heart, and in the track of the aorta and its branches. The peculiarity seen in the *pulse-tracing* from the sphygmograph consists in an obliquity or break of the line of ascent, which marks the greater duration of the ventricular systole, and the gradual entry of the blood into the vessels. In the following tracing (Fig. 37) from Dr. B. Foster these characters are shown:—

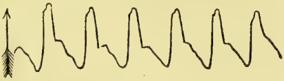

Fig. 37. *

Mitral obstruction, although less common than mitral regurgitation, is (now that its physical signs have been elucidated) recognised much more frequently than formerly. The lesion induces dilatation of the left auricle; and hypertrophy of this cavity and of the right ventricle more especially, is the means of compensation. This compensating hypertrophy, although it may defer for a long time systemic serous engorgement, cannot prevent a certain amount of pulmonary congestion. Hence, severe cough, dyspnœa (attacks of cardiac asthma), and severe palpitations are common. Congestion and œdema of the lungs, and not unfrequently pulmonary apoplexy, are the ordinary modes of death. In some cases, especially in younger patients, the lesion may be tolerated for years. In such cases the compensation effected by the right ventricle is nearly perfect, the pulse remains regular, but soft and of low tension, easily altered in form, or suppressed by any excessive pressure on the spring of

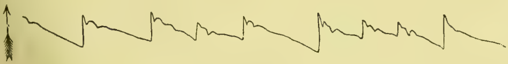

Fig. 38. †

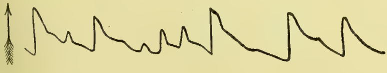

Fig. 39. ‡

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[*] Pulse-trace of aortic obstruction (Dr. B. FOSTER).

[†] Pulse-tracing of mitral obstruction (Dr. B. FOSTER).

[‡] Pulse-tracing of mitral obstruction (developed stage), in which the orifice only admitted the top of the little finger (Dr. B. FOSTER).

the sphygmograph (Fig. 38). In these cases the symptoms are, shortness of breath, troublesome cough, occasional severe palpitations, and great inability for any muscular exertion. In the later and more developed stages of the disease the pulse is markedly irregular (Fig. 39), and all the general symptoms are worse.

The pathognomonic indication of contraction of the mitral orifice is a "*presystolic*" or "*auricular systolic*" *murmur* (Fauvel, Gairdner, Hilton Fagge). Of the characters of this murmur Dr. Fagge gives the following account:—"(1.) The first and most important, although not an essential, quality of a direct mitral murmur, is its *place in the cardiac rhythm.* It is '*pre*systolic,' and its relation can probably be expressed in no way so well as by a diagram, after the manner first suggested by Dr. Gairdner (Fig. 40). (2.) The direct mitral murmur has a special *seat.* It is loudest over the apex of the heart, and is generally confined to the region of the apex. It is inaudible or scarcely audible at the base, and is very rarely carried round the axilla to the back. (3.) The *quality* of a direct mitral murmur is, in most cases, peculiar. It has a *rough, churning,* or '*grinding*' character, which enables the auscultator to suspect its nature and origin. It is frequently accompanied by a markedly palpable thrill, or '*frémissement cataire*'" (*Guy's Hospital Reports,* XVI., 3rd Series, 1871).

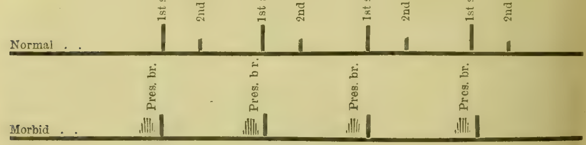

Fig. 40. *

Obstruction of the pulmonary orifice is a very rare lesion; and *tricuspid orifice obstruction* is also very rare.

Aortic regurgitation, from incompetency of the sigmoid valves, is one of the most common and most swiftly fatal of all forms of valvular diseases. The heart finds compensation in excessive hypertrophy and dilatation of the left ventricle (the true "*cor bovinum*"). By these changes an extra quantity of blood is thrown into the aorta at each systole, and the regurgitation of a portion of this extra charge can consequently take place without materially disordering the normal balance between the arterial and venous blood. The occurrence of too great dilatation of the left ventricle and degeneration of its muscle is, however, almost inevitable; for, as the coronary arteries are mainly filled in health during the diastole of the ventricle by the recoil of blood from the closed aortic valves, insufficiency of these valves has a direct influence in impeding the coronary circulation and diminishing the blood supply to the hypertrophied muscle.

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* Diagram to show position of "*presystolic*" or "*auricular systolic*" murmur (Gairdner, Fagge).

Hence, gradual degeneration of the walls and dilatation of the cavity follow. After a time, the mitral valve becomes incompetent, and the pulmonary and systemic circulation become rapidly embarrassed (FOTHERGILL, FOSTER). But Drs. Clifford Allbutt and Stone do not admit such relations of aortic regurgitation to the coronary circulation. They deny that the coronary circulation is influenced by the integrity of the aortic valves in any important respect: for the pressure upon given square areas of surface above and below the semilunar valves is equal, and whether these valves be present and efficient, or be absent, the tension is equal at any and all points. They contend that systolic failure in aortic regurgitation must be sought for in causes other than differential pressure at the mouths of the coronary arteries; such as in atheromatous or embolic blocking at the mouths of these vessels (*Brit. Med. Journ.*, June 5, 1880, p. 840). On the other hand, it is difficult to admit that the coronary arteries are filled at the moment of *cardiac systole* when the heart is contracted and hardened. They can only be injected at the moment of *aortic systole* or recoil; and the closure of the valves and their competency are necessary to maintain the blood pressure required for this. Hence, integrity of two valves is better than if all were diseased. For this reason amongst others there are many recognisable degrees of favourableness of aortic regurgitation depending on the amount of aortic second sound still appreciable (Dr. DOUGLAS POWELL, *Brit. Med. Journ.*, June 19, 1880).

Aortic regurgitation is accompanied by a peculiar and very characteristic pulse-beat. The prolonged swell imparted to the blood at each systole is not sustained by the perfect closure of the valves. The waves of blood are short and abrupt; the pulse jerks and leaps, and gives a sensation as if successive balls of blood were being shot suddenly under the finger. These peculiarities are consequences of the low arterial tension which the regurgitation of blood into the ventricle during its diastole produces. The traces of aortic patency given by the sphygmograph are marked by abnormally great amplitude. The vertical line of ascent marks the sudden ventricular contraction; and is often abruptly terminated by a sharp-pointed process (Fig. 41, Dr. FOSTER). The summit of the pulsation-trace is in many cases very short; but in others it presents a horizontal or curved line, especially if any constriction of the aortic orifice exists, or other cause producing delay of the passage of the blood into the vessels. The distinctive features of aortic regurgitation are to be found in the line of

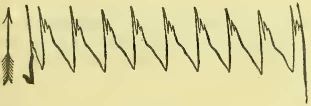

Fig. 41.*

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* Pulse-tracing of aortic regurgitation (Dr. B. FOSTER).

descent. There are often one or more extra secondary waves. The first secondary wave occurs earlier than in the normal trace, and the notch which precedes it is often very much exaggerated. But the main characteristic *is the suddenness of the fall of the line of descent, and the comparatively small size of the second secondary wave or true dicrotism* (Dr. FOSTER). Dr. Sanderson gives a different explanation (*Brit. Med. Journal*, July 20, 1867, p. 40). "Fig. 42 represents the pulse of a middle-aged man, who had acute rheumatism several times during eight years. When the tracing was taken he was suffering from extreme orthopnœa and præcordial pain. The impulse of the heart was to the left of the mammary line, and occupied a space as large as the palm of one's hand. A loud diastolic *bruit* was heard at the fourth cartilage, and a systolic bellows-sound over the aorta. Posteriorly, there was dulness at both bases, and abundant subcrepitant *râles* in inspiration. There could, therefore, be no doubt as to the nature of the case, which soon terminated fatally. After death, it was found that the aortic valve was so altered that the most copious regurgitation of blood must have taken place during each diastolic period. In this case the pulse exhibits characters which are not met with, excepting in connection with aortic incompetence." These do not consist, as is often supposed, in the unusual verticality of the expansion; for, as has been already seen, this peculiarity may be produced by merely functional disorder. The distinctive peculiarity consists in the collapse. The tracing shows that the artery becomes completely emptied during the interval between each beat and its successor; so that the diastolic expansion is no longer indicated. The explanation is, that immediately after the heart has ceased to contract, the blood injected into the aorta rushes back into the relaxed ventricle; so that, although the arterial equilibrium is for a moment disturbed, it is almost immediately re-established, the excess of pressure in the great arteries being at once relieved. In other words, the elastic force for diastolic expansion is wasted in regurgitation.

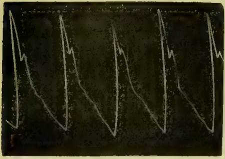

Fig. 42.*

Mitral regurgitation is the most common of chronic valvular diseases. It depends for compensation mainly on hypertrophy of the right ventricle. Hypertrophy of the left ventricle and dilatation of the left auricle also occur, leading to systolic auricular impulse at the second intercostal space, by admitting transmission of impulse from the ventricle. When excessive, it causes a vibration, a thrill, or a purring tremor, perceptible on the chest walls in the cardiac region, which is not transmitted along the aorta or great vessels. The pulse-tracing (Fig. 43) differs from the normal dotted line principally in the depth and amplitude of the diastolic notch. It closely resembles the undulating pulse of typhus (Fig. 44). In the one disease the contractile

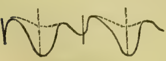

Fig. 43.†

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* Pulse of aortic regurgitation.—H. R., aged 36.

† Dicrotic feeble pulse of mitral regurgitation.

force is *weakened,* in the other it is *wasted.* The effect is the same: the systole is ineffectual (SANDERSON).

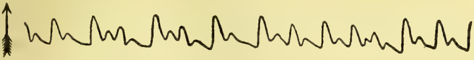

Fig. 44.*

When regurgitation is combined with obstruction, from contraction of the auriculo-ventricular orifice, the left auricle becomes hypertrophied as well as dilated; and this additional lesion always diminishes the irregularity of the pulse characteristic of pure mitral regurgitation. The tracing from the sphygmograph is as follows (Figs. 45 and 46, B. FOSTER).

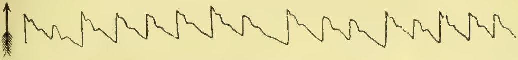

Fig. 45.†

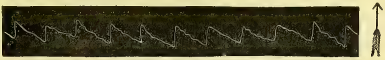

Fig. 46.‡

Fig. 47 shows a pulse seen in rheumatic valvular disease, with large mitral regurgitation. The heart acts irregularly. At times the ventricle contracts effectually; at other times the systolic expansion of the arteries is imperfect and abbreviated, in consequence of which the mean arterial tension declines. The pulse then assumes a form which is as dicrotic as that of typhus; so much so, that its double character can be readily recognised by the finger (Dr. SANDERSON). *Mitral regurgitation* interferes with the circulation through the lungs, producing cough, dyspnœa, and other symptoms of pulmonary congestion. Pulmonary apoplexy is common. The pulse is irregular in rhythm, and unequal in force and fulness.

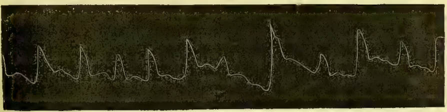

Fig. 47.§

Regurgitation through the pulmonary orifice is so rare that its effects have scarcely been verified by clinical observation.

Regurgitation through the tricuspid orifice, although rare as a primary

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[*] Typical mitral regurgitant pulse-trace (Dr. B. FOSTER).

[†] Less irregular form of mitral regurgitant pulse-trace when some obstruction is associated with the regurgitation (Dr. B. FOSTER).

[‡] Irregular pulse of pure mitral regurgitation (FOSTER).

[§] Pulse of mitral valve regurgitation (SANDERSON).

disease, is not uncommon as a consequence of dilatation of the right ventricle, which then becomes hypertrophied, the right auricle dilated, the *venæ cavæ* distended, with a strong tendency to congestion of the systemic and cerebral capillary circulation. Well-marked systolic pulsation of the liver may be regarded as one of the most certain signs of regurgitation through the tricuspid orifice, especially when combined with pulsation in the jugular veins (Dr. FREDERICK TAYLOR).

The **Symptoms** of chronic valvular disease mainly depend on the impediments offered to the pulmonic and systemic capillary circulation. In the more advanced stages all forms of valvular mischief end in a certain amount of capillary engorgement; and therefore all forms of chronic valvular disease have many symptoms in common. Oppression at the chest, breathlessness, speedy exhaustion on exertion, a general sense of lassitude, headache, restless and disturbed sleep, with frequent starting and frightful dreams, cough, palpitation, dropsy, occasional pain in the region of the heart, and sometimes severe *angina*, are amongst the earlier phenomena. The cough is due to pulmonary engorgement, and varies with the amount of *mitral* obstruction or regurgitation. Dyspnœa, while it is an early and constant symptom of mitral lesions, is frequently absent in affections of the aortic valves until the later stages. Albuminuria is then not uncommon, generally preceded by scanty urine, of a high colour, high in gravity, and loaded with urates. The scanty urine indicates the low arterial tension, causing insufficient pressure in the capillaries of the Malpighian bodies. The albumen points to the venous engorgement of the kidney. Thus, the urine of cardiac albuminuria differs from that depending on chronic renal disease.

The dyspnœa of cardiac disease is peculiar and characteristic. It is rather a breathlessness than a difficulty of breathing, of a panting, gasping character. Oppression, rather than tightness, is complained of, with a strangling, choking-throat feeling. The breathing is always accelerated, and the dyspnœa is intolerant of movement, or of any exertion whatever. These are often the only circumstances under which dyspnœa is felt. As long as the patient remains at rest there may not be the slightest appearance of dyspnœa, but the moment any exertion is made the breath is gone. It is also intolerant of the recumbent posture, hence the name "*orthopnœa*," which signifies "upright breathing," the patient being compelled to sit erect in order to breathe. In extremely severe cases the patient may not lie down for many days and nights; and should he momentarily fall off into a dose, he is instantly awakened by a sense of impending suffocation, and is in a death-struggle for breath. No suffering can compare with this. It is not wonderful that the patient longs for the sleep of death. "It were indeed a sad story, to tell how patients with disease of the heart die—the tragedies, so to say, of the medical wards of our large hospitals. . . . How some, wrung with pain, have struggled in the week-long agony of death. How some have for days together fixed themselves in the most fantastic postures, the only way in which they could find relief; some leaning forward, resting their hands on a stool, to catch a few minutes' sleep; some on their hands and knees, till the approach of death, blunting their sensations, allowed them to lie down—a sure sign of coming dissolution" (Dr. ORMEROD).

The dropsy of cardiac disease is usually a late occurrence. It makes its appearance as *œdema* or *anasarca,* very partial and slight at first—a puffiness merely of the eyelids, or more frequently slight œdema of the ankles. By slow degrees it ascends, and ultimately involves the upper extremities and the face, the scrotum in men and the labia in females becoming enormously swollen. Towards the close, effusion is apt to occur into one or both pleura, but ascites is not common. The first appearance of dropsy marks an eventful period in the progress of cardiac disease. It indicates that a new law takes effect in the circulation, and gains the mastery over the law of health, which has hitherto been able to retain the watery elements of the blood within the blood-vessels. Now, the serum escapes from the blood-vessels, and accumulates in the areolar tissue of the body (LATHAM). The forms of valvular disease, in the order of their frequent complication with dropsy, are,—(1.) Considerable contraction of the left auriculo-ventricular orifice; (2.) Dilatation of the right auriculo-ventricular orifice, with hypertrophy and dilatation of the right ventricle; (3.) A state of the mitral valve and orifice permitting free regurgitation; (4.) Considerable contraction of the aortic orifice. As a general rule, it supervenes earlier, the earlier that general venous congestion ensues. A varicose condition of the blood-vessels in the air-vesicles of the lungs is soon established, which aggravates the dyspnœa.

Prognosis is most unfavourable in mitral and tricuspid regurgitation; least so in cases of aortic obstruction; and, generally, it may be said that the form of disease which is most rapidly fatal is that which is most rapidly productive of systemic or pulmonic capillary congestion. If the heart be healthy at the date of the occurrence of valvular disease, if the extent of the lesions be not excessive, if the blood be of a normal character, if the viscera be healthy and the secretions free, if the patient's mode of living be regular, temperate, and sedentary, life may be prolonged; but if the heart be hypertrophied and dilated, if these conditions progress, if the blood be spanæmic and the lungs or liver unsound, if the secretions be irregular or defective, or if the patient leads a laborious life, the disease will run a comparatively rapid course, proving fatal probably within two or three years. In either case death is apt to occur suddenly from syncope. This is especially true of aortic regurgitation. In mitral valve lesions the approach of death is more commonly through pulmonary complications and general dropsy. The occurrence of albuminuria shows that the general venous stasis has so affected the small vessels of the kidney as to produce a local dropsy, and hence the albumen in the urine. The albuminuria marks the thorough development of the systemic venous engorgement, and the prognosis is generally the worse the longer this symptom has existed, or the more frequently it has occurred.

Treatment.—See under "Hypertrophy and Dilatation."

(*c.*) *Diseases of the Muscular Structure of the Heart.*

MYOCARDITIS.

LATIN EQ., *Myocarditis;* FRENCH EQ., *Myocardité;* GERMAN EQ., *Myocarditis;* ITALIAN EQ., *Miocarditide.*

Definition.—*An inflammation of the muscular structure of the heart (extremely rare as an idiopathic disease), which becomes softened, flabby, and finally degenerates in various ways.*

Pathology.—(*a.*) *Causation.*—It occurs not seldom as a consequence of *acute rheumatism* or of *pyœmia.* It is also generally associated with *endocarditis* or *pericarditis,* or both; and its effects are especially obvious in the strata of fibres nearest the inflamed membranes. Syphilis is also sometimes a cause.

(*b.*) *Morbid Anatomy.*—The left ventricle, especially towards its apex, or in the septum of the heart, just below the aorta, and the papillary muscles, are the parts most often affected (NIEMEYER, DITTRICH). There is profuse germination of the connective tissue of the sarcolemma, and absorption of the primitive fasciculi, leading to cicatricial-like contraction of the affected parts. The result may take the form of an abscess, or give an opportunity for the formation of an aneurismal dilatation. The lesion is recognised by the greyish, reddish-brown, or rusty colour of the muscular fibres, which are softened. Microscopically, transverse and longitudinal striations of the heart's muscles have disappeared, and the fibrillæ are broken down into a finely granular detritus, with fat globules and granules. When this condition has passed away, a cicatrix-like scar remains, which ramifies in various directions as the inflammation did, of which it represents the effect. *Indurations, fibroid degeneration, infiltration, and cirrhosis* of the walls of the heart, are occasional results of *myocarditis.* The walls may be almost tendinous; and the *carneæ columnæ* so increased in density as to split; or the walls of the right ventricle undergo a cartilaginous transformation. Chronic congestion of the heart leads to this condition, and then to defective contractility ending in dilatation (JENNER, *Med.-Chir. Trans.*, Vol. XLIII.) The more usual mode of induration is by ossification—a change which usually begins in the coronary arteries, and frequently stops there; but in some rare cases extends so that the walls of the auricles, of the ventricles, or of both, and sometimes also of the cardiac partition, become converted into bone. There are specimens in many hospital museums which make it remarkable how life could have been continued, looking to the unyielding nature and great extent of the ossification of the walls of the heart and great vessels. Dr. Joseph Coats, Pathologist to the Glasgow Royal Infirmary, described two cases, in one of which lime-salts were deposited in the muscular fibres of the heart in the form of minute round granules, resembling fatty degeneration: the patient suffered from chronic bronchitis and emphysema, with a possible syphilitic taint. In the other case the lime-salt (*carbonate*) was deposited more homogeneously, converting the heart's fibres into rigid cylinders, with a crystalline texture. The patient died of pyæmia (*Glasgow Med. Journ.*, Aug., 1872). *Albuminoid degeneration, cancer,* and *tubercle,* and parasitic cysts, are also to be found associated with myocarditis.

Symptoms.—There are no symptoms characteristic of myocarditis. The general symptoms are pyrexia of an adynamic type, with signs of blood-poisoning and collapse, especially in connection with *rheumatism, malignant scarlet fever, septicæmia, valvular disease, endocarditis, pyæmia, embolism,* or *syphilis.* If rigors set in, with swelling of the spleen, vomiting, or pain in the region of the kidneys, and albumen or blood in the urine in any of these diseases, with cardiac symptoms, myocarditis may be suspected.

Treatment, under such circumstances, cannot be indicated beyond what has been stated under "Endocarditis": but free stimulation will be called for.

ENLARGEMENTS OF THE HEART: HYPERTROPHY AND DILATATION.

LATIN EQ., *Hypertrophia;* FRENCH EQ., *Hypertrophie;* GERMAN EQ., *Hypertrophie;* ITALIAN EQ., *Ipertrofia.*

Definition.—*An abnormal growth of the muscular substance of the heart, increasing its volume by thickening of the cardiac walls; and generally with dilatation of its cavities.*

Pathology.—The subject of hypertrophy of the heart can scarcely be described without a consideration of dilatation of the heart; and although the College of Physicians have separated hypertrophy and dilatation (regarding them as distinct substantive diseases), yet the phenomena of each must be considered together, for they are generally associated in causing enlargement of the heart.

(*a.*) *Causation.*—Hypertrophy is very rarely an idiopathic disease. Spontaneous uncalled-for hypertrophy is less and less believed in. It is more commonly a secondary affection; and, in general terms, it is always a compensating growth to overcome some obstacle, except when it may be the result of plethora. As with the external muscles—*e.g.*, the blacksmith's arm, the leg of the ballet dancer and the mountaineer, so with the internal—whenever they are subjected to constantly recurrent and vigorous action with sufficient supply of nutritive material, they will increase in growth and volume. Hypertrophy of the heart occurs whenever the function of the organ is permanently or repeatedly overtasked, and when the resistance which it should normally encounter is increased. Anatomically also the nerve arrangements are such as to regulate its action, and its energy is so adjusted that activity increases as obstacles arise. The beautiful dissections of the heart by Mr. Pettigrew, in the University of Edinburgh and in the College of Surgeons of London, show the heart consisting of several folds of organic muscular fibre-tubes folded on each other, associated with a system of motor and co-ordinating innervation capable of rhythmical contraction and distension. Its motor power is under the control of minute nerve ganglia, "each with a morsel of muscular fibre under its direction" (FOTHERGILL). These ganglionic cells act directly on the muscular substance: and the action of the heart is carried on and excited in a tumultuous manner, if its connection with the vagus nerve be severed, which exercises a co-ordinating (VON BEZOLD) or an inhibitory action, by spinal fibres passing to the ganglionic cells (BIDDER), and acting indirectly through them. The application of a stimulus, such as electricity, to the vagus, retards the cardiac contractions, and may even arrest the action of the heart in diastole. When its cavities are filled with blood, such distensions lead to more or less rhythmical contractions. These contractions the vagus nerve regulates so that they only occur when the stimulus of distension is sufficient, and then contraction (truly peristaltic) takes place so swiftly that it seems a simultaneous general contraction of the whole heart. Thus, to some extent not yet determined, the *vagus nerve regulates the rhythmical contractions of the heart; and whatever may influence the vagus nerve may produce irregularity of the heart's action.* Next in importance to the nervous influence in hypertrophy is that of *nutrition.* There must be the necessary amount of nutrition and growth to meet the demand. If increased strain be thrown upon the heart to overcome obstacles to the passage of the blood through its orifices (as occurs with the simple growth and development of the body), increased growth and bulk of the muscular tissue take place, so that the balance is maintained between the blood to be driven and the power to drive it (FOTHERGILL). But if there be a deficiency in this compensatory nutrition, dilatation of the cavities sets in, from over-distension of the heart's fibres. This *system of innervation of the heart* may be compared to that of the *iris:* and is complex, so as to involve contraction and reduction of the capacity of the cavities on the one hand, and distension on the other. This implies a power of accommodation which seems similar to that possessed by the *iris* and the *ciliary structures;* so that the cardiac chambers may for a time become blood-reservoirs, from which a portion only of the blood may be expelled. The conditions which give rise to obstructions of the heart's action, and therefore to hypertrophy in its efforts to overcome the resistance, are—(1.) Abnormal enlargement of its cavities. Dilatation may be *permanently* established under any of the following circumstances (FOTHERGILL):—(1.) When mitral regurgitation leads to enlargement of the left ventricle, pouring in of the blood takes place under increased pressure. (2.) Muscular failure from defective nutrition, as in fever, atheroma of the coronary arteries of the heart, or pericardial adhesions. (3.) Obstruction to the flow of blood forwards, as by deposits on the semilunar valves, and diseased arteries, such as atheroma or *endarteritis deformans.* (4.) Functional disturbance of nerve influence. (5.) Excessive exertion (and consequent cardiac exhaustion), especially in the use of the arms in laborious occupations, gymnastic exercises, rowing boats, or climbing mountains. (6.) Valvular insufficiency.

The greater the distension, the more frequent and greater are the efforts at contraction; which tend to increase the arterial distension and tension, and so far relieves systemic symptoms.

It may be stated generally that—(1.) The larger proportion of cases of valve-lesion tend to thicken the walls and enlarge the cavities of the heart. (2.) Aortic regurgitation, with narrowing of the aperture, and still more without such narrowing, induces active dilatation of the left ventricle, followed consecutively by enlargement of the left auricle and the right ventricle and auricle. (3.) Disease of the pulmonic valves causes dilatation of the right cavities. (4.) Mitral narrowing with regurgitation leads to enlargement of the left auricle, followed in succession by dilatation of the pulmonary veins, congestion in the lungs, enlargement of the right ventricle and auricle, distension of the venæ cavæ, engorgement of the liver, congestion in the systemic capillaries, and at length (in nearly one-half of the cases) enlargement of the left ventricle itself. (5.) Combined disease of the aortic and mitral orifices causes enlargement of the left ventricle, and to a less, but nearly to the same extent, of all other cavities. Thus a great pathological fact—originally stated by Senac, and confirmed by Morgagni, is fully established, namely—that *dilatation*, with *hypertrophy*, acknowledge as their cause a force acting *a tergo*, attempting to overcome an obstacle in advance. (6.) But there are also other morbid states which, upon the same principle, tend to these results, and which are independent of valve disease—namely, *bronchitis*, *emphysema*, and any lung disease in which there is an obstacle to the flow of blood through the lungs. (7.) Disease of the arterial trunks, such as *atheroma* (*endarteritis deformans*), or morbid states of the blood, like anæmia, dilatation or narrowing of vessels, whether of the lungs or of the system, may induce dilatation of the right and left cavities of the heart followed by hypertrophy (ROKITANSKY). (8.) The retrograde influence of the systemic capillaries tends to exercise a similar influence, as shown by the effect of sudden fright, causing rupture of the left ventricle, or by the influence of *Bright's disease*, altering the quality of the blood, tending thereby to retard its progress through the systemic capillaries (SIBSON, *Med.-Chir. Rev.*, Oct., 1854, p. 434). (9.) General plethora from over-indulgence in nitrogenous food and alcoholic fluids, is also a cause of cardiac hypertrophy. Increased arterial resistance may be connected with a variety of causes, either from a contracted state of the capillaries, from diminished elasticity of the arteries, or from narrowing of the aortic valve. The following (Fig. 48) is the pulse-tracing of a patient in whom the heart, with the adherent pericardium, weighed thirty ounces. The left ventricle was both thickened and dilated, and the aortic valve so deformed and beset with vegetations, that the orifice would scarcely admit the tip of the index finger (*aortic stenosis*). The other valves were healthy. All the other morbid appearances found were referable to the cardiac lesion as their

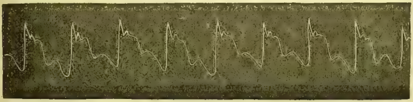

Fig. 48.*

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* G. M., aged forty. Pulse of aortic obstruction, with hypertrophy of the left ventricle (SANDERSON).

cause. The systolic expansion of the artery is of nearly equal duration with the diastolic interval (SANDERSON). The patient whose pulse is represented in Fig. 49, at the time when the observation was made, had dyspnœa on the slightest exertion, and præcordial pain. He was subject to nocturnal paroxysms of cardiac distress, which always came on with pain at the scrobiculus, followed by palpitation and nausea, with violent respiratory efforts. The cardiac dulness extended from the sternum to the mammary line, and the præcordial impulse was diffused and expansive. The systolic sound was prolonged, but no abnormal murmur could be made out. The man died several months afterwards, when it was found that the heart weighed twenty ounces; and the left ventricle was both hypertrophied and dilated, *without valvular disease.*

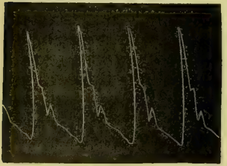

Fig. 49.*

(*b.*) *Morbid Anatomy.*—The bulk or volume of the normal heart I have found to range from 12·5 to 19·8 cubic inches. The anatomical description of the condition known as hypertrophy is in reality a compromise between two opinions. By some it is regarded as a multiplication or increase in the number of the muscular fibres or primitive fasciculi—an *Hyperplasia* (see Vol. I., p. 118). By others the condition is considered due to a thickening of existing fibres—an increase in bulk of the primitive muscular bundles—a true *hypertrophy.* It may be general or partial—that is, may affect the whole heart, or one side of the heart, or one ventricle, or one auricle, or the ventricle of one side and the auricle of the other, or both ventricles or both auricles, or, indeed, every possible combination of the four cavities. The auricles, however, are much less frequently affected than the ventricles. Two forms are recognised by the College of Physicians, namely:—(a.) *Of the left side;* (b.) *Of the right side.*

A hypertrophied heart may weigh from sixteen to thirty-two, or even over forty ounces. In hypertrophy of the right ventricle the walls are more uniformly thickened than in hypertrophy of the left: and the increased thickness is always more marked about the tricuspid valves, and at the origin of the pulmonary artery. The greatest thickness observed has been seldom more than four or five lines, which (taking the natural thickness at two and three-quarter lines) is scarcely a twofold increase. In malformations of the heart (*foramen ovale* open) it has been found much greater—from twelve to sixteen lines (BERTIN and LOUIS). Besides an increase of thickness, the walls of the right ventricle, when hypertrophied, acquire a greater firmness, so that on cutting through them they do not collapse. Hypertrophy is sometimes most marked in the muscular walls of the left ventricle, and sometimes in the trabeculæ and papillary muscles of the right. These forms of hypertrophy are distinguished mainly by the *capacity of the chambers* of the heart which accompanies the hypertrophy, namely:—(*a.*) Where the capacity of the cavities is normal—*i.e.*, retain their relative capacities to each other, although the walls are increased in thickness—*simple hypertrophy.* (*b.*) Where the cavities enlarge or dilate, and the muscular walls thicken—*i.e.*, *eccentric hypertrophy*, or *hypertrophy with dilatation.* The normal heart weighing from eight to eleven ounces, according to age, the weight in this form of hypertrophy is often double or triple that amount; and Bouillaud speaks of eighteen, twenty, and twenty-two ounces being not uncommon. Peacock records in man even more than forty ounces—the ox's heart—the true *cor bovinum.* (*c.*) Where the capacity of the cavities is diminished and the walls thickened—*i.e.*, *concentric hypertrophy*, a condition generally due to the *post-mortem* contraction of an hypertrophied heart (CRUVEILHIER, BUDD). From this cause the ventricle has been found so reduced in size as to be not larger than an unshelled almond. The capacity of the cavities may be enlarged during life; but the hypertrophy may be so great, and the contraction at or after death so powerful and energetic, that all dilatation has disappeared. This is especially apt to occur in the hypertrophy of the heart which complicates *Bright's disease.*

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* W. S., aged fifty-nine. Hypertrophy of the left ventricle, without valvular disease.

Symptoms.—In detecting enlargement of the heart, and thickening of its walls, and dilatation of its centres, its size and the force of its action, ascertained by *palpation*, *percussion*, and *auscultation*, furnish the principal data. (See page 552, *et. seq.*) It is by the extent and power of the impulse that the heart's muscular condition is ascertained. As a rule, persistence of valve disease implies an enlarged heart, with an impulse increased in extent and in power. There are also cases to be guarded from mistake—namely, those where a murmur exists with a preternaturally strong, troublesome, quick, and smart impulse, *but limited within a diminished cardiac region.* Such a murmur is of anæmic origin, and the heart is usually lessened rather than enlarged. The symptoms of *hypertrophy* are *local* and *general.* The *local* symptoms are,—a more powerful impulsion, a wider range of action, and some change in the sounds; a greater extent of dulness in the cardiac region, and sometimes a bulging out of the left side. The increased impulsion is in proportion to the greater thickening of the walls. In slight cases it is only sensible to the hand, in others the heart "knocks against the ribs," and even raises the hand of the auscultator resting on the chest. This greater impulse not only causes a vibration of the *præcordial* region, but even shakes the whole of the chest. The abnormal action in these cases is often sensible to sight, each contraction agitating the patient's dress, and sometimes even moving the bed-clothes. The point of the heart deviates more to the left, and its motions may be traced from the second or third rib as low as the sixth or seventh intercostal space. The increased thickness of the walls of the heart is unfavourable to the transmission of sound; and in simple hypertrophy without enlargement of the cavity, the natural sounds will be duller than in the normal state. When the cavities are enlarged the sounds are clear, full, and louder than natural. In *hypertrophy of the left ventricle* the impulse is stronger immediately under the inferior portion of the sternum than between the fifth and sixth ribs. In *hypertrophy of the right ventricle* there is swelling of the jugular veins, which pulsate synchronously with the carotids (LAVOISI, LAENNEC). In general, this pulsation is limited to the inferior parts of the jugular veins; but it has been seen to extend to the superficial veins of the arm. Hypertrophied heart is very common in Bright's disease; and many persons affected with hypertrophy suffer severely from *angina pectoris*, with palpitation.

Dilatation of the heart implies that the capacity of its cavities is increased disproportionately to the thickness of their walls. It occurs in three forms—namely, (1.) *When dilatation predominates over hypertrophy;* (2.) *Simple dilatation, where the thickness of the walls is normal;* (3.) *Dilatation where the walls of the heart are attenuated, or thinner than normal.* Practically, therefore, the physician has to deal with the diagnosis and treatment of—(1.) *Simple hypertrophy;* (2.) *Hypertrophy with dilatation of one or more of the cavities;* and, (3.) *Simple dilatation with or without attenuation of the walls of the cavities.* Hypertrophy of a sufficiently compensatory nature entails no distress. Accordingly, those symptoms are to be recognised which mainly point to insufficient compensation between hypertrophy and dilatation, and to disturbance of the balance of efficient circulation.

Inefficient power of the heart's action is first made obvious by the following conditions:—(1.) *Palpitation*, which indicates increased action or effort on the part of the heart, with inefficient results. It is not accompanied by a bounding pulse or increased apex-beat, as after exertion or excitement. It is generally associated with irregularity of pulse, with chronic valvular insufficiency, and with *Bright's disease.* (2.) The *irregularity of rhythm* in hypertrophy is a sign of debility. The commencement of the *peristaltic* contraction or heart-stroke is arrested. It suggests a resemblance to the change of rhythm in the sound of a horse's feet when cantering (FOTHERGILL). It is evidence of over-taxation of the heart's power, often the result of over-exertion, and indicative of ventricular distension. (3.) *Persistent intermittence* in hypertrophy is usually associated with an impaired first sound, defective apex-beat, and other signs of degeneration. The most perfect action of the heart is when we are unconscious of its labours; but when a combination of the phenomena just noticed renders the patient apprehensive of evil, or causes distress, then we must seek to discriminate whether the distress is arising from the predominance of *dilatation* or of *hypertrophy*—learn in which direction compensation is deficient, and aid the efforts of nature to repair the defects.

Evidences of distress on the part of the heart are to be recognised by the following signs:—(1.) *Cardiac engorgement*, indicated by a feeling of anxiety about the præcordia, with a general unaccountable anxiousness; a sense of difficulty of breathing, which is notably increased upon exertion (*cardiac dyspnœa*); fluttering about the left breast, amounting to palpitation upon effort or exercise; pulse irregular, or if regular, compressible; a dusky complexion and impeded respiration. (2.) *Palpation* conveys a feeling of diffused impulse, from a large mass apparently being thrown into contact with the thoracic walls. (3.) *Percussion* shows increased general dulness, frequently in the direction of the right side of the sternum. (4.) *Auscultation* discloses a short slapping sound, with or without irregularity, or, perhaps, evidence of laboriousness—a heavy swell with obvious effort—not followed by a corresponding effect—in filling the arterial vessels. (5.) *General phenomena* of distress embrace a tickling cough, shortness of breath on exertion, slight attacks of bronchitis, pulse small and feeble, cardiac excitement, with irregular rhythm—an aggregation of sounds and phenomena demonstrating embarrassment and laboriousness in the right ventricle, which may lead to permanent dilatation of the right ventricular cavity, with more or less hypertrophy, frequently found in persons advanced in life, and not uncommon in adult life. Expulsive power is deficient until hypertrophy can compensate and restore the balance. The heart is distended, and repeated contractions fail to expel *all* the blood, the ventricles remaining more or less full on systole. It is in this partially filled condition of the ventricles that the difficulty lies. If the ventricles were not partially full, the auricle and veins would be somewhat relieved; but there is what would fill well an empty ventricle waiting to be discharged into one more or less full to begin with. The action may be moderately regular on quiet being maintained; but it is at once disturbed on motion, especially if this be at all active; and then we get palpitation and irregularity, or even intermittency, the regular action being again restored by quiet. There is a constant contest going on between the stimulus of the contained blood and the inhibitory action of the pneumogastric nerve fibres, and the distension excites the muscular walls to overcome the restraining influence. An incessant play goes on between the condition of over-distension and the restraining fibres of the vagus; the balance between the muscular walls and their work remaining confessedly disturbed" (FOTHERGILL).

Such are the phenomena when dilatation predominates over hypertrophy. When the reverse obtains—that is, *eccentric hypertrophy* of the left ventricle—a certain grouping of the following subjective and objective phenomena declares its presence:—There is visible pulsation of the carotids, a loud systolic sound in the larger arteries, and a full pulse, visible even in the smaller arteries; an abnormally strong heart-stroke, extending over the length of the heart; a depression of the apex, extension of cardiac dulness, intensification of the heart's sounds in the left ventricle and aorta, and sometimes a metallic click. When eccentric hypertrophy of the right side prevails, there is augmented heart-stroke, often extending along the sternum and left lobe of the liver, dislocation of the apex of the heart, which extends *outwards* rather than downwards, extension in width of the cardiac dulness, intensification of the cardiac sounds in the right ventricle and pulmonary artery (NIEMEYER). These combined phenomena indicate general eccentric hypertrophy of both sides.

The table on the following pages (compiled from Dr. Walshe's treatise) exhibits the main points in the symptoms of these forms of disease for the purposes of comparative diagnosis.

Prognosis.—As a complication of other diseases of the heart, hypertrophy, when compensatory, is a favourable rather than an unfavourable condition, tending to mitigate the danger of the chief disease. When death occurs, it is generally due to hæmorrhagic effusions into the brain or lungs, for the prevention of which, treatment such as to favour the compensatory powers of the circulation must be had recourse to with great care and judgment, combined with careful management of the diet. When the phenomena of hypertrophy have persisted for a time, they are sometimes replaced by another form of lesion—also preservative, as has been shown by Sir William Jenner. A fatty degeneration of the heart commences, from impaired nutrition due to atheroma of the arteries.

Its activity is thus impaired and lessened, and there is less risk of its violent action rupturing the diseased and friable arteries of the brain or lungs. The failure of the right side of the heart in asthenic disease or affections of the respiratory organs is often the channel through which death may suddenly approach. The right ventricle, taxed to the utmost,

TABLE CONTRASTING THE MAIN SYMPTOMS OF THE FORMS OF HYPERTROPHY AND DILATATION.

A.—General Physical Signs.

Simple Hypertrophy.	Symptoms superadded to constitute Hypertrophy with Dilatation.	General Simple Dilatation.
Arching of the præcordial region, with widening and bulging of the left intercostal spaces from the third to the seventh.		No prominence of cardiac region.
Impulse increased in extent to the left of the sternum.	Extent of visible impulse much greater; pulse may be felt in the back, and its character is less heaving, sharper, and more knocking, than in simple hypertrophy.	Apex-beat indistinctly visible or actually invisible, the rounded-off form of the heart destroying the apex form.
Maximum impression felt below and about the left nipple.	Point of the apex-beat carried downwards and outwards beyond the line of the nipple, so as to reach the seventh interspace.	Impulse conveys an undulatory sensation to the hand, and feeble in proportion to the purity of the dilatation.
Heart's impulse slow and heaving, as if pressing steadily against an obstacle—in rhythm regular, in force unequal.	Force increased, so as to shake the head or trunk of the patient, or the bed on which he lies.	Force of successive beats is unequal. Rhythm irregular, to a slight or to the very highest degree. The hand applied to the region where the impulse is felt does not feel the beat always strike at the same place.
Superficial and deep-seated dulness augmented in area, but its shape retaining the triangular form.	The dulness tends to assume a square form in place of a triangular one, and may reach from the second interspace to the eighth rib, and from an inch and a half to the right of the sternum to three inches, or even more, outside the vertical line of the nipple. It may be detected in the back.	Intensity of superficial percussion, dulness not increased; and in cases of attenuated walls of the heart's cavities, the resistance is less marked than in health. General areas of dulness widened.
First sound is dull, muffled, prolonged, and weakened, almost to extinction, directly over the ventricle.	Sounds gain greatly in loudness and extent of transmission, especially if the valves are not thickened.	*First sound* short, abrupt, and unnaturally clear at the apex and base, appears near the surface, and its maximum point of expression is slightly lowered.
Second sound full and clanging, *post-systolic silence* shortened.		*Second sound* not specially affected. Upon the quality of the heart's texture, as regards softness or flabbiness, will depend the extent to which the sounds are transmitted. Intracardiac murmu always regurgitant.
Mitral regurgitant murmur, as a clinical fact, exists at one time and disappears at another.	In consequence of the altered direction of the orifice of the aorta to the cavity of the ventricle, a systolic basic murmur may be generated.	

B.—GENERAL FUNCTIONAL SYMPTOMS.

Simple Hypertrophy.	Symptoms superadded to constitute Hypertrophy with Dilatation.	General Simple Dilatation.
Strength unimpaired. Power of walking or of ascending a hill diminished, on account of dyspnœa induced by the effort.	Strength tends to become impaired.	Strength fails, and the patient is habitually irritable and melancholy.
Face florid.	Purpleness and lividity great in proportion to the valvular or pulmonary obstruction.	Lividity and mottling of the face prevails, and of the lower extremities generally, with chilliness of surface. Softly pitting anasarca spreads from the feet to the abdomen, external genitals, thorax, face, and neck. Ascites follows anasarca.
Constipation habitual.		Bowels habitually constipated or alternately relaxed—discharges dark.
Dyspnœa occasional.	Paroxysmal attacks of dyspnœa.	Dyspnœa, sometimes called cardiac asthma, becomes complete and habitual, with asthmatic paroxysms, in which the cough is dry, harassing, and convulsive. Expectoration serous, sometimes streaked with blood.
Radial pulse full, strong, firm, tense, resisting, and prolonged, without jerk or thrill.	Fulness of pulse continues, but strength and power of resistance lost.	Pulse small and feeble, and abnormally late in time after the ventricular systole. It may be regular, or narrow, feeble, fluttering, and irregular.
Pain rare.	Pain not uncommon.	Palpitation and cardiac uneasiness most distressing.
Rarely, and never rapidly, the direct cause of death.	Indirectly, and more or less rapidly, leads to a fatal issue.	Faintness occurs from time to time, and may lapse into syncope and sudden death.

becomes gradually paralysed by the carbonic acid of its contained blood *anæsthetising* its action, and if its action can be maintained the patient may tide on to recovery (FOTHERGILL). In every individual case, the following points in prognosis present themselves for solution, or may be presented to us by the patient or his friends:—(1.) Is there danger of sudden death ? (2.) What is likely to be the progress of the case and the dangers attending it ? (3.) How long will the disease last ? (4.) What are the prospects of cure or relief. Sudden death may be expected in *aortic regurgitation*, and may occur in mitral regurgitation. *Obstructive disease* on the left heart will affect the heart, lungs, and circulation generally; *aortic* and *mitral* obstruction often last a long time. *Mitral disease* is dangerous from its effects on the lungs. *Tricuspid regurgitation* causes most distress as time goes on, and its progress is often slow and tedious.

Treatment.—As the bad symptoms in valvular affections arise from defective compensation, and as the compensation depends on the integrity of the heart-muscle, the maintenance of its nutrition is of prime importance. Palliative treatment is necessarily directed to aid the mechanism by which the ill effects of valvular incompetency are to be met. *In affections of the aortic valves,* when the heart-muscle is well nourished, an almost perfect compensation may be maintained for years upon a nutritious (albuminous) diet, with fluids in small quantity—tonics, especially preparations of iron, being indicated. In the earlier stages a moderate amount of exercise is beneficial. In *mitral* affections, the compensation can never be so complete as to do away with the tendency to pulmonary complications. While the same nutritious diet and tonics are indicated to promote the nutrition of the cardiac muscle, exercise must be taken more cautiously; the object being to regulate and moderate the action of the heart, to control the tendency to local congestion, and to mitigate or remove the symptoms which result from the cardiac derangement.

Any blood-letting required ought to be effected by the application of a few leeches to the *præcordial* region, and very general relief will be obtained by hydragogue purgatives, aided by dry-cupping, mustard poultices, and turpentine fomentations. When, however, active congestion of the lungs exists, venesection, cupping, blisters, and sinapisms may be necessary. The distress from simple hypertrophy may, in the majority of cases, be greatly mitigated by such means as tend to tranquillise the action of the heart. No known drug possesses the power of controlling the growth of the heart. Saline and aloetic purgatives aid the calmative influence of the local abstraction of blood. Diuretics are useful independently of the existence of dropsy. Sedatives of the heart's action are indicated throughout, such as *hydrocyanic acid*, *acetate of lead*; *digitalis*, *belladonna*, and *aconite*, in the form of the alcoholic extract, given in doses of *one-eighth of a grain* (WALSHE). It is chiefly useful in excessive hypertrophy, with *hydrocyanic acid* and *caffeine*. In repeating the doses, the effects must be watched, while they relieve the painful sensations and disquietude about the heart. If anæmia prevails, animal food should be permitted; and the more soluble and less astringent preparations of iron should also be given. Fluids must be taken in small quantities. Like *hypertrophy*, *dilatation* of the heart is not removable by treatment. To improve the tone and strengthen the action of the heart, without exciting its irritability, are the objects to be aimed at in the management of cardiac hypertrophy with dilatation. For this purpose the beneficial influence of *digitalis* has been ably shown by Dr. J. M. Fothergill, in his "Hastings Prize Essay" for 1870, published in the *British Medical Journal*, commencing July 1, 1871; and also by Dr. Balthazar Foster in the *Medico-Chirurgical Review* for July, 1871. It is an efficient agent in helping to co-ordinate, by restoring the regularity of the heart's movements, when they have become tumultuous and irregular. The mechanism of compensation in each form of valve disease, therefore, requires to be considered. In hypertrophy *digitalis* is only of use when hypertrophy exceeds the limits of compensation. It slows the pulse and regulates the heart-muscle; but if the heart-muscle is unsound it will not be of service. As a general rule, the drug is most useful in mitral affections, and is contraindicated in aortic valve disease, except in cases in which the hypertrophy has exceeded the limits of compensation, and become the chief cause of the symptoms. It slows the pulse and increases its tension, while strengthening and regulating the heart's action. The slowing of the pulse increases the period of ventricular diastole—that is to say, the period during which regurgitation takes place when the aortic valves are incompetent; consequently, digitalis does harm in this lesion by augmenting the regurgitation. In *mitral obstruction* it is most valuable when the rapid, irregular pulse tells of the varying quantities on which the ventricle contracts. Sometimes the interval between the ventricular contraction is so short, and the ventricular charge is so small, that the systolic wave does not reach the wrist. Under these circumstances the auricle must have more time to fill the ventricle. This is what digitalis effects; for by slowing the action of the heart, the period of time during which the blood from the distended auricle can flow into the ventricle is increased; and as the extra time allows more blood to pass through the narrowed mitral orifice before the final effort of the auricle is made, that effort is made on a smaller quantity of blood, and is consequently more effective; for the smaller the quantity of blood which the auricular muscle has to push before it, the greater will be the velocity given to the current. The ventricle, though contracting less frequently, contracts more effectually. Instead of eighty or ninety irregular contractions per minute, we get some sixty steady equal beats. The pulse grows in force, fulness, and regularity; the arterial tension rises; the pulmonary congestion diminishes; the kidneys, before inactive, wake up to their work; and the advancing dropsy subsides (Foster, *Med.-Chir. Rev.*, July, 1871). Digitalis is also most useful in *mitral regurgitation.* In place of a large number of ineffective contractions, it concentrates the power of the ventricle on a smaller number of well-directed steady beats, each throwing a larger charge of blood into the arteries, and so diminishing, beat by beat, the over-distension of the right heart. It may be prescribed as an *infusion* or *tincture*, and continued as long as the quantity of urine increases or keeps up to the maximum which the digitalis produced. Such diuretic effect is the outward or visible sign of its beneficial action. The high-coloured scanty urine, loaded with urates, is then replaced by a clear and copious stream, which tells of a steadily beating heart and a firmer pulse (Foster). The *tincture* is the most convenient form for continuous administration (♏v. to ♏x., three or four times a day). The freshly-made *infusion* acts most rapidly (ʒss. to ʒi., three or four times a day) along with *potash* or diuretics, and is conveniently added to vegetable infusions. Where it is desired to keep up the action of digitalis for a long time, *the powder of the leaves* is the most desirable form. It can then be given in pill, with the dried *sulphate of iron*, carminatives, laxatives, or both twice a day. Half a grain to a grain of *powdered digitalis*, with an equal quantity of *sulphate of iron*, and a small portion (¼ of a grain) of *cayenne pepper*, in *extract of gentian* or *aloes* and *myrrh* pill, is a useful form which may be continued for months. This pill should be taken shortly *after* food. The addition of *iron* to *digitalis* is of great importance. The absorption of *digitalis* through the skin, by using poultices of the leaves, or flannel cloths soaked in the infusion and laid over the skin of the abdomen and thighs, is often most beneficial where it cannot be given by the mouth (Christison, Trousseau, Fothergill).

There are some circumstances, however, under which it is necessary to withhold the administration of digitalis in cardiac disease—namely (1.) The presence of *atheorma* (*endocarditis*) to any appreciable extent, and in aortic valve disease. (2.) Reith, Gull, and Brunton object to its use in fatty heart. (3.) Intermittency of pulse is also a contraindication. If that symptom comes on during its use, the further administration of digitalis must be suspended, more especially if the pulse becomes thready, and the quantity of the urine diminished. On the other hand, where intermittency is shown to be the result of the heart's inability and distress, the quantity of digitalis must rather be increased (FOTHERGILL). (4.) The occurrence of persistent vomiting or noises in the head under its use, suggests that it should be left off, for a time at least.

The action of the heart is also markedly influenced by *veratrum viride.* The dose of *the tincture* (from *five* to *twenty mimims*) should be gradually increased from the smaller to the greater dose, till some obvious effects are produced. If the pulse is reduced, or nausea occurs, no increase of the dose should be made. If vomiting occur, it should be suspended; and when resumed, the dose should be diminished. *Veratria* may be given in doses of from *one-sixteenth* of a grain to *half a grain.* When the pulse is sufficiently reduced, the doses should be diminished one-half. *Morphine* or *laudanum,* with brandy, is an antidote for an overdose of *veratria,* which is an exceedingly powerful remedy as a cardiac sedative, and requires to be used with great caution, the patient being constantly watched. Its depressing effects on the circulation and nervous system are often very striking—a pulse of 75 or 80 being reduced in the course of a few hours to 35 or 40 (*Record of Pharmacy and Therapeutics,* No. V., p. 35, J. C. BRAITHWAITE). Sleeplessness being one of the most distressing symptoms, opium in some form might be considered advantageous; but "to give sedatives in such cases would be the refinement of cruelty. What keeps the patient awake is not a want of tendency to sleep, but a condition that makes sleep impossible. Relieve him of his orthopnœa, and he would be asleep in ten seconds, and so dead asleep that it would take a great deal to rouse him, like a half-asphyxiated child on whom tracheotomy had just been performed. His great struggle is the struggle between sleep and life; with opium thrown into the scale of sleep, the struggle for life would only be so much the harder. Only in one way would opium give him ease; the narcotic of opium, added to the narcotic of the carbonic acid already circulating in his veins, might accelerate by some hours, or even days, the final coma, and make him sooner sleep th sleep of death. But the euthanasia that is purchased by anticipating th natural process of death comes very near to homicide, and is an alternativ that few would adopt" (HYDE SALTER, *Brit. Med. Journal,* Feb. 8, 1862). *Morphia given hypodermically* is the best form for relieving the dyspnœ and sleeplessness of some valvular affections, especially in mitral valv disease. The dose to commence with is one-eighth to one-sixth grain the *bimeconate of morphia* (Dr. CLIFFORD ALLBUTT). But all sedativ require the utmost caution in their use.

The groundwork of medicinal remedies consists in the administration general tonics in the form of *bitters, mineral acids,* and preparations of *iro* *Belladonna* may be employed to tranquillise undue excitement with great safety than any other sedative remedy. A plaster of *belladonna* and *coniu* over the heart is of signal service when pain is usually felt near the ap associated with palpitation. Due action of the bowels must be dai obtained, to accomplish which the aloetic medicines are the best, aided by the gentle action of an occasional mercurial aperient.

Careful regulation of the patient's mode of life is above all things necessary in diseases of the heart. Excitement of all kinds must be avoided; and the diet should be light, nutritious, and of moderate quantity; the clothing warm, and cold should be carefully avoided, especially by those affected with mitral disease.

When *anasarca* supervenes, the hydragogue cathartics are required to insure copious watery discharges from the bowels. *Electuary of bitartrate of potash*, or of *gamboge*, *elaterium*, *podophyllin*, and *compound jalap powder* should be given on alternate days. Stimulants also may be required, the most suitable being Hollands, gin, or whisky.

When *dropsy* appears, diuretics yield most relief. *Acetate, nitrate, iodide,* and *bitartrate of potass, nitric æther, compound tincture of iodide,* the *infusion* and *spirits of juniper,* or *gin*, may all be employed in successive changes, variously combined. Small doses of *blue pill* and *compound squill pill* at bedtime, will facilitate their action generally; and so will cupping over the region of the kidneys, if symptoms of congestion of these organs prevail. Hydragogue cathartics aid the diuretics in subduing the dropsical effusions, such as *elaterium, gamboge, bitartrate of potass,* and the *compound jalap powder* —the two last made into an electuary.

CARDIAC ATROPHY.

LATIN EQ., *Atrophia;* FRENCH EQ., *Atrophie;* GERMAN EQ., *Atrophie;* ITALIAN EQ., *Atrofia.*

Definition.—*Abnormal wasting and loss of the muscular substance of the heart.*

Pathology.—(*a.*) *Causation.*—This condition is not to be confounded with contraction of the ventricle diminishing its cavity. Except as a congenital malformation, its idiopathic occurrence is not believed in. *As an acquired morbid condition* it is the result of a variety of diseases, especially the following:—(1.) In common with the wasting of other parts, as during *tubercular consumption*, *cancer*, and *old age;* also in prolonged *typhus* and *typhoid fever*. (2.) The result of pressure, as from chronic pericardial effusion, thickening of the visceral pericardium, and pericardial adhesions. (3.) Imperfect circulation through the coronary arteries, as from disease in them; or in hypertrophy with aortic valve disease, leading to degeneration and atrophy of the muscular texture of the heart.

(*b.*) *Morbid Anatomy.*—The walls of the heart may be so atrophied that it has been found to weigh only four ounces two scruples, instead of nine and a half ounces, while its parietes were reduced to little more than a thin membrane. This atrophy may be general or partial. In some cases it takes place without any notable alteration of the capacity of the chambers of the heart—*simple atrophy*. More commonly, when the walls are thinned, the chambers are diminished. The whole heart may be atrophied, as in phthisis. Fluid is usually found in the pericardial sac.

Symptoms generally are—slowness of the pulse, occasional palpitation, frequent attacks of fainting, difficulty of breathing, and tendency to dropsy. There are also signs of defective nutrition of the body, and great muscular debility (HOPE).

FATTY AND OTHER DEGENERATIONS OF THE HEART.

LATIN EQ., *Degeneratio Adiposa;* FRENCH EQ., *Dégénérescence Graisseuse;* GERMAN EQ., *Fettije Entartung;* ITALIAN EQ., *Degenerazione Grassosa.*

Definition.—*A change in the muscular substance of the heart, which results in the elements of the muscular fibres being replaced by molecular fatty particles. The change tends to sudden death by rupture of the heart, or by syncope.*

Pathology.—(*a.*) *Causation: Age.*—The disease is one of advanced life, and occurs in all ranks and social conditions. From an analysis of cases I find the average age of females is *forty-six* years, and of males, *fifty-two.* Of twenty-nine cases the youngest patient was a male infant *six months* old, and a female of *ten years;* while the oldest male and the oldest female were each *seventy-six* years. Between the ages of *fifty* and *eighty,* in both sexes, the greatest number of cases have occurred. *The general health and condition of the patients previous to their fatal illness* have been variously described:—(1.) As delicate, weak, nervous, or reduced by previous illness, such as miscarriage, menorrhagia, hæmorrhoids. The patients thus described were of short stature, thin, and spare make. (2.) As strong, stout, fat, muscular, or hearty. Of these some were of sedentary habits, unaccustomed to active employment, but temperate. (3.) As having suffered from slight attacks of bronchitis, "liver complaints," "spasmodic pains of the stomach." (4.) As anæmic and pale, although at the same time stout and well-grown. (5.) As having suffered more or less from previous apoplectic-like seizures and headaches, although they had recovered from them to some extent. (6.) As having been exposed to malaria in India, where they had suffered from frequent attacks of intermittent fever. (7.) As having suffered from mental anxiety and domestic distress, inducing great irritability and nervousness. (8.) As having, to all appearance, enjoyed excellent health. (9.) It may follow myocarditis, and has been found in cases of poisoning from *phosphorus.* Living as we do in a tumult of incessant excitement, hurry, and competition, "the struggle for existence" is maintained till the heart fails almost unperceived. The physician can but indicate the fact; and if fatty degeneration of the heart is not more frequent, it is certainly obtruding itself more on public notice than hitherto, from the many cases of sudden death of which it is said to be the cause.

(*b.*) *Morbid Anatomy.*—Two varieties of fatty heart have been recognised:—(*a.*) In one form the fat, composed of oil in nucleated cells—the ordinary fat-cells—grows on the surface of the organ, and also between its muscular fasciculi and the reflected pericardium. It is especially abundant at the junction of the auricles and ventricles, in the groove or sulcus between the chambers, along the trunk of the coronary veins, at the edges of the ventricles, at the apex, and at the origin of the aorta and the pulmonary artery. The right ventricle is thus often almost entirely covered.

This is rather a form of fat accumulation, than of degeneration,—an abnormal increase of the amount of fat usually found in and upon the heart. But it so gradually encroaches on, and insinuates itself between, the muscular fibres, that it conceals, impoverishes, and ultimately causes them to waste, from its pressure; so that the muscular walls become thin, especially towards the apex and over the right ventricle. In these parts the fibrous structure wastes, and the *columnæ carneæ* appear to spring altogether from the *endo-pericardium.* In this form, some of the muscular fibres may remain healthy, while others undergo the fatty degeneration within the sarcolemma. It usually accompanies general corpulence, especially the obesity of advancing age, where much alcoholic aliment is taken. (*b.*) In the other form a degeneration of the ultimate muscle-fibre ensues. Its sarcal matter disappears, and its place is taken by fat in a molecular form. Minute oil-globules ultimately come to fill the sheathes, which previously contained muscular elements. It is a fatty metamorphosis (capable of microscopic demonstration) in the minute fibrillæ of the heart's substance.

As to the nature of other changes observed in the minute tissue of the heart connected with fatty degeneration, a variety of description has been given :—(1.) *There is sometimes only a very moderate amount of degeneration of tissue, or alteration in the bulk of the heart;* but then the functional disturbance is serious, and the case rapidly fatal in its issue (WILLIAMS, l. c., Vol. II., p. 186), probably implicating cardiac ganglia and nerves. (2.) *Amongst apparently healthy tissue, a portion only of the muscular substance has undergone degeneration,* shown by the disappearance of the cross-marking of the muscular fibrillæ, the fibres being filled with oleo-albuminous or fatty granules. The coronary artery in such cases is generally found ossified or obstructed, going to the seat of degeneration (QUAIN, *Path. Soc.*, Vol. II., p. 188; and Vol. III., pp. 262, 270, 273). In these cases the substance of the heart is discoloured where the degeneration is sufficiently marked—of a pale yellowish colour or brownish-red, or pale brown, and the texture tears readily. These changes may be seen in large masses, or pervading long tracts of tissue, or particular parts only, such as the papillary muscles or *columnæ carneæ.* This is the form most often associated with other evidences of the general marasmus of old age, which may be a ripe old age or a premature one. It is the form especially associated with the *arcus senilis* and fatty degeneration of arteries. (3.) *Cases in which the death could not be ascribed to fatty degeneration of the heart alone, but where other lesions existed*—such as fatty degeneration of the cerebral arteries, resulting in death by apoplexy; or in cases in which apoplectic seizures occur. (4.) *There are cases of degeneration which result in the "cardial apoplexy" described by Cruveilhier, where hæmorrhagic spots, or extravasations of various sizes, occur in the substance of the muscular tissue; the surrounding tissue is in the state of fatty degeneration, and the coronary arteries leading to the degenerate tissue and hæmorrhagic spot are ossified or obstructed* (QUAIN, l. c., Vol. I., p. 192; Vol. II., p. 190). To the naked eye the muscular tissue of such hearts presents a pale, mottled, or dirty yellow appearance. It has none of the flesh-red hue of health; but it is soft and flabby to the touch; and its texture at the degenerate parts so friable or brittle, that it yields to the slightest pressure, and may be torn without difficulty. The elasticity of the muscular substance is so completely lost that, when cut across, the walls at once collapse.

Symptoms occur sometimes suddenly after exposure to cold, with pain in the chest, shortness of breath, and palpitation. In some cases where *angina pectoris* prevailed for many years the pain was sometimes excessive, shooting down the left side and arm, especially after any excitement. In other cases *cardiac symptoms* consist of a dull pain in the region of the heart or ensiform cartilage just before death, associated with dyspnœa. The cardiac affection also betrays itself by cough, dyspnœa, and general debility, a sense of oppression at the chest, and desire to draw a deep breath, the breathing being sometimes embarrassed and aggravated by exertion. Involuntary sighing is sometimes a prominent symptom, with disturbance of the rhythm of breathing (Cheyne-Stokes respiration, p. 550, *ante*). The attacks of giddiness and apparent coma are generally due to syncope, from feebleness of the heart's action. The patient is pale, has a feeble pulse, and presents more or less lividity of the lips—symptoms which may serve as indications of the true nature of the disorder (FULLER). *The condition of the pulse* (having been observed after the cessation of acute attacks of disease, which afterwards terminated fatally) was noted as "irregular and unequal," or as "feeble, rapid, and irregular;" as "feeble and intermitting, every eight or ten beats," as "large, jerking, and compressible" (one hundred and six per minute—afterwards a hundred, but small, jerking, and regular to the last); or as "small and feeble only." In one case in which the pulse was observed for two years and a half previous to death, it was found to be irregular and uncertain in its action —a condition which disappeared during an attack of bronchitis, but which reappeared as the patient recovered from the bronchitic attack —varying from *seventy* to *eighty* in a minute. In other cases, where its condition had been long noted, it was weak, irregular, and intermitting, numbering at first *eighty*, becoming weaker, often intermitting, and more slow (*sixty* to *seventy*), or small and feeble, shortly before death. In extreme cases the pulse may fall as low as twenty-six or thirty in a minute, owing to the failure of certain systoles to communicate a pulsation to the radial artery, the heart itself beating at the rate of fifty-six or sixty in a minute (FULLER, C. J. B. WILLIAMS, GAIRDNER). Exertion tends to increase the frequency of the pulse, and promotes irregularity of cardiac action. *The sounds of the heart* vary in accordance with coexisting valve lesions. "A loud bellows murmur" heard all over the chest, or with the first sound, or a *diastolic bruit*, may exist in the region of the aortic valves, the heart's action ultimately becoming tumultuous, and the sounds obscured. But the cardiac sounds are said also to be so modified in the absence of valvular disease, that a murmur has been ascribed to the morbid condition of the muscular tissue of the heart. Its impulse and sounds are feeble, or of limited extent; but when present, they are well defined. *Other symptoms* associated with fatty degenerescence are, severe vomitings and faintings, or a peculiar sinking and sense of faintness, with profuse perspirations; paleness and lividity, with urgent dyspnœa for some weeks before the fatal result. There is generally sudden aggravation of cardiac symptoms, of cough, or of general debility, for some days before death. "Biliousness" and headache have also been common symptoms for many months before death, sometimes with a slight indication of embolism.

Diagnosis.—The diagnosis of a degenerate heart is by no means easy. Fatty degeneration may exist without clinical indications, or only such as are useless for diagnosis. A diagnostic value has been attached to the existence of fatty atrophy of the cornea—"*arcus senilis;*" but as a sign of fatty heart it is by no means to be depended upon.

Prognosis.—In the greater number of cases death is sudden and unexpected, cardiac disease having never been observed, or only suspected to be present. It may thus go on for years. *The modes of death* may be *sudden,* almost *instantaneous;* or *comparatively sudden in bed,* the patients having been previously up at the night-chair. Some are found lying dead, or dying in a death-struggle of from *three* to *five* minutes' duration; or paroxysms of rigor, nausea, and "spasms of the stomach," with small and contracted pulse, occurring at intervals within twenty-four hours, at last suddenly prove fatal; others have died in a paroxysm of *angina pectoris,* or under the influence of chloroform. Death by syncope is usual. Thus died Dr. Chalmers, the eminent Scotch divine. It is also a mode of death apt to occur in convalescence from injury; as in the cases of Dr. Pereira, a distinguished physician, who died while recovering from rupture of the *tendo Achilles,* and of Sir Cresswell Cresswell, an eminent judge, who died while recovering from an injured patella. Such cases suggest the necessity of a cardiac examination, and the maintenance of a sufficiently stimulating diet and wine, with careful avoidance of such circumstances as may tend to syncope, in handling the injured parts of men otherwise healthy, but advanced in years.

Treatment.—*Iron* in its various forms, *quinine,* and *mineral acids,* are the medicinal agents suggested by the nature of this disease. Freedom from anxiety, thorough repose of mind, entire avoidance of fatigue, gentle but regular exercise in the open air, careful attention to the state of the skin, with a generous and nutritious diet at regular intervals, in moderate and equable amount at each meal, are the main hygienic indications calculated to impart tone to the system, improve the condition of the blood, and induce a more healthy nutrition of the heart (FULLER). A salt-water sponging bath should be used daily. In cases where digestion is sufficient, cod-liver oil, cream, and milk may be given with great advantage (TANNER). The bowels should be so regulated as to render straining at stool unnecessary.

ANEURISM OF THE HEART.

LATIN EQ., *Aneurysma;* FRENCH EQ., *Anévrisme;* GERMAN EQ., *Aneurysma;* ITALIAN EQ., *Aneurisma.*

Definition.—*A protrusion of the textures of the heart covered in by the endocardium and the visceral pericardium, caused by a degenerate portion of the wall of the heart yielding to the pressure of the blood.*

Pathology.—(*a.*) *Causation.*—It is usually a result of *myocarditis,* where a portion of the heart's texture is broken down by the effects of inflammation into a finely granular detritus, which, yielding to pressure of the blood, forms what is known as a true (or *chronic*) *aneurism of the heart.* The following theories are entertained regarding their formation as a result of,—(1.) Incomplete rupture (BRESCHET, CORVISART, LOBSTEIN). (2.) Ulceration or laceration of the endocardium (KREYSIG, LAENNEC, BOUILLAUD), as from endocarditis. Thus an *acute aneurism* of the heart is formed. (3.) Alteration of the lining membrane, the nature of which is not stated (REYNAUD, OLLIVIER). (4.) Inflammatory softening (DANCE, CHASSINAT, and HARTMANN). (5.) Inflammatory changes by which the muscular substance is replaced by fibrous material, *fibroid degeneration.* In these cases there is sometimes an extensive interstitial deposit thickening and indurating the textures. The new materials and the sarcal particles alike undergo degeneration within the muscular sarcolemma. There was evidence of existing or of previous pericarditis, and sometimes of rheumatism. The fibrillæ exhibited undoubted evidence of extreme brittleness (CRUVEILHIER, ROKITANSKY, CRAIGIE, PEACOCK, TORGET, MERCIER, THURNAM, NIEMEYER, and most modern pathologists). Such aneurisms may attain the size of a hazel-nut, walnut, or a hen's egg, or may even become as large as the heart itself. The entire heart in such cases is generally dilated.

(*b.*) *Morbid Anatomy.*—Numerous examples of such forms of disease are to be found described in the *Transactions of the Pathological Society of London.* One unique case is related by Mr. Henry Arnott (l. c., Vol. XIX., p. 149), "leading out from the upper part of the left ventricle, behind and below the aortic opening, forming a large pouch, extending backwards, upwards, and to the left, with a patent orifice two inches in diameter. Its walls, two to three lines thick, had undergone calcification to a considerable extent, studding the fibroid material of which they were composed. The aneurismal pouch springs most frequently from the left ventricle, and from the upper part of that cavity. One of the first cases related in the *Transactions* is an aneurism of the *left auricle*—a condition so rare that Rokitansky speaks of only one undoubted instance on record, besides one in the Pathological Museum at Vienna. A summary of seventy-four cases is given by Thurnam, in fifty-eight of which the left ventricle was the seat of the aneurism, and nine of these were of a very large size. One of them was so large, opening from the apex of the left ventricle, that it equalled the size of a second heart, and was filled with laminated coagula (*Med.-Chir. Trans.*, Vol. XXI., p. 227, 1858). In seventeen only of these cases was Dr. Thurnam able to obtain information as to the occupation of the sufferers; but of these seventeen cases he writes:—"It is a striking fact, that out of this number eight, or about one-half, should have been soldiers—a circumstance that would lead one to suspect that exposure, to which this class of persons is subjected, and the forced exercise they undergo, may have something to do with the production of this lesion."

Symptoms are obscure, and beyond general evidence of diseased heart and the physical signs of extended dulness, there are no reliable symptoms or signs. The sphygmograph may show evidence of prolonged arterial expansion, as in the case recorded by Mr. Henry Arnott, where the symptoms resembled those of aortic dilatation.

RUPTURE OF THE HEART.

LATIN EQ., *Diruptio;* FRENCH EQ., *Rupture;* GERMAN EQ., *Ruptur*—Syn., *Zerreissung;* ITALIAN EQ., *Rottura.*

Definition.—*Bursting or rupture of some part of the walls of the heart, including its visceral pericardium.*

Pathology.—(*a.*) *Causation.*—*Spontaneous rupture* of the walls of the heart has been occasionally met with during diseased conditions, especially *fatty degeneration, myocarditis, cardiac abscess,* and *true* or *acute cardiac aneurism.* Associated with any of those diseased conditions, the heart may rupture during some unusual exertion, death usually ensuing in such cases with symptoms of internal hæmorrhage.

(*b.*) *Morbid Anatomy.*—In these degenerated hearts the organ is flaccid, so that the walls collapse, and the changes are such as have been already noticed. Rupture of the right side of the heart is more rare than of the left. Rupture of the auricles is perhaps as frequent as that of the ventricles. The extent of the rupture, when it takes place in the ventricle, varies. It has been ruptured from its apex to its base, along the sulcus which separates the two ventricles. Generally the rupture is from ten to twelve lines; or the base of the ventricles may be severed from the aorta, and one of the aortic valves split transversely. It is remarkable, however, that the rupture from disease has seldom been found at the apex, where the walls of the heart have least force and consistency; but it is there that rupture from external injury takes place. The number of ruptures is as various as their seat. Out of forty-eight cases collected by Ollivier, eight were multiple (as many as from 2 to 5). Corvisart is the *first* who has given examples of another kind of rupture of the heart, that of the *carneæ columnæ,* or tendons of the valves. It is probable that rupture of these parts is owing more frequently to ramollissement, or to induration or ulceration, than to any other cause. Rupture of the *cordæ tendiniæ* sometimes occurs in connection with mitral valve disease, or in consequence of a violent fit of coughing, or it may follow some violent exertion. Evidence of such a lesion is seen after death in the ruptured filaments curling up, and forming little elevations of the retracted ends, which generally become covered with fibrinous coagula and hardened lymph. When several of the cords rupture, the edge of the valve-lappet recedes, forming a broad raised margin (*Navy Medical Report* for 1868, where excellent drawings are given of such a lesion).

Symptoms.—The first symptom in all the cases has been a sudden sense of suffocation, or a shriek followed by insensibility. The patient has in general suddenly died, but in some instances he has survived a few days. In rare instances a brief period of violent pain under the sternum has been experienced, shooting towards the left shoulder and along the arm. Usually death is sudden, the pericardium being filled with blood flowing from an outwardly irregular but smooth-edged rent of variable length, opening into the deeper flesh of the heart, which seems torn, mangled, and imbedded in blood-clot (NIEMEYER).

PARASITIC DISEASE OF THE HEART.

LATIN EQ., *Morbus Parasiticus;* FRENCH EQ., *Maladie Parasitaire;* GERMAN EQ., *Parasitische Krankheit;* ITALIAN EQ., *Malattia Parasitica.*

Definition.—*Lesions caused by the localisation of parasites in an immature or non-sexual condition in the substance of the heart.*

Pathology.—The nature of the parasites associated with such forms of disease has been already fully described in Vol. I., pp. 197 *et. seq.* It remains only to be noticed here that *cysticerci* have been found in the human heart when enormous numbers of them existed at the same time in other muscles of the body. The *echinococcus* and *trichina spiralis* have also been met with in the walls of the heart, beneath the inner membrane (Vol. I., p. 159).

MALFORMATIONS OF THE HEART.

LATIN EQ., *Deformitates Ingenitæ;* FRENCH EQ., *Vices de Conformation;* GERMAN EQ., *Missbildungen;* ITALIAN EQ., *Vizi di Conformazione.*

Definition.—*Deviations from the normal development of the heart, occurring in the earlier periods of gestation, and before the termination of fœtal life.*

Pathology.—After what has been written regarding malformations in Vol. I., p. 251, the more practically important and common malformations of the heart may be here mentioned:—(1.) *Misplacements of the heart* (*Ectopia cordis*), occurring congenitally, as in cases of transposition of the viscera. For an account of this subject, the reader is referred to a paper by Professor Allen Thomson in the *Glasgow Medical Journal* for July, 1853; and to the *Lancet* of August 8, 1863, where the condition is described by Professor W. C. Maclean as having been recognised during life. For an excellent report on cases of *Ectopia cordis*, see *Pathological Society's Transactions*, Vol. VI., p. 98. (2.) *Congenital deficiency, or absence of the pericardium*, in which the heart is, as it were, naked, and lying in one cavity with the lungs (*Pathological Society's Transactions*, Vol. III., p. 60; Vol. VI., p. 109). (3.) *Arrest of development of the heart at an early period of fœtal life*—as in hearts with one auricle and one ventricle, with imperfect separation of the ventricles; contraction or absence of the pulmonary artery, the aorta arising from the right ventricle, and the *septum* of the ventricles imperfect. In cases of imperfect *septa* the aorta sometimes arises from the two ventricles. Sometimes the ventricular *septum* is wholly absent; as in a case of *univentricular* or *tricœlian heart* described by Dr. Elliot, of Carlisle, in the person of a clerk in a public office, who lived to the age of nineteen years and eight months. There was transposition of the great vessels; a small aorta, and large-sized pulmonary artery; a total absence of the ventricular septum. There was all but total freedom from pericardial adhesion, with equally healthy and efficient lungs. The cyanotic signs were observed at the age of three months, and gradually increased as he grew older. The heart's action was thumping, with a whizzing bruit, which only occasionally accompanied the second sound. The pulse was "shabby," variable in force, but in frequency always about eighty. He died of gradual exhaustion. The heart was dissected by Dr. Embleton of Newcastle-on-Tyne; and photographs having been taken, they were kindly distributed by Dr. Elliot to the Museums of Netley, London, Dublin, Edinburgh, and Glasgow (*Journal of Anatomy and Physiology,* Vol. XI.). Somewhat similar cases have been described by Drs. Peacock and Cockle. (4.) *Premature closure of fœtal passages—the foramen ovale and ductus arteriosus—malformations causing changes which ought not to ensue till after birth.* The consequences of such premature results are chiefly cyanosis, combined with imperfect dilatation of the branches of the pulmonary artery. (5.) *Irregularity of the valves and origins of vessels, which may not in the first instance interfere with the functions of the heart, but which are apt to lay the foundations of disease in after life.* Examples are to be seen in cases where two aortic valves occur; where there is fusion or union of two of the valves; where there is transposition of the aorta and pulmonary artery, both auricles opening into the left ventricle (l. c., Vol. VI., p. 117). Excess of pulmonary valves (l. c., Vol. III., p. 301; Vol. IV., p. 102). (See also Vol. I., of this work, p. 251).

The **Treatment** of malformations which are associated with cyanosis is mainly preventive of dyspnœa and palpitation, by the avoidance of fatigue and mental excitement, the maintenance of temperature, and especially by a nourishing diet and warm clothing.

CYANOSIS.

Latin Eq., *Cyanosis;* French Eq., *Cyanose;* German Eq., *Cyanose*—Syn., *Blausucht;* Italian Eq., *Cianosi.*

Definition.—*A peculiar blue condition of the skin, symptomatic of various malformations or derangements of the heart and great vessels, so that a small portion only of the blood is subjected to aëration in the lungs.*

Pathology.—(*a.*) *Causation*—The blood-corpuscles absorbing oxygen in the lungs convey it through the circulation to every part of the body, and thereby render the metamorphosis of tissue possible. If this absorption of oxygen in the lungs by the blood-corpuscles is for any reason stopped or diminished, *arterial* blood retains the properties of *venous* blood, and the condition termed *cyanosis* arises. The causes of cyanosis are:—(1.) When the supply of oxygen to the blood, and consequently the oxidation of the blood-corpuscles, is prevented by derangements of the respiration or circulation (*i.e.,* by causes which are external to the blood); (2.) When the blood-corpuscles lose the property of absorbing oxygen. This occurs in some cases of *pyæmia* and the last stage of *pulmonary tuberculosis.* There seems to be a diminished capacity of the blood-corpuscles to redden themselves (Vogel).

Symptoms.—In its *minor degree* it is associated with forms of cardiac and pulmonary derangement, having the effect of obstructing the flow of blood in the veins of the lungs and of the system generally. The chief of these is constriction or partial obstruction of the pulmonary artery, combined with systemic venous engorgement. In such an acute form it may be associated with *croup, diseases of the heart*, and *cholera*. The skin is usually thin, the capillaries abnormally large; hence, when obstruction to the pulmonary and systemic venous circulation causes imperfectly aërated blood to flow throughout the system—and still more so, when, in consequence of some congenital malformation, a small portion only of the blood is subjected to the aërating influence of respiration—a dark, dusky, more or less livid hue, is imparted to the skin. *In its more severe form* it is usually associated with such a malformation, disease, or injury of the heart or great vessels, as permits venous and arterial blood to mix, and after mixture to be distributed to the systemic capillaries. A patulous condition of the *ductus arteriosus*, an open *foramen ovale*, a deficiency of part of the *septum* of the ventricles, a heart formed of one ventricle and one auricle only—the aorta and pulmonary artery rising from a common trunk—are amongst the usual conditions which lead to cyanosis. It is usually a congenital affection, and the physical signs vary with the precise condition of the heart and arteries to which the cyanotic discoloration is due. The action of the heart is more forcible than in health, and hypertrophy and dilatation of the right ventricle are almost always present. Deficiency of animal heat is a constant phenomenon.

Treatment must depend upon the nature of the lesion or disease which gives rise to the cyanosis. The inhalation of oxygen gas has given relief in some cases.

ANGINA PECTORIS, OR BREAST PANG.

LATIN EQ., *Angina Pectoris;* FRENCH EQ., *Angine de Poitrine;* GERMAN EQ., *Angina Pectoris;* ITALIAN EQ, *Angina Pectoris.*

Definition.—*Pain or spasm of a weakened heart* (CHEVERS), *referred to the lower part of the sternum, or to the præcordial region, extending through the chest to the left scapula, and up the sternum to the root of the neck. The pain is characterised by its suddenness, its severity, and by a sense of constriction or of burning. It compels the patient, if walking, instantly to stop, and almost prevents inspiration. The pain is felt likewise in the left shoulder, whence it sometimes reaches to the elbow, rarely to the hand, often with a sensation of numbness in the parts. A tendency to syncope exists, associated with intense anxiety, and a sensation of approaching dissolution.*

Pathology.—(*a.*) *Historical.*—This disease attracted little attention till Dr. Heberden drew the attention of the profession to it by two papers published in the second and third volumes of the *Transactions of the London College of Physicians* in 1772. He connected it with disease of the heart; and it has ever since been treated of in conjunction with cardiac diseases. It has been since elucidated by the writings of Black, Parry, and Jenner, and by many Continental physicians. Dr. Parry's work—*An Inquiry into the Symptoms and Causes of the Syncope Anginosa, commonly called Angina Pectoris*—will repay perusal, though published so long ago as 1799.

(*b.*) *Causation.*—The immediate cause of the "breast pang" appears to be a sudden impediment to the *coronary* circulation, and particularly to the return of the blood by the coronary veins—due to a temporarily over-distended state of the chambers of the heart, and an inability in them to empty themselves, whether owing to weakness of the muscular parietes of the left ventricle or to other causes. The organic lesions apt to be attended by *angina* are conditions of the aortic valves which permit of free regurgitation, with a rigid dilated state of the ascending portion of the arch of the aorta, which permits the blood to regurgitate, combined with either of the following conditions of the left ventricle :—(1.) Dilatation of the cavity; (2.) attenuation of the parietes; (3.) softening or degeneration of the muscular tissue (BELLINGHAM). *Angina* may not necessarily occur, so long as the circulation continues tranquil, and so long as the left ventricle is able to get rid of the blood which enters its cavity, and does not get over-distended. But if the heart's action becomes disturbed by some sudden mental emotion; or even without this occurring, if the stomach is loaded with indigestible food, and it and the intestines are distended with flatus, by which the cavity of the chest is encroached upon, and the heart's movements impeded, a paroxysm of *angina* is the general result. John Hunter, who suffered from this disease, used to say that his life was in the hands of any person or circumstance which acted powerfully on his mind; and he ultimately died in St. George's Hospital from strong but suppressed feelings on a point in which he was deeply interested. Ascending a staircase or other acclivity, or any active exertion, is a powerful exciting cause of a paroxysm. In persons who have had previous attacks, the paroxysm is liable to supervene during sleep, as the result of a frightful dream disturbing the heart's action, or of considerable distension of the stomach by flatus impeding the movements of the heart. *Plethora* becomes a very common complication, on account of the sedentary life which must be led, to the abandonment of all active bodily exertion. *Angina pectoris* ought, therefore, to be regarded as a *symptom of organic disease of the heart.* What *dyspnœa* is to the lungs, *angina* appears to be to the heart; and Bellingham has termed it the "*dyspnœa of the heart.*" It has also been defined as *hyperæsthesia* of the cardiac plexus (ROMBERG); and as *hypercinesis* (excessive motion), with *hyperæsthesia,* the cardiac plexus being assumed to be the source of the pain (BAMBERGER). It is most common in the male sex; and rarely occurs under forty-five or fifty years of age. Its paroxysmal character, with intervals of immunity, associates its pathology with disorder of the nervous system of the heart, to which the organic changes serve as a predisposing cause (NIEMEYER).

Symptoms.—The paroxysms generally supervene suddenly, and are characterised by a constrictive pain, most commonly in the left lower half of the sternum, and rarely extending above the fourth rib. Occasionally, it extends over the whole anterior portions of the chest, along the neck to the lower jaw, into the back and shoulder, down the arm to the elbow, and even to the hand and fingers. In this course it affects the superficial cervical plexus and its ramifications, as well as the anterior thoracic nerves, the cubital nerve and its divisions. The pain is sometimes substernal; it then follows the course of the nervous plexus placed between the folds of the mediastinum and the branches of the eighth pair which go to the large arteries and surround the bronchial tubes. The action of the diaphragm, and consequently the phrenic nerves, may be affected; while the lumbar and sacral nerves partake of the pain. The pulse may be rapid or hardly to be felt; and the breathing is sometimes accelerated, at other times it is imperceptible. Mr. Hunter, when labouring under the paroxysm, could scarcely feel his pulse, and thought he should die unless he exerted his voluntary muscles to aid respiration. Many have died so —asphyxiated. The gastric system is also affected, the patient being suddenly distended with flatus, only relieved by repeated eructations. The urine may be suppressed. The face and extremities are usually cold and pale. The paroxysm subsides gradually, when much flatus is discharged, accompanied by a copious flow of pale limpid urine, when the patient for the time recovers. The duration of the paroxysm depends on the persistence of the impediment to the coronary circulation. Sometimes the pain lasts a few minutes, at other times it will continue for two or three hours, a whole day, or even longer. The interval of respite from pain may be from a few hours to a few days, or a few months. Each repetition promotes the tendency of the paroxysm to return, and increases its violence. At length an aggravated attack may occur which puts a period to the patient's existence.

Prognosis.—The danger is in proportion to the nature and degree of the organic lesion on which the disease depends. Of sixty-four recorded cases of angina forty-nine died, almost all of them suddenly (Sir JOHN FORBES).

Treatment.—The indications are to be found in a study of the lesions on which the paroxysms mainly depend. Medicine can do little more than mitigate the severity of an attack. This is generally best done by diffusible stimulants, such as *brandy*, *ether*, *chloroform*, *ammonia*, *chlorodyne*. *Alcohol*, in small doses often repeated, *sesqui-carbonate of ammonia*, in doses of from *three* to *five* grains, the *muriate of ammonia*, in doses of from *ten* to *twenty* grains, have each powerful stimulant effects. Hot bottles and sinapisms should be applied to the feet. The bowels may require to be rapidly and efficiently acted upon. Thirty *minims* of *tincture* of digitalis, repeated at the end of half an hour, give great relief. It has also been beneficial when the angina was associated with fatty heart. In that form of *angina pectoris* where there is distension of the right ventricle, accompanied by palpitation, dyspnœa, and lividity of the face (*cardiac asthma*), the frequent use of digitalis exercises a stimulating influence on the sympathetic cardiac ganglia and muscular fibres (FOTHERGILL).

PALPITATION AND IRREGULARITY OF THE ACTION OF THE HEART.

LATIN EQ., *Palpitatio et Tumultus Cordis;* FRENCH EQ., *Palpitations;*
GERMAN EQ., *Herzklopfen und Unregelmässigkeit in der Herzthätigkeit;*
ITALIAN EQ., *Palpitazione ed Irregolarità del Cuore.*

Definition.—*Palpitation and irregularity of the action of the heart, unconnected with organic mischief, beyond, in some cases, a notable hypertrophy.*

Pathology.—(*a.*) *Causation.*—The interest which attaches to this conditio is due to the difficulty of recognising *functional* ("*neurosis*") as distinguishe from *organic* disease; and to the fact that great uneasiness and distress, both mental and bodily, result from the occurrence of functional disturbance, while its persistence is apt to induce organic disease. Death has resulted from simple functional disturbance (GRAVES).

The cardiac irregularities are of the following kind:—The heart may beat abnormally slow, may intermit, may have a rolling action, or its pulsations may be so frequent, and its actions so irregular, as to be termed *Palpitation*, or excessive action of the heart. These states are believed to be caused by irregular innervation of the heart, which renders it morbidly sensible or insensible to its natural stimulus, the blood. The excessively slow pulse may be caused by pressure high up in the cervical portion of the spinal cord, or by congestion or pressure on the brain. The other states are perhaps inexplicable, and may be considered as ultimate facts. The irregular and rolling action of the heart is in general accompanied by hypertrophy, or other lesion. Palpitation, however, may occur in the most healthy subjects, and in the most healthy hearts. The excitability of the hearts of young people is readily accumulated and as readily exhausted. Every one is aware how powerfully the passions and affections act on the heart and change its healthy beat; how every error in diet, or over-exertion, may produce the same effect. Moral as well as physical causes may thus be the remote agents in the production of palpitation, while many lesions may be accompanied by it. As a primary disease, palpitation seldom occurs before puberty; but after that period it is common, often to a most distressing degree, in both sexes. The female suffers more than the male, and especially during amenorrhœa, or at the period of menstruation, and in more advanced life when menstruation ceases.

While palpitation may merely be a disease of the function of the heart, as is evident from the number of young persons who suffer from it, and who afterwards attain a hale old age, it is also a common symptom of disease of the heart. Palpitation, regarded as over-action of the heart, is apt to engender the belief that it indicates excess of power. But the palpitating heart is more nearly allied to asthenia. It is evidently a laborious effort on the part of the heart, and indicates that it is over-taxed. Thus, habitual palpitation at length terminates in hypertrophy or dilatation, a result abundantly confirmed in military experience. It is generally intimately connected with more or less of ventricular engorgement, especially as a result of the obstruction to circulation from diminution in the calibre of the arteries, as in Bright's disease, jaundice, and whenever poisoned blood is coursing through the vessels. The causes of such phenomena are common to adolescence and middle adult life, and are most frequently traceable to excessive mental exertion and sedentary occupation; great anxiety and strong mental emotion; nervous exhaustion from various causes, such as uterine irritation, excessive venery, masturbation; the influence of poisons on the heart's action, such as tea, tobacco, opium, and alcohol in excess; *gout*, *rheumatism*, *dyspepsia*, characterised by the existence of flatulence and acidity (FULLER, HAYDEN). Functional disorder, under the name of "*irritable heart*," is common among soldiers, and is occasionally met with in young persons in civil life. It was very common among the soldiers during the late American war, and was considered due to *cardiac muscular exhaustion* (HARTSHORNE) It was familiarly known as the "trotting heart," characterised by great frequency of action, constantly recurring attacks of palpitation, and pain in the præcordial region. Palpitation happened chiefly during exercise, but often when at rest, as in the night, hindering sleep. Dizziness and headache were sometimes constant, or only during the fits of palpitation. Pain was dull and constant, or shooting and paroxysmal; its site generally over the apex, where the skin was sometimes also sensitive. The antecedents were generally—(1.) *Forced marches;* (2.) *fever;* (3.) *camp diarrhœa* (CLYMER, DA COSTA, HARTSHORNE, *Amer. Journal of Medical Science*, July, 1864).

Symptoms.—The very rapid action of the heart is accompanied by an abrupt jerky impulse of irregular rhythm, with a short sharp first sound, and a distinct second sound. Sometimes the first sound was hardly to be heard, as in the asthenia of continued fever. There were no cardiac nor neck murmurs, and the area of percussion dulness was not increased. The pulse was greatly influenced by position, being less by twenty beats when the patient was lying down, and usually compressible. The breathing was generally distressed on exertion, but otherwise there was no constant increased frequency of respiration, and the general health was often good. Palpitation may come on suddenly, or it may be preceded by acidity, flatulence, or other indications of indigestion. In young persons of a delicate constitution it often occurs in a slight degree nightly; so that, on going to bed, many sleepless hours may be passed, not only feeling the heart beat, but also hearing it. Subsequent sleep is unrefreshing, and the patient awakes in the morning more tired and jaded than when he went to bed. In young women labouring under leucorrhœa, palpitation may be constant, the pulse beating for many weeks at 150 to 180 strokes in a minute. In other cases it is paroxysmal, when the pulse may preserve a regular rhythm, only greatly increased in frequency, while its force may be increased or diminished. In severe cases the pulse is so rapid that it has a mere vibratory motion, and cannot be counted, while its rhythm is extremely irregular. The force of the heart's action is at one time excessive, and at another it may not be felt, and appears to act with every possible degree of irregularity. Thus, it is characterised by an accelerated and sometimes unrhythmical beating, accompanied usually by a feeling of dread and by dyspnœa. In general, other branches of the eighth pair are affected besides the cardiac branch; the patient becoming distressingly and suddenly distended with flatus, while his deep sighing shows the pulmonary as well as the gastric branch to be involved. Having lain in this state, pale, anxious, and restless, for a greater or less time, the fit at length terminates, and the pulse is restored to its natura frequency and healthy rhythm as instantaneously as the attack had been sudden. A considerable quantity of pale limpid urine may then be passed and, though feeble from exhaustion, the patient is once more able t sit up and so far to exert himself. Sometimes the paroxysm lasts a fe minutes, or a few hours, but with organic disease it may become more o less persistent. The interval between the paroxysm is also uncertai In young persons it may recur every twenty-four hours, two or three tim a week, or every month, or at other irregular intervals.

Diagnosis.—The fact of palpitation cannot be mistaken; but the natu and causation of the irregularity is not always easy of recognition. T forms of functional disturbance which simulate organic disease are, palpitation, fluttering, or a "rolling" action, sometimes associated with a valvular murmur and irregularity, feebleness, or altered rhythm. Exaggeration of functional acts is characteristic. The general symptoms indicate great distress, a sense of fulness and of deep oppression in the *præcordial* region, pain, breathlessness, and tendency to faintness. Frequent giddiness, with pain, heat of head, singing in the ears, flushing of the face, coldness of the extremities, are associated with forms of dyspepsia characterised by excessive flatulence, acid eructations, restlessness at night, depression of spirits, and mental anxiety as to the nature and probable issue of the disease, which all contribute to induce and maintain the functional disturbance of the heart (FULLER, also HAYDEN, *Brit. Med. Journ.*, June 5, 1880).

In the following table the more prominent characters of palpitation which depends upon organic disease of the heart, are contrasted with those of palpitation arising independent of disease of this organ (BELLINGHAM, *op. cit.*, p. 172):—

PALPITATION DEPENDING UPON ORGANIC DISEASE OF THE HEART.	PALPITATION INDEPENDENT OF ORGANIC DISEASE OF THE HEART (NEUROSIS).
1. More common in the male than the female.	1. More common in the female than the male.
2. Usually comes on slowly and gradually.	2. Usually sets in suddenly.
3. Constant, though more marked at one period than at another.	3. Not constant, having perfect intermissions.
4. Impulse usually stronger than natural; sometimes remarkably increased, heaving, and prolonged; at others irregular and unequal.	4. Impulse neither heaving nor prolonged; often abrupt, knocking, jumping, and circumscribed, and accompanied by a fluttering sensation in the præcordial region or epigastrium.
5. Percussion elicits a dull sound over an increased surface, and the degree of dulness is greater than natural.	5. Extent of surface in the region of the heart, which yields naturally a dull sound on percussion, not increased.
6. Often accompanied by the auscultatory signs of diseased valves.	6. Auscultatory signs of diseased valves absent; bruit de soufflet often present in the large arteries, and a continuous murmur in the veins.
7. Rhythm of the heart regular, irregular, or intermittent; its action not necessarily quickened.	7. Rhythm of heart usually regular, sometimes intermittent; its action generally more rapid than natural.
8. Not much complained of by the patient, occasionally attended by severe pain, extending to the left shoulder and arm.	8. Much complained of by the patient; readily induced by mental emotion; and frequently accompanied by pain in the left side.
9. Lips and cheeks often livid; countenance congested; anasarca of lower extremities common.	9. Lips and cheeks never livid; countenance often chlorotic; anasarca absent, except in extreme cases.
10. Increased by exercise, by stimulants, and tonics, &c.; relieved by rest, and frequently, also, by local or general bleeding, and an antiphlogistic regimen.	10. Increased by sedentary occupations; by local and general bleeding, &c.; relieved by moderate exercise, and by stimulants or tonics, particularly the preparations of iron.

Repeated examinations are necessary to arrive at a just conclusion. Perversion of rhythm has been attributed to functional disturbance merely; but cardiac rhythm, when altered, is not merely of temporary duration, but is attended with giddiness, faintness, or actual syncope; and then it is more likely, in the absence of positive knowledge as to the state of the heart's tissue, that such altered rhythm is due to organic or textural degeneration. Dr. Fuller found it so in every case he examined.

Dr. Meyers, Surgeon of the Coldstream Guards, has shown how this irritable condition of the heart is capable of demonstration by the sphygmograph from the marked dicrotism which exists. It proves that the heart is overtaxed long before any change can be detected by auscultation or percussion.

Prognosis.—Palpitation is seldom dangerous, unless conjoined with organic disease; and when merely functional in youth, it frequently subsides as the patient advances in life. It is, however, always an obstinate disorder, and wholly unfits a man for any active duty.

Treatment.—During the paroxysm the patient should lie flat on his back, bare his neck and chest, and allow the air to blow freely over him. The best remedies are, *camphor mixture* and *æther*, ʒi., with some slight opiate, as the *syrup of poppies*, ʒj., or *tinct. hyoscyami*, ♏xx. These should be repeated every quarter, or every half hour, or hour, according to the severity of the attack, till the heart's action is soothed. Cold brandy and water may be a substitute for, or adjuvant to, such medicines. If the attack occurs shortly after a meal, some purgative should be given to clear the stomach and bowels. *Bicarbonate of potash* (in 10 to 20 grain doses) is then also an excellent stimulant as well as antacid and sedative. Leeches to the *os uteri* in females, and to the *perineum* in males, sometimes relieve palpitation—proceeding in the one case from uterine disturbance, or from varicose veins, hypochondriasis, or hepatic congestion in either sex. *Bromide of potassium* and *ammoniated tincture* of valerian are also indicated. *Digitalis* is useful under the following conditions:—(*a.*) When there are palpitation and acute depression of the heart's action from *shock* (*Paralysis of the sympathetic* of Romberg), marked by a small feeble pulse, coldness of skin, cold breath, diminished temperature, and evidence of the organic system of nerves being acutely depressed. The heart's walls contract ineffectively. It does not respond to the stimulus of its contents. Combined with other stimulants, the administration of digitalis in a case of *shock*, following parturition, was followed by the most satisfactory results (Drs. FOTHERGILL and WILKS, *Med. Times and Gazette*, Jan. 16, 1864). In Dr. Wilks' case, the patient seemed to be *in articulo mortis*—the face livid, no pulse at the wrist, and a mere fluttering heard over the region of the heart. *Brandy* and *ether* had been given with no result; and dissolution being imminent, it was determined to administer *digitalis*, o which half-drachm doses of the *tincture* were given every hour. Afte four doses reaction took place, and after seven doses complete recover occurred. The full veins and condition of the heart indicated distensio and paralysis (FOTHERGILL, *Brit. Med. Journal*, July 29, 1871). (*b.* When there is the irregular action of cardiac asthenia. (*c.*) When ther is palpitation in dilated heart. It is useful to continue the medicin which have been mentioned, but at longer intervals, for some time. It important also to counsel the patient strictly as to diet. It is often four that the palpitation returns after tea or after breakfast, or whenev hot tea or hot coffee has been drunk. In these cases it is desirable avoid all hot slops, and to drink cold water at breakfast, and indeed every meal. There are few tonics so beneficial as the natural tonic of cold water; and persons once accustomed to it feel a return to a modern breakfast a punishment rather than a gratification. Wine should not be taken, unless largely diluted with water, to be prescribed as required.

CHAPTER XVII.

DISEASES OF THE BLOOD-VESSELS.

(*a.*) *Diseases of the Arteries.*

ARTERITIS.

LATIN EQ., *Arteritis;* FRENCH EQ., *Artérite;* GERMAN EQ., *Entzündung der Arterien*—Syn., *Arteritis;* ITALIAN EQ., *Arteritide.*

Definition.—*Inflammation of the textures of an artery.*

Pathology.—(*a.*) *Causation.*—Gout, rheumatism, and syphilis are active agents in the development of arteritis. The influence of irritants, such as alcohol, of local injuries, strain or distension, and of senile degeneration, must also be recognised as causes of arteritis leading to atheroma.

(*b.*) *Morbid Anatomy.*—Inflammation of arteries, chiefly affecting the outer coat—peri-arteritis—is a disease by no means rare. It is the only form of acute inflammation of arteries, and the results are grave and serious, according to the region where arterial inflammation occurs, especially in connection with the changes known as *atheroma.* These have been shown to be "in continuity with *arteritis,*" and to graduate from a condition in which no inflammatory results can be found into one in which inflammation is unmistakably present (MOXON). Mere redness of an artery, without any alteration of the inner membrane, may be simply cadaveric, owing to imbibition of blood-pigments. The most dangerous place for arteritis to occur is the aorta, especially the arch, where the induration and loss of elasticity are attended by such impairment of function as leads sooner or later to death. This is called "*aortitis.*" The *post-mortem* appearances of true arteritis are,—great vascularity, especially of the *vasa vasorum,* a thickened and pulpy state of the inner and lining membrane, which, having lost its glistening aspect, has an opaque, villous, rugous, or granular aspect. An "*albuminous exudation*" (BIZOT), more or less thick, at first of the consistence of jelly, transparent and smooth—sometimes rose-coloured or colourless, occurs in patches, solitary or numerous, covering the lining membrane. It is so transparent that it may escape notice, unless carefully looked for. The calibre of the vessel is diminished, so that in the smaller arteries the exudation may plug them up. The common positions of such exudation in the aorta are—(1.) at the orifices of arteries going off from it; (2.) sinuses of Valsalva and coronary arteries; (3.) arteries arising from arch; (4.)

mouths of intercostals; (5.) cœliac, mesenteric and renal. The more recent descriptions of the lesion by MM. Cornil and Ranvier do not differ essentially from that given by Bizot. Swellings in the form of flat patches of various size, colour, and consistence, are characteristic appearances. Microscopically these patches are composed of embryonic cells, the germination of the normal cell-elements of the internal coat. It is this multiplication of elements and activity of growth which characterise the more acute process from the chronic atheromatous changes, where similar germination penetrates into the deep layer of the internal coat.

Chronic endoarteritis commences with relaxation and infiltration of the texture, and leads up to atheromatous disease. In one form there is gelatinous thickening of the inner coat, so that a moist, pale, reddish layer seems to lie upon the inner aspect of the artery—sometimes in spots, sometimes spread over a considerable extent of surface. This layer consists of a liquid resembling mucus, in which fine elastic fibres and round or spindle-shaped cells lie imbedded, the whole covered by epithelium (NIEMEYER). There may also be semi-cartilaginous induration, in opaque, bluish plates, like boiled white of egg, lying on the inner surface of the artery. Under the microscope numerous fusiform and reticulate cells are seen in broad fasciculi of connective tissue. Lymph may be seen on the surface of the vessel, and slight excoriations or superficial ulcerations may be observed here and there. The elasticity of the artery no longer exists, and rupture takes place very readily. When inflammation begins in the middle tunic, the tissue beneath the internal coat is of a speckled redness. The spots soon become white or yellow, and elevated above the surface, resembling small pustules, which may liquefy, so that *abscesses* have been established in the wall of an artery (NIEMEYER).

Inflammation of the several tissues of the aorta is similar to the process in *pericarditis*, *myocarditis*, and *endocarditis*. The external coats (*tunica adventitia*) and the connective tissue are mainly inflamed by continuity, as when inflammation or ulceration of the adjoining lymphatic glands, the trachea, or other neighbouring organs, extend to the aorta. In the chronic form it is often involved in *pericarditis*, and in some cases of *endocarditis* its inner coats may be extensively infiltrated by diffused inflammatory products (NIEMEYER). The middle structures are most prone to undergo fatty degeneration, or simple atrophy. Chronic inflammation of the internal coat (forming the incipient stage of ossification and atheroma), is one of the most frequent of diseases (VIRCHOW, NIEMEYER).

Symptoms.—Local pulsation of large vessels (like the aorta) is extremely energetic; and as in inflammation of the carotid there is pain when the vessel is pressed upon, so pain in an inflamed aorta, or in any artery, may be aggravated by motion. A murmur is appreciable in *aortitis*—a loud, rough, systolic bruit—due to the passage of blood over the rugous and unpolished state of the inner membrane (GENDRIN, BAMBERGER, PARKES). In a case related by Parkes it was heard over the third dorsal vertebra, down into the lumbar region (*Med. Times*, Feb. 23, 1850). Hence the importance of a stethoscopic examination along the course of the descending aorta in all suspicious cases. So long as aneurism does not occur, nor rupture or stoppage of one of the smaller vessels by the formation or arrest of a clot of fibrine, there may be few or no symptoms to indicate chronic inflammation. But if the results of chronic arteritis are extensive, especially in the form of *atheroma*, the demands upon the heart become increased, and hypertrophy may arise. If the chronic inflammation spread from the arterial walls to the valves, insufficiency and stenosis may be the result. If insufficiency of the aortic valves occur, the hypertrophy, if existing, may not be maintained, from failure or imperfect supply of blood through the coronary arteries, and so degeneration of the substance of the heart may result. Then follow all the usual symptoms of retarded circulation, with overloading of the venous system—cyanosis, dropsy, and suppression of urine. Evidence of disease in the peripheral arteries warrants the belief that *aortitis* may also be present in a more advanced stage of development. As the peripheral vessels become more dilated, and their walls more rigid, the pulse generally feels hard and full; and the course of the elongated arteries, as seen in the temporal and radial regions in thin persons, is remarkably sinuous, and their curvature increases with each beat of the pulse, which is visible. The artery can be felt as a hard, irregular cord (*endoarteritis*).

Treatment.—Leeches should be applied freely over the course of the vessel; and large doses of *opium*, or of *ether* with *chloroform*, may be given to relieve pain and dyspnœa. *Iodide of potassium* or *colchicum* may also be indicated, according to the constitutional morbid condition which has brought about the lesion.

FATTY AND CALCAREOUS DEGENERATION OF ARTERIES—Syn., ATHEROMA, OSSIFICATION.

LATIN EQ., *Degeneratio, Adiposa et Calcarea*—Idem valent, *Atheroma, Conversio in Calcem;* FRENCH EQ., *Dégénérescence, Graisseuse et Calcaire*—Syn., *Atheroma, Ossification;* GERMAN EQ., *Fettige und Kalkförmige Entartung*—Syn., *Atheroma, Verknöcherung;* ITALIAN EQ., *Degenerazione, Grassosa e Calcarea*—Syn., *Atheroma, Ossificazione.*

Definition.—*Results of parenchymatous inflammation of the inner arterial tunics.*

Pathology.—*Causation and Morbid Anatomy.*—Chronic inflammation of the internal coat of the arteries is a frequent disease (VIRCHOW, NIEMEYER). The relaxation and infiltration of the internal coat already described, where there is gelatinous and semicartilaginous thickening of the inner arterial tunic, show that the development of atheroma proceeds from changes of the nature of parenchymatous inflammation. A microscopic examination of an atheromatous patch shows that it consists of soft yellow matter, and gives proof of a disorganised and degenerate state of the walls of the blood-vessels by the conspicuous yellow faded-looking patches, accompanied in graver states by a stony layer, or a soft matter, made up of the common products of tissue-degeneration—namely, granules of lime and fat, shreds of tissue and cholesterine plates (MOXON). In the form of *endoarteritis deformans* it is a common disease of advanced age. A large proportion of cases of inflammation of the larger vessels, ending in *atheroma*, are of syphilitic origin. Such spots of chronic inflammation are always found at the points most exposed to strains or distensions, such as the ascending portion and arch of the aorta, and the places of origin of the vessels which pass off laterally.

Fatty degeneration of small capillaries, and atheroma of large arteries, are not the same lesions; so that two processes must be distinguished in the blood-vessels, which are very similar in their ultimate results:—(1.) *Simple fatty metamorphosis*, which sets in without any discoverable preliminary stage, in which the existing histological elements pass directly into a state of fatty degeneration and are destroyed, so that a larger or smaller proportion of the constituents of the walls of the vessel perishes. (2.) A series of changes in which *a stage of irritation* precedes the fatty metamorphosis, comparable to the stage of swelling, cloudiness, and enlargement which we see in other inflamed parts (parenchymatous inflammation); thus the old view which recognises an inflammation of the inner arterial coat, is seen to be the starting-point of the so-called atheromatous degeneration, and this kind of inflammatory affection of the arterial coat is, in point of fact, exactly the same as what is universally termed *endocarditis*, when it occurs in the lining membrane of the heart. There is no other difference between the two processes than that the one more frequently runs an acute, the other a chronic course. There are, especially, *three constituents*—namely, *cholesterine, granule-cells and fat granules, and, finally, large lumps of half-softened substance, which give the atheromatous matter its pultaceous character*, and which produce a certain degree of resemblance to the contents of a pultaceous—sebaceous, epidermic—cyst of the skin.

It has been maintained that ossification may occur in such arteries and not a mere calcification; and that the plates which pervade the inner wall of the vessel are real plates of bone. I have ground down many, but have never found true bone structure—an observation which I find in accordance with the experience of Rindfleish. The essential difference which exists in a large vessel (as the aorta) between this process (*atheroma*) and simple fatty degeneration, is this, that in the latter a very slight swelling arises on the surface of the internal coat, a swelling which at once disappears if the superficial layers be removed, and beneath which there still remains a portion of the coat unaltered. In atheroma we have, in the extreme stage, a depot which lies deep beneath the comparatively normal surface, covered in by a thin film of internal coat, which separates the greasy paste from the current of blood (the *atheromatous pustule*), which afterwards bursts, discharges its contents, and forms the *atheromatous ulcer*. This commences as a small hole in the internal coat, through which the thick, viscous contents of the atheromatous depot are squeezed out on to the surface in the form of a plug; gradually more and more contents are evacuated and carried away by the blood-stream, until at last there remains an ulcer, which may extend as far as the middle coat, and not unfrequently involves it. We have, therefore, always to deal with serious disease of the vessel, leading to results as destructive as in other acute inflammatory processes. Although *atheroma* has generally been regarded as evidence of degeneration; and the term "*atheromatous*" a descriptive of "the defaced interior of arteries as commonly seen in peopl of middle age or older," yet *atheroma* ought to carry with it a meaning o active change, and be held as evidence of a previously active germination of cell-elements, the final ending of an *arteritis* (MOXON).

OCCLUSION OF ARTERIES.

LATIN EQ., *Arteriæ Occlusæ;* FRENCH EQ., *Occlusion;* GERMAN EQ., *Versachliessung;* ITALIAN EQ., *Chiusura.*

Definition.—*Lesions of a local kind which lead to a stoppage of the flow of blood through an artery at the seat of the lesion.*

Pathology.—(*a.*) *Causation.*—There are two sets of lesions whose nature requires investigation in all these cases—namely, (1.) How do the vessels become occluded? (2.) What are the conditions which are known to determine the formation of coagula within the circulating system? These two questions must be considered, (1.) as regards the arteries, and (2.) as regards the veins. Exclusive of abnormal narrowness and contractions, often congenital, *occlusion of arteries* arises from the following lesions:—(1.) *Contractions and obliterations,* as parts become disused in the course of development. (2.) Disease in the coats of the vessel, especially obliteration occasioned by the excessive growth of the lining membrane within the tube—a condition followed by atrophy of the vessel itself. The process may be seen on the inner surface of the aorta as laminæ of a delicate membrane, the growth of its inner coat. In smaller vessels the affected part contracts, until the passage is finally closed by the new growth. A clot or plug forms, limited by the movements of the collateral circulation. At last the artery shrivels and becomes atrophied throughout the extent of this coagulation and obstruction, becoming dilated above it. Another form of occlusion (in consequence of excessive deposition) occurs when fibrinous vegetations form on the inner surface of the vessel, which may be *cretified, ossified,* or otherwise rough; as in the smaller branches of the femoral arteries, in cases of senile gangrene. (3.) Coagulation of blood,—(*a.*) *from arteritis;* (*b.*) depending on an internal cause, such as is described by Velpeau, of closure of the aorta from the third lumbar vertebra downwards, with a part of the iliac artery, owing to coagulation of a cancerous character in a case of malignant disease. (4.) From persistent pressure, as may be exerted by different tumors, or by instruments. The obliteration, however, is very rarely complete, unless perfect coalescence of the opposed lining membrane takes place. The remainder of the occlusion is then completed by a plug reaching to the nearest branch, beyond which the vessel is finally obliterated, as in the case of tying of an artery. But the phenomena established by all of these lesions, alike in large and in small vessels, may be comprehended in the only two forms of occlusion recognised in the nomenclature of the College of Physicians, namely—(*a.*) from compression; (*b.*) from impaction of coagula. Under this latter two lesions are particularly referred to, namely:—(1.) *Thrombosis* (local coagulation), the formation of a clot *in situ.* (2.) *Embolism* (coagula conveyed from a distance). A knowledge of the nature of each of these lesions is necessary in order to appreciate the significance of the phenomena which are associated with each. There seems to be some ambiguity and uncertainty in the use of the words "*thrombosis*" and "*embolism*." The *phenomena of embolism* from the left side of the heart are apt to be expressed by gangrene of distant parts, as of the limbs, softening of the brain, or loss of vision. Gangrene, or decomposition of puriform matter, occurs most actively in the lungs, because here the process is brought most intimately in contact with the outer air; and here, therefore, the influence of impure air becomes so much more injurious; but cases of arterial embolism developed in this way (*i. e.*, primarily in the lungs) are not as yet known to be numerous. *The sources of embolism*, or of coagula conveyed from a distance, imply some knowledge of the circumstances under which the blood spontaneously coagulates *in situ*, or permits the separation of its fibrine to take place in the living vessels. When such separation takes place, the results are known as *ante-mortem* clots or *thrombi;* and the process of clot formation is named "*thrombosis*."

In a pathological point of view, the following events in their order may be regarded as the most important in bringing about *thrombosis*, or the formation of coagula within the circulating system:—(1.) *Local disturbance to the circulation;* (2.) *stoppage*, *stagnation*, *retardation*, *quiescence*, or *slowness* of the blood-stream, as in the case of an aneurism after ligature or compression of the artery; in the right side of the heart, when there is an impeded flow of blood through the lungs, or through the left side of the heart. The sources of local disturbance to the circulation are—(1.) Irritation of the outer as well as of the inner coats of the blood-vessels; (2.) changes which take place in them; (3.) the formation of a clot and increase of coagulating fibrine, corresponding to the site of irritation; (4.) changes take place in it which furnish ample materials for further mischief; or, (5.) organisation of the clot takes place, when it is converted into connective tissue, and generally remains harmless.

Arterial emboli, or coagula conveyed from a distance, are observed as a consequence of—(1.) Gangrenous phlebitis involving pulmonary veins or pulmonary tissue (*rare*), the débris passing into the left auricle; (2.) organic affections of the aortic or mitral valves on the left side of the heart, fibrinous concretions, and warty excrescences (*common*); (3.) atheromatous disease of the inner membrane of the artery. A first obstruction is generally followed by others, multiplicity and complexity of lesions being one of the characters of embolism. The most frequent sites of such arterial emboli are—(1.) Arteries in the fissure of Sylvius; (2.) internal carotids; (3.) arteries of the lower and upper extremities; (4.) splenic and renal arteries; (5.) external carotid and mesenteric.

The Symptoms, in sequence, characteristic of arterial embolism, are as follow:—(1.) There is evidence of valvular disease of the heart; and in the history of all such cases there is, (2.) after exertion, the occurrence of palpitation, with the cessation of which (3.) the pulse disappears in the affected artery below the site of the embolon or plug—*e. g.*, in the *radial*—if the plug is arrested, in the *brachial*, with pain in the hand of that side. These phenomena are due to the separation of fibrine from the warty growths on the valves. The particles, being carried along with the stream of blood, become fixed in the artery. Recovery may ensue for a time; but, (4.) repeated subsequent attacks occur, each associated with palpitation and irregular action of the heart; and with the cessation of the cardiac symptoms, simultaneous obstruction of peripheric arteries is found to have taken place. (5.) At last gangrene ensues in some parts, and death results. In the brain such infarction leads to yellow softening. In the spleen and kidney, the infarction takes the shape of a conical mass of material exactly limited, of a colour varying with the size of the lesion, and generally more dense than the surrounding parenchyma (*Med. Times and Gazette*, Vol. XVI., p, 273). The obstruction is at the point of narrowing of arterial branches (*capillary embolia* in tufts), immediately after bifurcation (*splenic penicilli*). (6.) Strong pulsation of vessel on cardiac side of occlusion (TUFNELL).

The origin of the clot in arteries, as to where it is formed, is not always easy to determine, except in the cases of valve vegetations. The coagula, which exist without simultaneous lesions of the arterial wall or adjoining capillary circulation, are never formed on the spot, but have been separated from some distant point in the circulation, and have been carried by the current of the blood as far as they could go. Such are genuine emboli. They are always found in places where a large arterial trunk, by giving off branches, suddenly acquires a more constricted calibre. Virchow, Paget, Malmsten, and Kirkes have shown independently of each other, that—(1.) Fibrinous concretions may separate from the valves of the heart during life; (2.) that they may cause obstructions of particular peripheric vessels; (3.) that they may have a poisonous decomposing influence similar to typhus or pyæmic poisons (*infective endocarditis*). In the lungs apoplectic foci become developed: infarctions occur, and their results, in broad masses of fibrine, remain: metastatic fibrinous wedges occur in the capillary vessels; and petechial spots occur on the skin and on mucous membrane, pericardium, and peritoneum. Similar fibrinous deposits accumulate in the spleen. (See also under *Phlebitis*, "Disease of the Veins.") Thus, on the one hand, *deposition* of morbid substances in various parts of the body distant from the heart is accounted for; and, on the other hand, results are explained on simple mechanical principles which hitherto have been obscure. Formerly, many of the cases thus capable of explanation would have been recorded as cases of "sudden death," or as "sudden retrocession of gout or rheumatism," or of "gout in the stomach," or "palsy of the heart."

These embolic processes and their consequences are of great interest, and have created a literature already extremely copious. The anatomical condition of embolism is now fully recognised; but the mechanical cause of the hæmorrhagic infarction of so much tissue is not so clear. The existence of free collateral circulation is a means of preventing the after-effects of an "*embolon*" or "*hæmorrhagic infarct.*" On the other hand, some believe that this same collateral circulation, leading to rupture of capillaries, is the cause of the infarction. Cohnheim's experiments (made on the frog's tongue) with pellets of black wax injected into the arteries, have shown,—(1.) That *instantaneously* the blood was driven with increased force through the branch immediately below, the increased rapidity being continued into its capillaries and returning vein. If the obstructed artery had a collateral anastomosis the pressure thrown upon the branch below it soon became equalised; but if the embolised artery had no anastomosis beyond the plug, the increased rapidity in the lower branch continued for some time, without leading to rupture and hæmorrhage, and became gradually less. In the obstructed artery the contents were found at rest for a distance on both sides of the embolon, on the cardiac side, as far as the next branch.

ANEURISM OF THE THORACIC AORTA.

Latin Eq., *Aneurysma;* French Eq., *Anévrisme;* German Eq., *Aneurysma* —Syn., *Schlagadergeschwulst;* Italian Eq., *Aneurisma.*

Definition.—*A spontaneous circumscribed partial dilatation of some portion of the aorta, consequent on lesion or degeneration of its walls.*

Pathology.—(*a.*) *Causation.*—Aneurisms of the thoracic aorta most frequently arise from the ascending portions of the arch, and seem to spring most frequently from those parts of the vessel against which the current of the blood is most forcibly directed. The effects produced vary with the seat and the size of the tumor; and aneurism is one of the most distressing and puzzling of thoracic diseases.

Several kinds of local lesion are recognised as giving rise to aortic aneurisms, namely:—(1.) *Chronic endoarteritis* resulting in *atheroma*, as already described, is the lesion which most frequently gives rise to aneurism, so that the morbid process which leads to aneurism is the same as that which leads to atheroma. (2.) Simple fatty degeneration, without any preliminary thickening or cell-growth in the tunics. There is opaque, white, or yellowish-white spots, slightly prominent above the surface, consisting of deposits of fat molecules in the tissue of the lining membrane of the arterial coats. (3.) Simple thinning of the aortic wall, not uncommon among elderly people (Niemeyer). (4.) Several forms of degeneration of blood-vessels, besides the fatty. In consequence of some one or other of these changes, the aorta loses its elasticity, so that one part gradually yields, and becomes dilated by the pressure of the blood. Then (*generally on the occurrence of some sudden strain*) the circular fibres of the arterial coat give way, leaving only the outer and inner coat, the dilatation of which then goes on more rapidly. This event is sometimes recognised and remembered by patients, as on the occasion of a sudden and violent muscular effort, lifting a heavy weight, and the like, when some sensation as of something having "cracked," "yielded," or "given way," is observed at the time. Hence the causes of thoracic aneurism are exceedingly obscure. There is reason for believing that the morbid conditions associated with *alcoholism, gout, rheumatism, syphilis*, are the circumstances under which aneurisms are most apt to be developed, the elasticity of the vessel being impaired by structural changes, the pathology of which has been described. With regard to the influence of syphilis, I may here observe that I have dissected (at Fort Pitt and at Netley Hospitals for invalids) bodies o numerous soldiers, in each of which a distinct history of syphilis wa present, associated with unmistakable syphilitic lesions. Of these by fa the greater number had the coats of the thoracic aorta impaired by characteristic changes—changes which are uncommon at so early a period of life as thirty-two years of age, which I have every reason to believe were due to syphilis—a *syphilitic endoarteritis.* The changes were obvious, from cicatricial-like loss of substance of the inner coats, small local dilatations of the artery, and in several cases aneurismal expansions, one as large as an orange, which proved fatal. Fifty per cent., at least, of these aortic aneurisms occurred in soldiers with syphilitic infection, and with no other ascertainable conditions present. A characteristic case of aneurism of the thoracic aorta resulting from syphilis is also recorded by Assistant-Surgeon Alfred Lewer, in the *Medical Report of the Army Medical Department* for 1862, p. 512.

Syphilis may not be so prominent a cause of aneurism of the aorta as the occasional occurrence of striking cases might lead us to believe; but in common with gout and rheumatism, it certainly tends to impair the nutrition and functional properties of the blood-vessels, especially affecting their outer coats and innermost connective tissue, so that any mechanical straining of the vessel finds out its weakest part, and readily sets up an *endoarteritis,* resulting in atheroma, or in aneurism, or in both. The records collected by Staff-Surgeon Dr. Peter Davidson, and subsequently by Dr. Welch, clearly show the influence of *atheroma* in the production of *aneurism.* (*Army Med. Dep. Report,* Vol. V., p. 481.) Men suffer from aortic aneurisms much more frequently than women; and the majority of aneurisms are found in persons who are called upon to make violent muscular efforts, especially under circumstances of restraint from clothing or carrying weights. It is, I think, undoubted that there is an excessive production of heart and blood-vessel disease in the army compared with civil life, and that the army excess is especially in aneurism—which Dr. Lawson finds to be eleven times more frequent among soldiers than among civilians (PARKES, *Hygiène,* p. 538). One condition—that of exertion, often rapid and long-continued—is constantly acting in the case of soldiers; and certain arms of the service show a greater loss of strength from diseases of the heart and blood-vessels than others. The following is the order in which the arms of the service appear to suffer most from these diseases, beginning with those who suffer the greatest:—*Military Train, Royal Artillery, Cavalry, Royal Engineers, Depot Brigade, R.A., Coast Brigade, R.A.* The exertions connected with the duties of all of these arms of the service are undertaken with a bad arrangement of dress and equipments. The cavalry and artillery men are very tightly clothed, and the men are overweighted, notwithstanding that the horse carries some of the burden (PARKES). The pernicious influence of exertion carried on under unfavourable conditions has been clearly shown in the experiments made by the late Dr. Parkes, or carried out by others at his suggestion. The influence of different kinds of knapsacks—*i.e.,* different kinds of constraint, has been clearly shown. "In the third Report of the Knapsack Committee are some experiments made by the surgeon of the Royal Marines at Gosport. Twelve men, with an average pulse of 82·6, after running 500 yards without knapsacks, had an average pulse of 114 (highest, 124; lowest, 104). After running 500 yards with the old knapsack, the average pulse was 148 (highest, 164; lowest, 132). All these experiments were made with comparatively light weights; but when the man is in full service order, the effect is much greater still, and the rapidity of the pulse continues for a long time after the work is done. With the knapsack alone the effect on the pulse is considerable. Thus, four strong soldiers carried the old regulation knapsack, service kit, greatcoat, and canteen, but no pouch, and no waist-belt (except on one man). The pulse (standing) before marching was on an average 88; after thirty-five minutes it had risen on an average to 105; after doubling 500 yards it had risen to 139; and in one of the men it was 164, irregular and unequal. After the double they were all unfit for further exertion. In a fifth man, who was not strong, the thirty-five minutes' marching raised the pulse to 120 from 94; after doubling 250 yards he stopped; the pulse then could absolutely not be felt. In another series the average pulse of four men, with the knapsack only, was 98 standing; after one hour's march, 112; after doubling 500 yards, 141" (PARKES' *Prac. Hygiène*, p. 540, fourth edition.)

Dr. Arthur B. R. Meyers, of the Coldstream Guards (in an essay which obtained the Alexander Prize in 1870, "On Diseases of the Heart among Soldiers") assigns the production of the majority of aneurisms of the aorta in the army to mechanical obstruction to the circulation when the soldier is undergoing exertion, caused by the general constriction of his neck and chest by faulty clothing and accoutrements. Their most frequent situation, as shown by Dr. Sibson, is in that portion of the aorta which is subjected to the greatest strain; and the statistics of the army corroborate the observation of Dr. Sibson as follows:—*Ascending aorta*, 37: *arch*, 38; *descending aorta*, 12; *thoracic aorta*, 7; *abdominal aorta*, 15 in 109 cases; or, ascending aorta and arch, 75; other thoracic aneurisms, 19 (MEYERS. See also THURNAM'S opinion, p. 636, *ante*.) The naval service suffers less than the army, probably because, however violent their great exertions may be when leaning bodily over the yards in reefing, hauling at ropes, and lifting heavy weights, there is no mechanical obstruction to their circulation by tight clothing (H. LEECH, *Path. Transac.*, 1865). The hereditary transmission of aneurism has been noticed by Dr. Fuller.

(*b.*) *Morbid Anatomy.*—The more important points in the morbid anatomy of aneurisms have reference to—(1.) Their form and size and relations; (2.) the constitution of their walls; (3.) the nature of their contents. Aneurisms are now classified as being—(*a.*) *Circumscribed*, which are dilatations of a short portion of artery, sometimes widened in all directions, the tumor involving the entire diameter. More frequently one side only is dilated, and the aneurism assumes the appearance of a tumor situated on the side of the vessel—mere globose or fusiform dilatations of limited length. Such circumscribed aneurismal tumors may have, again, secondary sac-like pouches, or dilatations of their walls (*saccular aneurisms*). At first the tumor generally corresponds to the character of a *true aneurism of Scarpa*, consisting of all the arterial coats; but when the sac has attained some magnitude, the inner tunic only extends for a short distance into it. When still more fully developed, the middle tunic dwindles away, and totally disappears, with traces of degeneration here and there of the inner coat (NIEMEYER). In the majority the tunics are morbid. The lining membrane may be traced, thickened and pulpy, over the lips of the aneurismal orifice, and with more or less distinctness over the whole inner surface of the aneurism. The external coat may be traced over the whole tumor. It is the middle coat which is usually attenuated, or absent in patches. The coats may be the seat of fatty or calcareous changes. The progressive enlargement of the cavity is the result of continued blood pressure. This force, exerted on a given aneurismal area, is exactly equal to the force of blood-pressure exerted on an equal area of the artery in its neighbourhood. In other words, the total pressure on the inner surface of an aneurism is in exact proportion to the superficial extent of that surface, and has no relation to the size of orifice or form of the aneurism, in accordance with a well-known hydrostatic law. It follows, also, that the larger an aneurism grows, the less capable its walls become of successfully opposing the blood pressure within, unless they undergo some compensative increase of strength, which is a very unusual occurrence. (*b.*) *Diffuse aneurisms* involve a considerable extent of the entire calibre of the vessel, and if the dilatation ends abruptly, it is a *cylindrical* aneurism, and if gradually, it is a *fusiform* aneurism. Such are usually met with in the ascending and transverse portions of the arch of the aorta. On the walls of such arteries circumscribed pouches often form (NIEMEYER).

The College of Physicians require the following forms to be distinguished—(*a.*) *Fusiform;* (*b.*) *saccular;* (*c.*) *diffuse,—i.e.,* where the sac is formed by the surrounding tissues.

The size of aortic aneurism varies from a pea or a small walnut, as within the pericardium (where they rarely attain any very great magnitude before giving way), to growths of very large size. Generally, outside the pericardium they may attain a very large size, such as of a cocoa-nut or child's head, pushing aside parts, and projecting in large tumors from the chest, destroying the sternum, ribs, and vertebræ by their constant and increasing pressure. From the ascending aorta aneurisms usually project towards the right half of the sternum, and become visible in the region of the upper ribs and costal cartilages of the right side. In the majority of cases they break into the right pleural sac, or burst externally. They are most frequent on the ascending portion of the aorta, before the origin of the innominate artery, and more common on the convex than the concave side.

Symptoms.—Aneurisms opening upon mucous surfaces, especially upon the air-passages, are generally attended with small and irregularly-repeated hæmorrhages. The persistence of these trifling amounts of blood in the expectoration justifies suspicion of aneurism in the absence of any other circumstances to account for the presence of blood. *Tumors,* such as aneurisms, or mediastinal tumors, often give rise to symptoms suggestive of laryngeal disease; therefore, in all cases the larynx ought to be examined on the one hand by the laryngoscope, and on the other a physical examination of the chest should be made for the signs of an aneurism or a tumor. The frightful and agonising dyspnœa is generally due, in cases of aneurism, to implication of the recurrent laryngeal nerve, and not to ulceration or disease of the larynx. Generally, it may be stated that the symptoms are the result—(1.) Of crowding together and compression of the organs within the thorax, caused by the growth of the aneurism; and, (2.) of obstruction to the circulation. Aneurism of the arch, pressing on the trachea, causes the most intense dyspnœa, accompanied by a whistling sound on breathing and coughing. The dyspnœa becomes spasmodic, asthmatic, and laryngeal, in paroxysms, if the pneumogastric or recurrent laryngeal nerves be stretched or irritated. The dyspnœa may have a spasmodic asthmatic character, without any tendency to laryngeal spasm, associated with the persistence of small hæmorrhagic expectorations. In such cases the aneurismal tumor presses upon the bronchi towards the roots of the lungs, evidence of valvular disease and of pneumonia being absent. The suffering may be paroxysmal, in the form of *angina pectoris*, due to interference by an aneurism with the thoracic nerves, or with the circulation in the heart itself. The variable character of the hæmorrhage, and the remarkable intermissions of such hæmorrhages from aneurismal sacs, are symptoms of thoracic aneurism which only began to be appreciated in their proper light since the case of Mr. Liston drew the attention of the profession to them. *Aneurismal hæmoptysis* may occur in one of the three following forms:—(1.) A frothy bronchitic sputum, streaked with blood; (2.) a rusty sputum, very like pneumonia, but usually more abundant, more frothy, and less viscid; (3.) a deeply-dyed purple or brownish-purple sputum, like the so-called "prune-juice" expectoration characteristic of the third stage of pneumonia, and of certain forms of pulmonary hæmorrhagic condensation from valvular disease of the heart; (4.) any of the preceding forms of hæmorrhage, alternating with small discharges of pure, unmixed, but generally imperfectly coagulated blood. The bronchitic varieties of sputum, either stained or streaked in different proportions with blood, occur chiefly in tumors pressing on the trachea and larger bronchi, and not producing consolidation of any part of the lung; while the "prune-juice" sputum occurs when the lung is directly involved in the tumor (W. T. GAIRDNER'S *Clinical Medicine*, p. 454, *et seq.*). Permanent contraction of the pupil on the affected side is, in some cases, another sign of aneurism (REID, GAIRDNER, OGLE). The *correlation of symptoms*, as possibly indicating thoracic aneurism, is the main point for attention; and in addition to those noticed, raucous voice and aphonia are sometimes found. Undue pulsation, dyspnœa at intervals, oppression at the præcordial region, with difficulty of swallowing solid food, are also, in combination, significant of aneurism. But the main combination of symptoms are—(1.) *Compression of parts*, as of the right auricle, vena cava, or innominate vein, leading to superficial venous congestion and œdema of the upper half of the body, with headache, dizziness, and buzzing in the ears, and even fits of unconsciousness. If the intercostal nerves and brachial plexus are subjected to compression, violent pains, generally paroxysmal, are experienced in the right side of the chest, the arm-pit, and right arm. Compression of the *arteria innominata* of the left subclavian may render the radial pulse extremely small or quite imperceptible; and an appreciable inequality between the pulsations at the wrist of either side may also be a result of distortion of the arterial mouths, or of their stoppage by clots (NIEMEYER). (2.) *Retarded circulation*, added to the phenomena already mentioned, is of great significance. The strongest evidence of this kind consists in the *pause, often so distinctly perceptible, which occurs between the beat of the heart and the wave of the arterial pulse*, AT A POINT BELOW THE ANEURISM, most striking when the aneurism is situated between the points of origin of the great vessels of the arch. The pulse is then felt *later* at one wrist than the other. The sphygmograph shows that with aneurism modifications of the pulse occur both in its form and force. In the pulse-trace collected from an artery below an aneurism, the movement of the blood in the vessel approaches that which is normally seen in the smaller arterial branches. The vertical line of ascent disappears, and often approaches in length that of descent. Thus, a more feeble pulsation is given to the finger; and, as the summit is slow to occur, there is a sensation of apparent retardation felt in addition. Figures 50 and 51 represent the modifying influence of an elastic aneurismal sac on the pulse-trace of the left radial artery (Dr. B. W. FOSTER). There was an aneurism of the left subclavian artery within the thorax; and the right pulse shows the trace of another lesion—namely, insufficiency of the aortic valves. So characteristic of the conditions in this case were the above traces, that they alone enabled Dr. Foster to arrive at the diagnosis to which ordinary means had led those watching the case. At the time when the above traces were taken, the tumor was evidently large and very elastic, hence the great modifications in the pulse-form produced. In *aneurisms of the aorta*, very much less striking indications are afforded by the pulse-trace. Nevertheless, Dr. Foster's experience leads him to believe that the position of a thoracic aneurism may be indicated by the pulse changes—*e.g.*, when a small aneurism or even a considerable dilatation of the ascending aorta exists, the pulse of the right radial is generally reduced in size when compared with the left (Dr. FOSTER'S *MS. Notes*). The indications pointed out by Marey are—(1.) Modifications in the force of the pulse. It is seldom much diminished—a character of small value in diagnosis. (2.) The modification in the intensity of the dicrotism may exist in one or both radial pulses, and is occasionally a sign of much value. In aneurisms of the descending thoracic aorta the dicrotism proper is much increased in both pulses, but especially in the right, while the left radial is smaller than the right (Dr. B. FOSTER). (3.) The presence of a constant dissimilarity in the pulse-traces of the radial arteries is the most valuable sign. In many cases the finger can perceive a want of parallelisms in the beats of the radials, but often this is too slight to be detected by the finger. It sometimes shows itself in the trace by a slight difference in the dicrotism only; at others, the difference in form is more evident. When the tumor

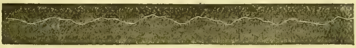

Fig. 50.*

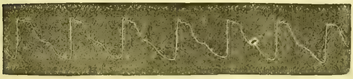

Fig. 51.†

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* Left radial. † Right radial.

is so situated that it can be handled, we can gain valuable evidence as to its nature by observing the changes in the tension of the arteries produced by its alternate compression and relaxation (Dr. B. FOSTER).

Diagnosis.—Tumors (*cancers*), abscesses (mediastinal or others) are the most common lesions with which aneurism may be confounded. Other lesions are, pulsating empyema, cardiac disease, phthisical consolidation.

Prognosis.—Aortic aneurisms are very rarely recovered from. But life may be prolonged if the disease is recognised early and judiciously managed. Death usually takes place by gradual asthenia, or from the effects of pressure, if the aneurism does not rupture.

Treatment.—Local bleeding is useful when there are pain and tenderness over the aneurismal sac. General blood-letting may be useful if the circulation is excited, and the patient be of full habit, but not on the principle advocated by or ascribed to Valsalva. Of all remedies *digitalis, aconite, belladonna*, and *veratrin* are the most useful in tranquillising and regulating the action of the heart. The deposition of fibrine from the blood is more prone to take place when the circulation is "slowed." It is the principle of treatment in the cure of aneurisms by pressure. The current of blood is not entirely stopped, but is rendered more slow, so as to have an amount of stagnation of blood in the sac favouring the separation of fibrine and its coagulation. A diminution of from *ten* to *fifteen* pulsations of the heart in the minute will greatly tend to the filling of the sac with coagula (FULLER). The medicinal agents which tend to promote this result are, *ergotine, gallic* or *tannic acids, tincture of steel, acetate of lead*, and *iodide of potassium.* Mr. Jolifie Tufnell, of Dublin, has advocated the treatment of aneurisms of the thoracic and abdominal aorta on the principle here enunciated—namely, that of "slowing" the circulation. It consists of restricted diet and perfect rest in the horizontal position, for periods varying from eight to thirteen weeks, combined with the employment of such remedies as may be necessary for special ends. The horizontal posture must be *strictly and absolutely maintained*, in a light and cheerful airy room, into which the sun shines, and from which the patient may be able to have as cheerful a view as possible out of the window. The diet must be confined to *three* meals, served at regular intervals, and restricted to the following in kind and in amount:—*Breakfast*—Two ounces of white bread and butter, with two ounces of milk or cocoa. *Dinner*—Three ounces of broiled or boiled meat, with three ounces of potatoes or bread, and four ounces of water or light red wine. *Supper*—Two ounces of bread and butter, and two ounces of milk or tea. These diets should make, in the aggregate, *ten ounces of solid and eight ounces of flui in the twenty-four hours, and no more.* The object of the special diet is t maintain life on as little food as possible, without inducing restlessness, as in some irritable constitutions, but if such restlessness should occur, a little more food may now and then be allowed. *Anodynes, aperients, narcotics sedatives*, and *tonics* are also useful aids in the management of the case Of anodynes, *lactucarium* is the most valuable, given in the form of a pill, b itself or combined with *humulin* and *hyoscyamus* (*Med. Rep. Army Med. Dep* 1862, p. 472). The patient must avoid everything which tends to increas the action of the heart. Moderate living, without the plethora of exces but with sufficient nutrition to maintain the circulation at a *uniform* flow, the point to aim at. Fatal results may speedily follow any marked change of diet and regimen (COPLAND). Pain and depression will generally be subdued by the hypodermic injection of *morphia,* commencing with one-fourth of a grain. Cough is to be relieved by sedatives and expectorants. Dyspnœa may require *tracheotomy.* Dropsy may be lessened by *mercury, digitalis, squills, juniper,* and *decoction of broom-tops.*

Since 1861, the following three special modes of treating aneurisms have been proposed, besides Mr. Tufnell's dietetic method:—(1.) The introduction of a quantity of fine iron wire into the aneurismal cavity, with the intention of supplying an extensive surface over which fibrine may coagulate. The late Dr. Murchison and Mr. Charles H. Moore practised this method in a case of saccular aneurism of the ascending aorta projecting through the anterior wall of the left side of the chest. Twenty-six yards of wire were passed through a small canula inserted into the tumor when it was evident that the patient could not live many days. With some modifications it was shown that the experiment might be justifiable in some cases of sacculated aneurisms only (*Med.-Chir. Trans.,* Vol. XLVII., p. 129, London, 1864). (2.) The method of RAPID *pressure treatment,* as practised by Dr. William Murray, of Newcastle-upon-Tyne, and Lecturer on Physiology in the University of Durham. It has been applied to aneurisms of the abdominal aorta. The cure by this method takes place rather by the coagulation of blood in the sac from sudden cessation of the current, than to any deposit of fibrine. The patient requires to be completely under the influence of chloroform, so that he may suffer the application of a powerful pressing instrument on sedative parts, and so as to restrain all muscular movement. All movement of the blood in the sac must be completely arrested, and maintained in a motionless state, as in the application of a ligature to the vessel for aneurism. The duration of the treatment and maintenance of pressure can only be measured by the result as to the time when pulsation ceases in the tumor. The first condition of success is, that *complete arrest* of circulation in the tumor must be *steadily* maintained. *Irregular* pressure for ten hours has been known to fail. Consolidation has occurred in twenty minutes in a case of Dr. Heath's, of Sunderland, when complete and steady pressure was maintained, having failed in a first trial with irregular pressure. (3.) Dr. Roberts, of Manchester, and Dr. G. W. Balfour, of Edinburgh, have each collected cases, some of them under their own care, in which the treatment of aneurism of the aorta by *iodide* of *potassium* was persistently carried out with remarkable beneficial results. The dose varied from *five, seven, ten, fifteen,* and *twenty* to *thirty grains* three times a day; and the relief of pain, one of the earliest symptoms of amendment, does not follow till an efficient dose has been taken. It is better to begin with twenty or thirty grain doses at once, and intermit or suspend them for a day or two, if circumstances indicate the necessity of such a step. The object is to saturate the system with the drug as speedily as possible. It is believed to owe its curative agency to its power of increasing the coagulability of the blood. A few weeks may be sufficient to bring about the curative result; but Dr. Balfour's experience is, that any considerable amendment can only be procured by keeping the patient for many months, perhaps twelve or more, persistently saturated with the drug. (BALFOUR, *Edin. Med. Journ.*, July, 1868, p. 33; CHUCKERBUTTY, in *Brit. Med. Journ.*, July 19 and 26, 1862; ROBERTS, *Brit. Med. Journ.*, January, 1863). The strict enforcement of the recumbent position is an adjuvant of paramount necessity in the treatment of all aneurisms. Tracheotomy may prolong life in some cases where *stridor* exists, if the laryngeal symptoms are the source of immediate danger (W. T. GAIRDNER).

INFLAMMATION, ANEURISM, DILATATION, AND MORBID GROWTHS OF THE PULMONARY ARTERY.

Pathology.—Any reference to diseases of the pulmonary artery, as distinct from diseases of *arteries* and *veins*, has not been made by the College of Physicians; yet, inasmuch as this artery is an anomalous vessel, whose coats are constructed like those of the arteries in general, while they are pliable and extensible, like the texture of veins; and, as the lesions are important, in connection with the pathology of pulmonary consumption and hæmoptysis, diseases of the pulmonary artery are worthy of notice here. It is a short wide vessel for about two inches of its course, when it divides into its right and left branches, which accompany the bronchial tubes into the substance of the lungs, and, ramifying *without anastomoses*, at length terminate upon the walls of the air-cells and on those of the bronchia in a fine and dense capillary network, from which the radicles of the pulmonary veins arise. It conveys dark-coloured venous blood from the right side of the heart to the lungs.

(*a.*) *Morbid Anatomy.*—(1.) *Deposits of blood or lymph* in the pulmonary artery and its branches occur as a result of inflammation of veins and venous embolism, and will be considered under diseases of the veins. (2.) *Acute inflammation* is rare; but *atheroma*, as a result of chronic inflammation, is not uncommon in cases of deficiency of the tricuspid valve, with consecutive hypertrophy of the right ventricle, the atheromatous products leading to hæmorrhagic infarction in the lungs (DITTRICH). (3.) *Acute inflammation* of the pulmonary artery has been described by Dr. Norman Chevers as occurring under the following circumstances:—(*a.*) As a consequence of inflammation of the veins. (*b.*) In cases of Bright's disease, and especially in persons habitually intemperate. (*c.*) In rheumatic patients, as a result of exposure to cold. (*d.*) In connection with certain forms of pneumonia. (4.) *Aneurisms of the pulmonary artery* are described as exceedingly rare, and never attain any considerable size. Skoda reports a case where the aneurism was as large as a goose's egg, associated with cyanosis and dropsy (NIEMEYER). (5.) *Aneurisms in branches of the pulmonary artery* occupying phthisical cavities in the lungs are by no means uncommon; and it seems probable (though comparatively few cases have been recorded), that aneurismal dilatations of branches of the pulmonary artery within vomicæ may be more common than is generally supposed and would be discovered if carefully sought for, particularly in cases o fatal hæmoptysis (CHRISTOPHER HEATH). In the *Lancet* for 1841, a aneursim of this kind is described by Mr. Fearn, of Derby, in a man fort one years of age, proving fatal after several attacks of hæmoptysis. Sim lar cases are also recorded by Dr. Cotton, in *Med. Times and Gazette* f

Jan. 13 and Oct. 20, 1866, and *British Med. Journal*, Oct. 24, 1868; by Dr. Quain, in *Path. Soc. Transactions*, Vols. XVII. and XVIII.; and Dr. Moxon, in Vol. XVIII., p. 55, the third case he has met with, and reported in the *post-mortem* records of Guy's Hospital. In all his cases there was a history of hæmoptysis, and one of the patients died suddenly by the rupture of the aneurism. Dr. Vald. Rasmussen, of Copenhagen, also reports, in his excellent paper on "Hæmoptysis," translated by Dr. Daniel Moore, of Dublin (*Edin. Med. Journal*, 1868, Vol. I., p. 389), that hæmorrhages from a cavity are due to ruptures of small sac-like aneurisms, developed on branches of the pulmonary artery running in the walls of cavities. The size of these aneurisms varied from that of a walnut to that of a pea, and were formed by the dilatation of a vessel in contact with the inner wall of the cavity, the part of the vascular wall touching the cavity at the point of contact being dilated, while the remainder lay imbedded in the condensed vascular wall. The form is generally sacculated, with a tolerably uniform transition between the walls of the aneurism and those of the vessel, without any proper neck. The surface is smooth, and in its cavity most frequently only freshly coagulated blood is found, and only in one instance fine decolourised adherent coagula. The walls are of various thickness, often two or three times thicker than the vascular wall from which they proceed; and when the vessels are small from which they grow, the aneurisms form only a slight dome-like dilatation. When large, the walls are thin and particularly attenuated up to the point where rupture has taken place. Degeneration of the walls of the aneurism has also been met with, as separate, or confluent, and yellow sharply defined points. The rupture takes place always at the most prominent point of the sac; and there is usually found an irregular fissure-like rent, rarely exceeding in width two or three millimètres; most frequently it is only large enough to allow the knob of an ordinary probe to pass. In the opening, loose dark coagula adhere, more rarely firm and somewhat decolourised. The edges are attenuated and yellow, showing distinct fatty degeneration. The number of the aneurisms vary. In general one only is met with. In one case two were found close to each other. In another there were as many as four, two and two, close to each other on the same branch. The vessels on which these aneurisms had formed were on an average from one to three millimètres in width. The development of these aneurisms stands in a definite causal relation to the formation of the cavity in the lung—want of support being one of the chief elements favouring the growth of the aneurism, combined with increased intravascular pressure, which during a fit of coughing is very considerable, still more increased by condensed pulmonary tissue and obliteration of branches beyond the aneurism. One form of pulmonary phthisis may originate in disease of the pulmonary artery. A section made through the centre of a tubercle-mass, inclosed a little stem, knuckle, or portion of a minute pulmonary artery, about one-twentieth of a line in size. This knuckle or loop of artery was surrounded by tubercle. The walls soften by the new formation of cells; and the more the cells grow, the larger the tubercle-mass becomes, till it is finally lost in the larger knots (DICHLER). These formations are due to a diseased state of the stems of the blood-vessels; evidence of which may be found in cases of *ectasis*, or aneurisma distension of the walls of the pulmonary capillaries, as seen in the air-cells. Buhl and Zenker have described such cases (VIRCHOW'S *Archiv.*, 1862, p. 183). Bearing upon this point, and upon aneurism of the pulmonary artery, I dissected (1861) a soldier who died suddenly by hæmorrhage from the lungs. On opening into one of the cavities, it was found filled with coagulated blood; and projecting from a spot on the wall was a round tumor about the size of a walnut. This tumor had ruptured, and the rupture held a coagulum of blood. It was found to be an aneurism of the pulmonary artery; and several other tumors of a similar nature, but of variable and much smaller size, existed in other cavities in the same lung, projecting from the pulmonary artery. They were proved to be continuous with this vessel—(1.) By the injection of spirits into them, and their distension thereby through the pulmonary artery as it left the heart; and, (2.) By microscopic examination, which showed a delicate epithelial lining to these tumors, continuous and similar to that in the artery. The preparation is preserved in the Museum of the Army Medical Department at Netley. (6.) *Diffused dilatation of the pulmonary artery* occurs with great frequency in cases which cause hypertrophy and dilatation of the right heart. An unusually large pulmonary artery, which had four valves, is described by Dr. Theophilus Thomson, in *Med.-Chir. Transactions,* Vol. XXV., p. 247, London, 1842; and other morbid conditions, such as *ulceration* from pressure of aortic aneurism, may also be seen.

(*b.*) *Diseases of the Veins.*

PHLEBITIS.

LATIN EQ., *Phlebitis;* FRENCH EQ., *Phlebite;* GERMAN EQ., *Phlebitis*—Syn., *Venenentzündung;* ITALIAN EQ., *Flebitide.*

Definition.—*Changes in the texture of a vein, and local coagulation of blood within the diseased vessel (thrombosis), are the essential characters of phlebitis, often leading to the subsequent phenomena of pulmonary and hepatic embolism.*

Pathology.—(*a.*) *Causation and Phenomena.*—The varieties of *phlebitis* are described as, (a.) *Adhesive;* (b.) *Suppurative.* The first set of phenomena to be considered are those which accompany the early stage of inflammation of the coats of a vein, because they mostly concern the physician in connection with the important and dangerous phenomena of venous *embolism.* For other aspects of inflammations of the veins in the varieties of phlebitis, the student is referred to his text-books on surgery. So long as pus-corpuscles, as such, were looked upon as the noxious material which poisoned the blood in cases of malignant or infective phlebitis (see Vol. I., page 796), it was supposed that the tissue of a vein being inflamed, pus would be secreted from its inner wall, just as from a serous membrane. John Hunter has the credit of having suggested this; but he did s merely *as a query,* and subsequent writers adopted the suggestion withou further examination and without evidence, (see ARNOTT "On the Effect of Inflammation on Veins," in Vol. XV. of *Med.-Chir. Trans.*) Veins ar exceedingly slow to inflame. When they do, the inflammation-change begin in the connective-tissue, towards the outer parts of the vessel, and this even when irritant bodies are introduced into the cavity of the vein itself, (see LEE'S "Essay," in Vol. XXV. of *Med.-Chir. Trans.*) Mr. Lee introduced cotton-wool into a portion of vein emptied of blood. The lining membrane remained unchanged, and the lesions commenced outside the vessel. There the pus formed, and thence the inflammation spread by the connective tissue, and simply by continuity. The generation of secondary multiple abscesses is attended by quite a different process. It was first shown by Cruveilhier that in the (so-called) inflammation of veins a clot of fibrine forms, and is always present, independent of any lesion in the contiguous vascular wall; and so he passed to the extreme belief that all inflammation essentially consisted in coagulation of blood in capillary vessels. But one important fact has now been quite substantiated—namely, that long before any visible effects of inflammation occur in the lining membrane of a vein, a clot of fibrine is formed at the part, and in this clot fluid comes to be produced, in all external appearances resembling pus. It must also be remembered that blood, when arrested in the vessels or extravasated out of them, coagulates at the ordinary temperature of the body—98° Fahr. (*Phil. Trans.* Vol. LXXXVI.)—a most valuable event in the cure of aneurisms. From this observation as a starting-point, Virchow has developed his beautiful explanation of the various phenomena connected with phlebitis. Inflammation of veins by continuity, or continuous inflammation, as the cause of secondary phenomena and of multiple abscesses, is to be rejected; but "coagulation of the blood in the living vessels"—the formation of a clot or "thrombus" (*thrombosis*)—are the phenomena which attend the formation of multiple abscesses. The impulsion or projection onwards of a coagulum-clot, or thrombus, or substance detached from the walls or valves of the vascular system, and its subsequent arrest in the course of the circulation, give rise to the phenomena described under the name of *embolism—venous embolism* in this instance. The coagula may travel in particles or larger masses from the veins to the heart, and thence to the pulmonary artery and peripheric vessels of the lungs. All the cases, fully recorded, which illustrate the phenomena of multiple abscesses from venous embolism that I have ever examined, have shown that the affection essentially begins by a real coagulation of the blood at *some definite fixed point;* and this is the most obscure part, and the most difficult to discover, in the history of cases of multiple abscesses from venous embolism. But where this beginning is traceable, the history points to some sources of *local irritation,* which, by simple disturbance of the flow of blood, determines in some way its coagulation in the living vessels. The experiments of Professor Lister throw much light upon this subject ("Croonian Lectures," *Lancet,* August 8, 1863). Any occurrence which sufficiently interferes with, lowers, or disturbs the vitality of the structures inclosing blood, affects its fluidity within the inclosing vessels, and tends to permit its spontaneous coagulation *in situ.* Very simple ailments sometimes illustrate this point by morbid anatomy. Chilblains, for example, have been the starting-point (M. RIBES). Clots form in the veins, and proceed upwards even to the superior *vena cava,* into the right auricle and ventricle. In one of the most striking cases of this description, "a venous clot of fibrine, twenty inches long, was found in the right auricle and ventricle. It showed such marks on its surface as clearly demonstrated its formation in a vein; and other circumstances pointed to the chief vein of the thigh as the site of the primary formation of this *clot* or *thrombus*. Œdema of the limb prevailed, and the disappearance of the œdema was associated with those sudden cardiac and other symptoms which indicated the passage upwards of this *coagulum* or *thrombus* to the heart, where it was found coiled up in the right auricle and ventricle" (Druitt, *Med. Times and Gazette*, July 19, 1862). In cases of fracture of bone, of amputations, of enlarged glands, of ulcer on the foot (imperfectly healed by scabbing), of open wounds, gunshot passing near or between vessels, we have great and many sources of irritation, leading to inflammation of a vein at a spot, or the radicles of veins, and the accompanying *thrombosis*. The contiguity of these must be to more or less large veins, or to smaller vessels in or about these sources of irritation and disturbance. In no case, therefore, of swelled legs, enlarged glands, subacute inflammation or hardening over the course of lymphatics or superficial veins, should th possibility of the mortal accident of *embolism* towards the right side of th heart be overlooked; or the possible supervention of pyæmia. The nex set of phenomena characteristic of phlebitic embolism with the formatio of multiple abscesses, are those connected with the softening, disintegratio and breaking up of these thrombi or clots. Virchow was the first demonstrate the results that ensue. He showed (1847) the embo characters of certain products previously thought to be inflammatory their origin (*e.g.*, white fibrine-like masses in the spleen, &c.); and arrived at the following conclusions:—(1.) The occurrence of fibrin plugs or clots in the pulmonary artery long before death is alw secondary, where obstruction is independent of pneumonia or ot changes in the parenchyma. They are apt to arise in any part of venous vascular system anterior to the lungs in the course of the circ tion—*e.g.*, in the veins of the limbs, especially when varicose and inflan in the pelvic veins, as after the operation of ligature for internal p whence the clots are carried by the current of the blood to the right of the heart, and thence into the pulmonary artery, and so to the l Experiments on animals support such views. The pith of the elder as well as animal substances, have been introduced into the veins, prod violent pneumonias, commencing with inflammatory hyperæmia. localised pneumonias extending, led to rapid deposition in the air-c fibrinous exudations, which became purulent, or the portion of gangrenous. As these changes advanced in the lung, pleurisy soon was developed at the periphery—at first producing fibrinous lable exudation slowly over the affected portion of the lung; but r as it progressed towards the other side of the chest, inducing hæmorrhagic exudation into both serous cavities. The pleur became gangrenous, and finally gave way to pneumothorax. severe lesions may be completed in so short a time as five days viously, however, to these important demonstrations of Virchow had appeared the observations of Sir James Paget on the obst of the branches of the *pulmonary artery*, and the sudden mode c to which they gave rise (*Med.-Chir. Trans.* for 1844). Cru n, and Dubini had also recorded similar cases; but the importance 'irchow's observations consisted in demonstrating the transportation ots or plugs of fibrine or of blood from different parts of the venous ɔls to the heart. Such plugs are known also to be sometimes arrested ıe liver, giving rise to so-called metastatic abscess; and while those s which find their way to the lungs have their origin in any part of the us periphery, those which find their way to the liver originate either e *portal venous system*, or in those veins round the *rectum, prostate, vagina, erus,* which (communicating alike with the *systemic veins* and with the *ior mesenteric veins of the portal system*) may induce multiple centres of nmation, and abscesses in the liver as well as in the lung (*Med. Times,* ary, 1862). Simultaneously with Virchow's, or a little after his :vations, we have the observations of the late Dr. Kirkes, on "The chment of Fibrinous Deposits from the Walls of the Heart" (*Med.-Chir. s.*, Vol. XXXV., for 1852). Two most instructive cases of this kind been recently related by Dr. Goodfellow, in which extensive arterial :uction, gangrene of the lower limbs, and death, followed the separation rdiac vegetations, (*Med.-Chir. Trans.*, Vol. XLV.)

ıus it has been gradually proved, that as clots occur in the veins, so also may form in or find their way into the heart's cavities. The ection is thus established between *peripheral clots* and *cardiac clots*, and far the softening of these clots or *thrombi* may give rise to the lesions scriminately described as *pyæmia* or *phlebitis*, but which are really part ıe phenomena of *embolism*. Clots in peripheral veins, however small, hus the sources of great danger. As a rule, they lead to secondary multiple deposits and abscesses in the lungs; and it is chiefly rences in the size of the capillary vessels which determine their ıate locality, where they act as any foreign body would. The débris ots, and large cell-elements from clots, in the mesenteric veins, and ulcers of intestines, passing through the liver capillaries and proing to the lungs, where they are arrested, illustrate this. The lungs the smallest capillaries of all. They average from $\frac{1}{3000}$ to $\frac{1}{2000}$ of a (scarcely sufficient to let pass a white cell of blood or of pus, which n average measures $\frac{1}{200}$ of a line), whereas the liver capillaries have ıch larger range—namely, from $\frac{1}{5000}$ to $\frac{1}{100}$ of a line.

he most common cases of *thrombosis* in veins are to be seen in bed-en dropsical persons, whose veins in connection with the buttocks, as those of the thighs and genitals, contain *ante-mortem* clots, proy acknowledging the source of the irritation giving rise to them as to the pressure of the weight of the body on the radicles of these s, and the gravitation of the blood against them (MOXON) as in cases of *beri* and *phlegmasia dolens*. The coagula found in the veins are thus products of local stasis, often caused by roughness of their inner ıce, by alteration of structure, and relaxation of elementary parts. ɜn the coagulum adheres only to one wall, with the effect of narrowing passage, such a thrombus is never of an embolic nature. It is a clot, *mbus,* plug, or coagulum of fibrine formed or forming there as a result ɔcal causes of irritation, the evidence of its local origin being present he bands of fibrine fixing it. The softening of *venous thrombi* is a gerous process. Wherever it is in contact with the free current of the blood, parts of it may be loosened and conveyed onwards; and accordin to whence they come such fragments may reach—(*a.*) The lungs throug the *right heart*, from the radicles of vena cava or from the systemic veins (*b.*) various organs of the body through the *left heart*, from the radicle of the pulmonary veins; (*c.*) *the liver*, from the radicles of the porta veins.

Such are some of the causes which may determine the formation c clots, either in large or in small vessels; and after Cruveilhier's observations, the next link in the chain of evidence regarding the nature c *metastical dyscrasiæ* was established by an arduous worker (then in th Army Medical Department, and since a distinguished Professor in th Royal College of Surgeons of England)—namely, Mr. Gulliver, formerl Surgeon of the Guards. He showed that the puriform mass in th interior of clots does not originate in the wall of the vessel or clot, bu is produced by transformation of the central layers of the clot—a transformation which may be imitated, as he did, by a chemical process. Si James M'Grigor communicated Mr. Gulliver's observations to the *Medica Chirurgical Transactions.* Mr. Gulliver examined the clots microscopically and found that the fluid was not pus; and to show that his observatio was intimately connected with the observation of Cruveilhier, I found (b accident) that a remark on this point, in Mr. Gulliver's handwriting exists on one of Cruveilhier's drawings, contained in the Library of th Army Medical Department at Netley. This is the point from whic Virchow starts in his interesting account of this subject—namely, th character of the contents of these clots—a puriform, but not a purulen substance (as Gulliver first showed), composed of granules chiefly. Th question, then, immediately suggests itself, "What becomes of them? Secondary disturbances—not so much by the softened mass as by th detachment of larger pieces, and just so large as to be arrested in vessel more or less remote from the seat of the original clot—are known t occur; and on the advance of clots and their débris from small int large vessels, bits break off and flow on into the stream of blood.

Symptoms.—When coagula arrive in the right side of the heart, the are attended with symptoms of exhaustion, pulse small and intermitting followed by collapse and powerlessness of muscle. The patient, althoug inhaling deeply, seems to suffer from apnœa; the veins become highl turgid; and sudden death occurs if large concretions separate an suddenly obstruct the pulmonary artery, as in the case recorded b Druitt, and noticed at p. 666 (see also cases recorded by Dr. GEORG JOHNSON in *Brit. Med. Journ.*, Nov. 30, 1872). The symptoms were,— hurried and anxious breathing; pulse rapid and scarcely perceptible features intensely pale, bluish, and distressed; the whole surface of th body cold, but drenched in perspiration; no pain, but great agitation an feebleness; the air entered the air-cells freely, but the beating of the hear was a confused and feeble "*wobble*"—its rhythm and force were gon The intellect was clear.

The *Phenomena of Pulmonary Arterial Embolism* are, (A.) *Parenchymatous.*— (*a.*) Collapse of lung; (*b.*) peripheral pleuritis; (*c.*) hæmorrhagic effusion (*d.*) capillary bronchitis, with cough and expectoration if the embolis is capillary. (B.) *Functional.*—(*e.*) A craving for air (*anxietas*); althoug eep breath may be drawn without pain, yet every movement of the ly tends to increase the anxietas; (*f.*) lowering of temperature; (*g.*) tolic murmur; (*h.*) increased impulse of the right side of the heart; irregularity of rhythm; (*k.*) undulation of veins in the neck; (*l.*) nosis, vertigo, albuminuria, œdema of limbs. These phenomena are lom all present, death being too sudden.

Phenomena of Pigmental Embolism.—A peculiar form of embolism is ociated with accumulations of pigment in the blood. It is primarily eloped in the spleen, whence the thrombi are conveyed to the *vena tæ*, gradually increasing in circumference. Malarious fevers establish h pigments in the blood (PLANER, of Vienna); when sanguineous ravasations, in great numbers, are found in the brain, abscesses in the r; laceration of hyperæmic capillaries is also a result—*e.g.*, in the lpighian glomeruli of the kidneys.

PHLEGMASIA DOLENS.

TIN EQ., *Phlegmasia Dolens;* FRENCH EQ., *Phlegmasia Alba Dolens;* GERMAN EQ., *Phlegmasia Dolens;* ITALIAN EQ., *Flemmasia Dolente.*

Definition.—*Obstruction of the veins, radicles, and capillary vessels, with bable implication of the lymphatics, causing a painful, non-œdematous, hard wny swelling of one or more extremities, attended with great prostration.*

Pathology.—*Causation and Morbid Anatomy.*—The disease has been med "milk-leg" or "white-swelling." It is most common after parition; and not unfrequently attends malignant uterine disease, and diastinal or thoracic cancer. In such cases, as well as in gout and thisis, the thrombosis may be in the axillary veins, and the characteric lesions in the arm. Any great drain from the system is apt to followed by no less rapid absorption; and the co-existence of foulness parts from malignant disease, or decomposition of textures in conction with lesions of the uterus after childbirth, or with bits of ained placenta, furnish just such infective material for absorption. rapid absorption of infective fluid occurs, leading to coagulation of od, or to inflammatory changes in the uterine veins. The external ac and femoral veins are also commonly affected. In some way not t fully known, a severe *irritation* is set up amongst the nerves, muscles, d lymphatics of the upper part of the thigh and leg, the lining embrane of the veins, and the areolar tissue of the limb. The results e an elastic hard brawny swelling, till the leg may be twice its usual e, with pain and complete loss of power of motion, painful lymphatics, structed veins, and small venous radicles. At the same time it is to be ted that about 40 per cent. of the cases are non-puerperal, about e-third of which occur in males (F. W. MACKENZIE). In the parturient ses in which the inflammation of the vessels in the limb may be ntinuous with uterine lesions, it is not less probable that an infective aterial, in the form of decomposing particles, passes from the uterine ins into the general blood-stream, and returning through the systemic teries, determines coagulation of blood in the capillaries and venous radicles. It is an occasional sequela of typhus and (one of the most troublesome) typhoid fevers, especially in the left leg (MURCHISON, WARBURTON BEGBIE). In these fevers pulmonary engorgement is a common condition. Coagula may then form in the pulmonary capillaries and subsequently passing into the systemic circulation, may cause venous and capillary obstruction in any or all of the extremities. In phthisis pleurisy, and pneumonia, similar phenomena may occur; and also in the lobular pneumonia following extensive burns, when duodenal ulcerations may be similarly explained (Dr. GEORGE JOHNSON, *Brit. Med. Journ.*, Nov. 30, 1872). Opinions vary as to the share which *phlebitis* or *lymphadenitis* has in the production of phlegmasia dolens. It is also to be noted that phlebitis or plugging of the veins is not confined to the veins of the lower limbs; but occurs in other parts of the body, and affects several parts, as in gouty subjects and in other conditions, when a plugging of the veins may be found in all the four limbs at the same time. The late Sir James Simpson described such coagula occurring in the left arm and side of the face some weeks after an exhausting labour (*Clin. Lec. on Diseases of Women*, p. 354).* The clinical facts rather point to an (infective) blood-poison as the cause of thromboses in phlegmasia; while the site of the coagula may be determined mainly by hydrostatic influences (Dr. FANCOURT BARNES *Brit. Med. Journ.*, Jan. 19, 1880, p. 921.)

Symptoms of *phlegmasia dolens* may set in immediately after labour, or at any time during the next five or six weeks; expressed by fever headache, thirst, nausea, and pain, especially in the lower abdomina and pelvic regions, with great prostration. A chill or a rigor may usher in the disease, with or without pain or swelling of joints. Within twenty-four or thirty-four hours the swelling of one or both lower extremities may be apparent, commencing about the foot or lower part of the leg, from which it extends upwards. The acute stage lasts about fourteen to twenty-one days; and after recovery, if life is spared, many deep veins remain obliterated, compensated for by varicosity and enlargement of superficial ones; so that the limbs are useless for many weeks or months, and too often never recover their wonted power and shape.

Prognosis.—The disease has usually a favourable termination; and as the general health improves, the swelling and tenderness diminish Great care must be taken before and during subsequent pregnancies that the general health is maintained as well as possible. In the infective forms the cases are usually fatal.

Treatment.—Leeches and blood-letting, as recommended by Drs. Davi and Lee, are usually inadmissible. Sedatives and alkaline fomentations perfect rest, simple diet, and opiates to relieve pain, is the most rationa

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*Mechanical causes seem to have some influence in determining the site of coagula Thromboses originate almost invariably in the upper part of the limbs, and at the base of the skull—*e. g.*, femoral veins in legs; axillary veins in the arms, and sinuses of dura mater in the skull. The reason is, that those vessels are so situated that the walls of the veins are no longer supported by neighbouring structures; and, where the force of thoracic aspiration tends to diminish, disappear. At these points there is always a tendency to stasis of the blood-current in the venous system; and it i natural that at these points coagulation should occur (*Lancereaux-Traité D' Anatom Pathologique*, tome premier).

eatment to be followed. "The fluid in which the fomentation flannels e to be wrung out is made by adding one pound of *bicarbonate of soda* d one ounce of *extract of poppies* to one gallon of boiling water. The nnels ought to be changed every thirty minutes, and applied over the iole limb, and over the groin and lower part of the abdomen wherever ere is tenderness. The heat and steam from these are to be retained means of impermeable cloths." Wine, brandy, milk, raw eggs, animal od, ammonia, and bark are sure to be required; and after the acute mptoms have subsided, there is no remedy so beneficial as efficient ndaging, together with the preparations of *iron*, *quinine*, and *tonic etable bitters*, like *calumba*, aided by change of residence (TANNER).

CHAPTER XVIII.

DISEASES OF THE RESPIRATORY SYSTEM.

ECTION I.—AIDS TO THE DIAGNOSIS OF DISEASES OF THE THROAT AND LARYNX.

THE LARYNGOSCOPE.

OR upwards of a century attempts, more or less successful, have been ade to examine the lower part of the throat during life. In connection th the unsuccessful, or only partially successful, efforts may be mentioned e names of Levret (1743), Bozzini (1807), Cagniard de Latour (1825), nn (1827), Babington (1829), Avery (1844), and Garcia (1854). It is not until 1858 that Professor Czermak, of Pesth, brought to perfec- on and practical application the laryngoscope of Garcia, which Türck, of ienna, had unsuccessfully endeavoured to employ a few months previously. this country, the profession is indebted to Drs. Morell-Mackenzie, alker, Johnson, Gibb, Sieveking, and Mr. Durham, for making known e practical application of the laryngoscope, and for many valuable ditions to the literature and armamentaria of this branch of practice.

The following description of instruments used in laryngoscopy, and the ethod of examining the larynx, are taken from Morell-Mackenzie's work, *the Use of the Laryngoscope*, third edition, published by Longmans:—

.) *The laryngoscope* is "an instrument for obtaining a view of the larynx ring life. It consists of two parts; first, a small mirror, called the *ryngeal mirror*, fixed to a long slender shank, which is introduced to the ck of the throat; second, an apparatus for throwing a strong light (solar artificial) on to the small mirror. For thus projecting the luminous ys another (larger) mirror, which reflects the light from a lamp, or the lar rays may be employed; or artificial rays may be concentrated by a ns directly on to the small mirror. When artificial light is employed, e illuminating mirror is slightly concave; when sunlight is used, its surface is plane." (2.) The *laryngeal mirror** is of circular shape, made glass, backed with amalgam, set in a German-silver frame, and attached a shank of the same metal, at an angle of 120°: the shank is fitted int slender wooden or ivory handle. The mirrors, which are of three sizes No. 2 being the most convenient for ordinary cases—are about o twentieth of an inch in thickness; the shank is about four inches in leng and the handle about three inches in length. (3.) The *reflector* is a circu mirror, about three inches and a half in diameter, with a small oval h in its centre; it is fixed to the headpiece by a ball and socket joint. T mirror is by some attached to an elastic band, which encircles the head the operator; by others fixed to a horizontal arm, which is connect with the body of the lamp. Dr. Mackenzie's method of attaching it to spectacle-frame, from which the upper halves of the rims have be removed, is not only the most comfortable, but the most convenient f adjustment and removal.

Light.—For practical purposes the solar light is too inconstant to be service in this country. It is from artificial sources that light must obtained. Any lamp that gives a bright steady light answers the purpo perfectly well. An argand gas burner, constructed on the reading-lam principle, with flexible tubing to connect it with an ordinary burner, convenient for practitioners who only require to use the instrume occasionally; and this lamp is equally convenient for microscopic ophthalmoscopic purposes. For a fixture, Mackenzie's rack-moveme lamp, which admits of both perpendicular and horizontal movements, everything to be desired. In all cases a concentrating lens is required intensify the light. In country practice, a sheet of white paper place behind the light is a good substitute for a concentrator. For demonstr tion and class purposes, the oxyhydrogen light, as arranged at th Hospital for Diseases of the Throat, is most convenient. The apparat is so contrived that as many as six people, in addition to the demonstrato can simultaneously see the larynx of a patient.

The *method of making an examination* is as follows:—The patien should sit upright, facing the observer, with his head inclined ver slightly backwards. The observer's eyes should be about one foo distant from the patient's mouth, and a lamp burning with a stron clear light should be so arranged at the side of the patient, that the flam of the lamp is on a level with the patient's eyes. The observer shoul now put on the spectacle-frame, with the reflector attached, and directin the patient to open his mouth widely, should endeavour to throw a dis of light on to the fauces, so that the centre of the disc corresponds wit the base of the uvula. The patient should be directed to put out hi tongue, and the observer should then hold the protruded organ gentl but firmly between the finger and thumb of the left hand, previousl enveloped in a small soft cloth or towel. Then, holding the mirror (previously warmed to prevent condensation of the moist expired air) like pen in the right hand, it should be quickly introduced to the back of th throat, and kept as far as possible from the tongue, the face bein directed downwards. The posterior surface of the mirror should rest on the uvula, pushing it rather upwards and backwards towards the posterior nares. The exact angle which the mirror should bear to the laryngeal aperture must depend on the degree of flexion backwards of the patient's head, the particular angle which the plane of the laryngeal aperture bears to the horizon in the case undergoing inspection, and on the position of the observer. Undue faucial irritability may exist, and there may be other accidental physical difficulties, but in the majority of instances the difficulty is with the observer, not with the patient. For the purpose of investigating the action of the vocal cords the patient should be directed to inspire deeply, or to produce a vocal sound, as, "ah," "eh," &c. Where the epiglottis hangs low, it is useful to make the

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* Square mirrors are used in France, but they are not so convenient as round ones Oblong mirrors are of service where the laryngoscope has to be used on patient affected with enlarged tonsils.

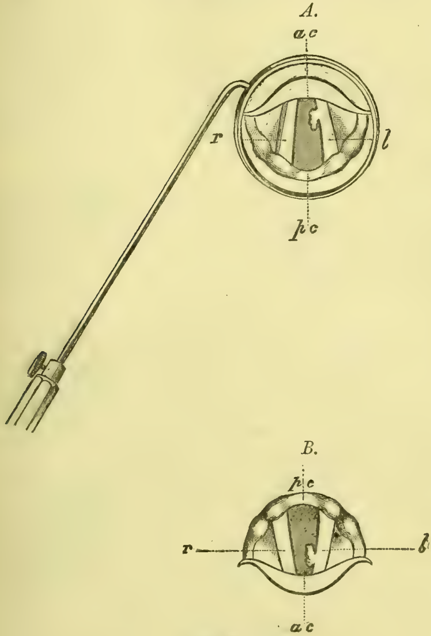

Fig. 52.*

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* Drawing showing the relation of parts in the larynx (*B*) and the laryngeal mirror (*A*). *a c*, Anterior commissure of the vocal cords; *p c*, Posterior commissure of the vocal cords; *r*, Right vocal cord; *l*, Left vocal cord, with a wart on it (after Dr. Morell-Mackenzie).

Description of Woodcut—Fig. 53.

A. Laryngoscopic drawing, showing the vocal cords drawn widely apart, and the position of the various parts above and below the glottis, during quiet inspiration :—*ge*, Glosso-epiglottic folds; *u*, Upper surface of epiglottis; *l*, Lip of epiglottis; *c*, Cushion of epiglottis; *v*, Ventricle of larynx; *ae*, Ary-epiglottic fold; *c W*, Cartilage of Wrisberg; *cS*, Capitulum Santorini; *com*, Arytenoid commissure; *vc*, Vocal cord; *vb*, Ventricular band; *pv*, Processus vocalis; *cr*, Cricoid cartilage; *t*, Rings of trachea (after Dr. Morell-Mackenzie).

B. Laryngoscopic drawing, showing the approximation of the vocal cords and the position of the various parts in the act of vocalisation :—*fi*, Fossa innominata; *hf*, Hyoid fossa; *ch*, Cornu of hyoid bone; *c W*, Cartilage of Wrisberg; *cS*, Capitulum Santorini; *a*, Arytenoid cartilages; *com*, Arytenoid commissure; *pv*, Processus vocalis (after Dr. Morell-Mackenzie).

C. Constriction of the glottis, erroneously called *respiratory glottis*, the arytenoid processes converging, and causing the posterior section of the glottis to be almost triangular (after Elfinger and Czermack).

D. Complete relaxation of all the parts, as when the glottis under ordinary circumstances is open for breathing (after Elfinger and Czermack).

E. Bifurcation of trachea and commencement of bronchial tubes, seen on widely opening the glottis, as in deep inspiration, and straightening the trachea (after Elfinger and Czermack).

F. The posterior nares, as seen in rhinoscopy :—*sn*, Septum nasi; *s*, Superior turbinated bone; *m*, Middle turbinated bone; *i*, Inferior turbinated bone; *a*, Superior meatus; *b*, Middle meatus; *c*, Inferior meatus; *e*, Eustachian orifice; *r*, Ridge between the Eustachian opening and lower border of the nasal fossa; *u*, Uvula (after Dr. Morell-Mackenzie).

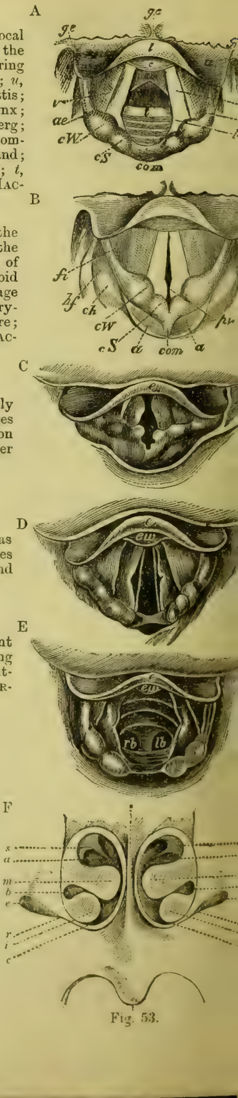

Fig. 53.

patient laugh or strike a high or falsetto note. The beginner must remember that in examining the larynx the objects are reversed in the nirror, *not as regards right and left*, but with reference only to the antero-posterior direction. The part which in reality is nearest to the observer, the anterior commissure of the vocal cords, becomes farthest in the image, and the posterior or inter-arytenoid commissure, which in reality is farthest from the observer, becomes nearest in the image (Fig. 52). The instrument is absolutely necessary, not only for precision of diagnosis, but for accuracy in applying local remedies to the inferior part of the pharynx, the larynx, and trachea, as well as to the posterior surface of he pillars of the fauces, and the posterior nares. The scientific investigation of the latter part, by means of mirrors, is called Rhinoscopy.

The series of woodcuts (Fig. 53.), A, B, C, D, E, and F, on the previous page, are in illustration of appearances seen by the laryngoscope and rhinoscope.

SECTION II.—DISEASES OF THE LARYNX.

LARYNGITIS.

LATIN EQ., *Laryngitis;* FRENCH EQ., *Laryngite;* GERMAN EQ., *Entzündung des Kehlkopfs;* ITALIAN EQ., *Laringitide.*

Definition.—*Inflammation of the lining membrane of the larynx.*

The disease presents itself in three forms, viz.—(*a.*) Acute catarrhal aryngitis; (*b.*) œdematous laryngitis; and, (*c.*) chronic laryngitis.

(*a.*) *Acute Catarrhal Laryngitis.*

Pathology.—(*a.*) *Causation.*—Relaxing habits and previous attacks are the strongest *predisposing* causes of this affection. Males are more iable to it than females; and adults are more frequently attacked than children, though the greater urgency of the symptoms in the latter has led some to an opposite conclusion. Amongst *exciting* causes may be mentioned, exposure to cold draughts of air; and irritating drinks, pungent vapours, and a dusty atmosphere may be considered as *traumatic* causes. *Acute laryngitis* may also be caused by extension downwards of a similar affection from the pharynx. It rarely occurs from spreading of nflammation upwards from the trachea and bronchi.

(*b.*) *Morbid Anatomy.*—The disease is essentially a catarrhal inflammation of the mucous membrane, and is usually accompanied by a similar condition of the trachea. The hyperæmic condition of the mucous membrane is followed by a slightly increased secretion; and, cell-growth taking place at the surface, the mucous membrane becomes swollen and sodden. There is a very slight, and, indeed, scarcely perceptible deposit n the submucous tissue. The dysphonia or aphonia is due, partly to the altered density of the vocal cords, and partly to imperfect muscular action; for the slight pain occasioned by the use of the muscles causes them to be insufficiently employed. The secretion from the inflame membrane is at first scanty and watery, containing imperfectly develope epithelial cells. As the disease progresses and begins to abate, th expectoration becomes thicker and more rich in young cells, whic rather resemble pus-cells" (NIEMEYER). In rare cases acute catarrha laryngitis passes into œdematous laryngitis.

Symptoms may commence as a slight catarrh, and gradually take th form of acute inflammation; or it may from the first be ushered in b rigors, and rapidly followed by fever and elevation of temperature Locally, a sense of uneasiness in the throat, generally referred to th *pomum Adami,* is soon followed by a feeling of constriction and strangulation. In all cases, modifications of the natural functions of the part ar the most striking phenomena. Vocalisation, cough, and respiration ar all more or less modified. The voice is at first hoarse, but as the diseas advances there is *aphonia.* The cough is at first clear and shrill, the harsh and croupy, and finally aphonic. In a well-marked case, the brass tone peculiar to the disease terminates in a hissing noise, and begin similarly by a hissing inspiration in a muffled manner; because the lip of the glottis being thickened, irregular, and rough, cannot be sufficientl closed to begin a sharp sound (HYDE SALTER). The respiration is not, a a rule, affected in adults; but in children the breathing is often embarrassed, owing to the encroachment of the swollen mucous lining on th small available breathing space. In such cases there is a peculiar nois like a loud whisper, which accompanies both inspiration and expiratio Inspiration is from the first laborious and wheezing; afterwards it is ver much lengthened and stridulous, and starts sharp from the conclusion o the previous expiration. In the case of children the respiration is ofte severely interfered with during sleep, owing to the blocking up of th rima glottidis with viscid secretion. In this event the child ma suddenly awake with a most serious suffocative attack, which doe not pass off until the larynx is cleared of all adherent mucus. Mucou râles are heard over the whole larynx. In the rare cases in which œdem supervenes, and the calibre of the larynx is consequently narrowed, th respiratory process becomes most laborious and painful, and the anxiet of the patient extreme.

With the laryngoscope general or partial hyperæmia is seen, togethe with some swelling of the ventricular bands and epiglottis. There ar also occasionally scattered erosions, or superficial ulcers. Evidence o various paralyses of the laryngeal muscles is also not unfrequently found.

Diagnosis.—Where respiration is much embarrassed, acute laryngit has to be distinguished from laryngismus stridulus, from diphtheria, an from a foreign body in the larynx. The first-named disease is identifi by the complete intermission of all serious symptoms in the interv between two paroxysms. Diphtheria may be recognised by the fal membrane, and the greater severity of its symptoms; while the sudd onset and persistence of the dyspnœa will in most cases be sufficient identify a foreign body in the larynx.

The **Prognosis** is almost invariably good, though less so in child than in adults.

Treatment.—In its early stages a warm, moist, and uniform temp ure, with complete rest of the parts, is of the first importance. Not nly should all use of the voice be interdicted, but the tendency to cough hould as much as possible be arrested by the administration of small loses of *morphia*, especially in those cases in which paroxysmal cough is a prominent symptom. Inhalations of *hot steam*, or of *steam* impregnated with the volatile principles of *benzoin*, *hops*, or *conium*, are of the greatest service. Hot poultices and fomentations may also be ordered. Neither local nor general *blood-letting*, *blistering*, *mercury*, *antimony*, nor other lowering remedies are to be recommended; after the acute symptoms have passed off, astringent solutions may be applied to the larynx to hasten the cure.

In children, a warm moist temperature, poultices, warm emulcent drinks, and if there is much dyspnœa, emetics of *sulphate of copper* or *sulphate of zinc*, are the most serviceable methods of treatment. When laryngitis occurs in a subacute form, or shows a tendency to become chronic, a regular system of inhaling should be prescribed. Any of the appended forms may be employed; and there are many others contained in the very useful Pharmacopœia of the Hospital for Diseases of the Throat, published by Churchill, 1876. For the administration of the following remedies, the Eclectic Inhaler possesses great advantages; for whilst the vapour to be inhaled is thoroughly saturated with the volatile principles employed, and is kept at a uniform temperature, the patient is able to inhale without effort.[*] The inhaler devised by Dr. James Adams, of Glasgow, is also to be commended.

℞. Creasoti, fl. dr. iij.; Glycerine, fl. dr. iij.; Aquæ ad. fl. oz. iij.; *mix.* A teaspoonful to be added to a pint of water at 150° Fahr., and inhaled for five minutes twice or thrice daily.

Ol. Pini. Sylvestris, fl. dz. ij.–iij.; Mag. Carb. Lev., gr. 60–90; Aquæ ad. fl. oz. iij.; *mix.*, and use as above.

In addition, Oil of Juniper, fl. dr. i., ad. fl. oz. iij.; Oil of Santal, ♏xv.; Oil of Myrtle, ♏xx., are all of service.

It will be found convenient to prescribe light carbonate of magnesia as a medium, in the proportion of one grain to every two minims of the oil; and the addition of twenty grains of camphor to any of the above makes the inhalation additionally stimulative. When laryngitis is associated with inflammation of the pharynx, lozenges containing guaiacum are very useful in removing the hyperæmic condition. Even when the pharynx is not affected, the experiments of Fournié have proved that lozenges have a decidedly beneficial effect on diseases of the larynx. It is necessary after an attack of acute laryngitis to caution a patient that he is very liable to a recurrence of this affection, and that he should therefore be very careful to guard against all preventible causes of the disease. As laryngitis is more common to those engaged in in-door than out-door occupations, and to those living in towns than in the country, Niemeyer recommends out-of-door exercise, with proper precautions, as a valuable prophylactic measure. Acute laryngitis sometimes occurs as a complication of erysipelas; but *erysipelatous* inflammation of the larynx originating in that part is rare. The local treatment should at first be directed towa arresting the inflammation, by sucking ice, &c.; but when the mor process is advanced, the treatment is the same as that required for ordin acute laryngitis. The constitutional treatment should be that applica to other forms of erysipelas. Tracheotomy, although offering a favourable chance than in simple laryngitis, should be performed if symptoms are urgent. Of other *catarrhal* forms of laryngitis may mentioned those of measles and scarlatina. In *measles* the affection of larynx may be either a simple catarrh, or a severe diphtheritic affecti The catarrhal form may occur before the eruption appears, a day or t after the rash has come out, or when it is beginning to decline. Althou the inflammation is often severe, it is seldom sufficient to cause anxie The diphtheritic form is much less common, and "seldom begins until eruption of measles is on the decline, or the process of desquamation commenced. Its appearance is most frequent from the third to the si day after the appearance of the eruption; but it oftener occurs at a la than at an earlier period" (WEST). The treatment should be the same that for primary croup. In *scarlatina* the laryngitis may be eit œdematous or croupous: they are fortunately both rare complicatio The œdematous form may be one of the manifestations of the exanth or may be dependent on debility, or it may be due to the renal affecti which is so common a sequel of scarlatina. The croupy form is common, and seems to have been peculiar to some epidemics. In all ca of laryngitis associated with scarlatina, there is a great tendency to ulcerative process. The treatment required is of a tonic and nourishi character, with the free use of well-diluted stimulants. Tracheotomy m be necessary when there is œdema; but scarification should always first tried.

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[*] It can be obtained of Messrs. Bullock & Reynolds, 3 Hanover Street, Hanover Square, London; or of Maw, Son, & Thomson, London.

(*b.*) *Œdematous Laryngitis.*

Pathology.—(*a.*) *Causation.*—Œdematous laryngitis may occur either a primary, or as a secondary affection. The primary, or idiopathic for is much less common than is usually supposed, simple inflammation of t larynx seldom assuming an œdematous character. The majority of cases œdematous laryngitis are the result of extension from neighbouring pa generally from the pharynx; the inflammation is probably in many instanc of an erysipelatous nature. A third variety of the disease is that whi occurs as a complication or sequel of some other disease. Thus, acu œdema may supervene in the course of small-pox and typhoid fever, a occasionally in scarlet-fever. In all these cases the œdema is more lik to be followed by ulceration than in ordinary acute laryngitis, wh resolution is not unfrequently the termination. Œdema is also an alm constant consequence of disease of the laryngeal cartilages. It is a occasionally met with as a complication or sequel of renal disease, but so frequently as to justify the specific form described by Fauvel as "*apho* *albuminurique*." Œdema is likely to occur where areolar tissue is abunda Hence, we most frequently find it in the folds of the mucous membra of the larynx, viz.—the glosso-epiglottic, ary-epiglottic, inter-arytenoid, he mucous membrane over the vocal cords being immediately in con-ct with the elastic tissue of the cords—there is no submucous areolar ssue in this situation—the œdematous fluid cannot pass the barrier of ie vocal cords. Hence, we have a supra- and a sub-glottic œdema. The vourable results likely to accrue from tracheotomy in the former case ill be at once apparent when the anatomical arrangement is taken into nsideration.

Symptoms.—The first sign of œdematous laryngitis is the sensation of foreign body in the throat. This, as a rule, is speedily followed by all the idences of a profound impediment to respiration. If the epiglottis or y-epiglottic folds are first attacked, the feeling of strangulation and fficulty of swallowing will be among the more early phenomena. If, on e other hand, the ventricular bands, or vocal cords, are the primary seat the disease, loss of voice will precede the other symptoms. As soon as it ecomes fully established, the dyspnœa is constant and most distressing; id unless speedily relieved, the patient becomes cyanotic, and death occurs om asphyxia. With the laryngoscope the mucous membrane, at first ily hyperæmic, is seen to become œdematous; and from the swollen ndition of the epiglottis, the rest of the larynx may be hidden from ew. If the vocal cords are visible, they are generally congested and ightly swollen, but not œdematous; the ventricular bands, however, enerally quickly take on the œdematous character, and may sometimes e seen to meet on the middle line at each inspiration.

Diagnosis.—Where a laryngoscopic examination can be made, this should resent no difficulty; but where no satisfactory view of the interior of ie larynx is attainable, the disease may be mistaken for laryngismus ridulus, laryngeal polypus, foreign body in the larynx, or diphtheria. careful consideration of the history and symptoms will, however, enerally guide the practitioner to a correct conclusion.

Prognosis.—In giving an opinion, the age of the patient is the most nportant consideration. The disease is rare in childhood, but in the econd decade of life it is extremely fatal, and even in adults a very uarded opinion should always be expressed. Not only does the danger epend on the amount of œdema present, but, where tracheotomy has been erformed for relief of the symptoms, there is still the risk of extension f the disease to the trachea and bronchi.

Treatment.—In all cases of œdema of the larynx, whatever the cause, carification and hot steam inhalations, as recommended in acute laryngitis, re the most serviceable aids. When suffocation is imminent, tracheotomy hould be performed.

(c.) *Chronic Laryngitis.*

Pathology.—(*a.*) *Causation.*—In addition to those causes which predis-ose to or excite the acute disease of which this is often a sequel, the buse of alcohol and tobacco, especially the former, sometimes gives rise o chronic inflammation.

(*b.*) *Morbid Anatomy.*—This disease is essentially a chronic inflammation f the mucous membrane of the larynx, accompanied with slight enlargement and tortuosity of the vessels of the submucous tissue. Cell germination takes place at the surface; and instead of the pale semi-transparent mucus usually secreted in small quantities, a more abundant discharge occurs, in which pus is mingled in varying quantities with true mucous corpuscles.

Symptoms.—The principal symptom is modification or even complete loss of the voice; the respiration is generally but little affected; but there is often a tickling cough, with expectoration of small pellets of mucus, and a frequent desire to clear the throat. Occasionally the patient complains of a pricking or tickling sensation in the throat. With the laryngoscope general or partial congestion of the mucous membrane is at once visible. There is usually a certain amount of thickening of the epiglottis and ventricular bands when the affection is of old standing. It is of great importance in those cases, by comparing the local manifestations with th
constitutional condition, to decide whether the thickening is simply th
result of inflammatory tumefaction, or œdematous infiltration, or whethe
it is due to tuberculous exudation. In all cases of chronic laryngitis c
long standing the lungs should be carefully examined.

Treatment.—Applications of mineral astringents to the larynx, eith
with the laryngeal brush, or in the form of atomised inhalations, are
the greatest service; and the "alternation of topical remedies is often
efficacious in the cure of chronic laryngitis as it is in the treatment
chronic inflammation of other mucous passages." Although Niemeyer
of opinion that "induration of the submucous tissue (of the larynx)
incapable of resolution," it is the daily experience that local treatment
chronic thickening of this region, unless, indeed, it be due to phthisis,
attended with the very best results. Stimulating inhalations may be us
with great benefit. "Amongst the remedies I have found most efficaciou
writes Dr. Morell-Mackenzie, "are solutions of *perchloride of iron* (gr. 60-1
ad. fl. oz. i.), *chloride of zinc* (gr. 20-30 ad. fl. oz. i.), *sulphate of copper* (gr.
ad. fl. oz. i.), *sulphate of zinc* (gr. 5 ad. fl. oz. i.), *alum* (gr. 30-60 ad
oz. i.), *chloride of aluminium* (gr. 30-60 ad. fl. oz. i.). The *perchloride of*
and *chloride of zinc* are the solutions I use most largely. *Glycerine*
also be found a most useful solvent for these agents, as its consistenc
better calculated than that of water to keep up prolonged and
contact between the remedy and the affected membrane. I seldom em
solutions of nitrate of silver as applications to the larynx, for whilst I
not found them more beneficial than other mineral astringents, the
much more likely to produce spasm and nausea" (*Use of the Laryngo*
third edition, page 98). Of atomised solutions, those of *tannin* (gr.
fl. oz. i.), and *perchloride of iron* (gr. 2 ad. fl. oz. i.) are most u
Bergson's Atomiser, known in this country under the name of An
Clark's Spray Producer, Dr. Richardson's Ether Spray Apparatus, an
James Adams's Siegle's Inhaler, are the most convenient for adminis
atomised liquids. Functional rest is, of course, of great importa
those cases of chronic laryngitis in which the voice is much affecte
complete silence cannot be enforced, the patient should be recomm
to speak only in a whisper. Relaxation of the uvula, being a fr
cause of irritation of the larynx, should be treated by astringent lo
of *tannin*, *rhatany*, or *kino*; and if necessary the elongated part sho

t off. A warm dry climate is of essential service in obstinate cases of ronic inflammation of the larynx; as are also some mineral waters, rticularly those of Ems, Ober-Salzbrunnen, Les Eaux Bonnes, Luchon, uterets, Aix-les-Bains.

Besides the common form above described, there are certain varieties chronic laryngitis to which it will be necessary to give a brief considera- on. These are—first, those due to a morbid state of some special tissue the larynx; and, secondly, those due to a morbid condition of the neral system, and mainly dependent on constitutional causes. Of the rmer, we have glandular laryngitis and phlebectasis laryngea; of the ter, syphilitic laryngitis and laryngeal phthisis.

Chronic glandular laryngitis, or chronic inflammation of the minute race- ose glands of the larynx, is generally associated with disease of the llicles of the pharynx and tonsils; but, as the glandulæ of the larynx are l of the racemose character, it is better to use the generic term "glandular ryngitis." It is this disease which has most improperly received the name *dysphonia clericorum*, or clergymen's sore throat. The most common roat affection of the clergy is not, however, glandular pharyngitis or ryngitis, but merely chronic inflammation of the pharynx and larynx, th paresis of the laryngeal muscles. Glandular laryngitis, on the other nd, is not at all peculiar to those whose profession requires them to ake sustained use of the voice, but generally occurs in people of feeble nstitutional powers.

The *symptoms* are similar to those of ordinary chronic laryngitis, but e disease is more intractable. There is a constant sensation of a foreign dy in the throat, and a corresponding disposition to hawk and clear the roat. With the laryngoscope, the enlarged orifices of the laryngeal nds may sometimes be made out. *Pathologically*, the disease is essen- lly one of the secretory system, the normal secretion of the minute cemose glands, instead of being clear and transparent, becoming thick, ite, and opaque. By some observers it is thought to be due to im- rfect or perverted digestion; but it is more probable that the glandulæ the stomach are simultaneously affected.

The *treatment* is the same as that recommended for simple chronic ryngitis, except that the use of strong solutions of *nitrate of silver* is more ten indicated than in that affection. There is frequently considerable ebleness of the voice, in which case stimulating inhalations and the plication of electricity are the appropriate measures.

The second variety, that of *phlebectasis laryngea*, first described by Dr. orell-Mackenzie (*Lancet*, July 6, 1862), consists of a venous congestion the larynx. It is very uncommon. The symptoms are slight, and ually consist in no more than an uneasy sensation in the larynx, and a ore or less frequent cough. With the laryngoscope, dark vessels may be en on various parts of the larynx, particularly on the ventricular bands. he *treatment* consists in the destruction of the vessels by electric cautery, d the use of vigorous constitutional remedies.

The varieties of *syphilitic laryngitis* and *phthisical laryngitis* depend on special constitutional condition, and will be treated under the next bject, that of ulcer of the larynx.

ULCER OF THE LARYNX.

LATIN EQ., *Ulcus;* FRENCH EQ., *Ulcère;* GERMAN EQ., *Geschwür;*
ITALIAN EQ., *Ulcera.*

Definition.—*Loss of substance of the larynx, in rare cases the result of sim*
inflammation, caused either by syphilis or phthisis.

(*a.*) *Syphilitic Ulceration.*

Pathology.—(*a.*) *Causation.*—The great frequency with which t
larynx is affected in both the secondary and tertiary forms of syphi
does not appear to justify the belief of Gerhardt and Roth, that t
localisation of syphilis in this organ is in some degree determined
fortuitous catarrhal inflammation. Mr. Durham states "that, accordi
to his own experience, a very large proportion (from thirty to forty
cent.) of the cases of laryngeal disease met with in hospital practice amo
the surgical out-patients, are of syphilitic origin" (HOLMES'S *System*
Surgery, Second Edition, Vol. IV., page 556). Nor can the laryng
complications in secondary syphilis be ascribed solely to extension of t
disease from the pharynx, as not only do cases frequently occur in whi
the larynx is affected without the mouth, fauces, or pharynx presenti
any signs of the malady, but the larynx is frequently attacked some wee
after the disease has disappeared from the pharynx.

Ulceration of the larynx is not very frequently met with in seconda
syphilis. When present it generally occurs from six to twelve mont
after the primary infection, and is as a rule quite superficial, yielding rapi
to appropriate treatment. The parts most frequently affected are t
epiglottis and interarytenoid commissure. *Condylomata* are, on the ot
hand, of much more frequent occurrence. Dr. Mackenzie met with for
four cases of condylomata in 110 patients suffering from the ea
symptoms of laryngeal syphilis, while Gerhardt and Roth found th
present in eighteen out of fifty-six patients suffering from the constitutio
disease. Condylomata appear in the larynx in the form of smooth yell
projections of from three to seven millimetres in diameter, and genera
affect the same regions as the superficial ulcerations described above.

The most common phenomenon of secondary laryngeal syphilis
obstinate congestion of the mucous membrane. This is not necessar
always due to syphilitic infection, but the fact that it is met with, acco
ing to Dr. Mackenzie, in forty-three per cent. of cases of early laryng
syphilis, shows that there is a close etiological relation between the t
conditions. *Tertiary syphilitic ulceration of the larynx* always occurs in
late stage of the disease. The process may extend from the palate
pharynx to the epiglottis; but much more frequently when a pati
seeks relief on account of recent laryngeal disease the scars of form
ulceration will be seen in the pharynx. The characteristic of the ulcerati
is its disposition to cause permanent loss of substance. It commo
attacks the epiglottis; but the arytenoid cartilages and ary-epiglottic fo
are often affected, and the vocal cords and ventricular bands not unfrequen

fer. The destructive process destroys both the mucous and submucous sues, and occasionally involves the cartilages, large portions of the iglottis being thus not uncommonly eaten completely away. (Fig. 54, and B.[*])

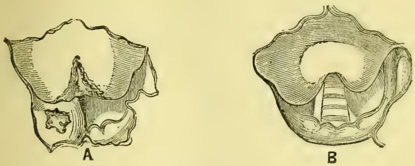

Fig. 54.[*]

Ulceration of the larynx occasionally originates from gummatous deposits, ost frequently seen on the posterior wall of the larynx as smooth, round vations of considerable size, which become inflamed, burst, and leave a rge open ulcer, which, if not checked, will speedily spread and penetrate the deeper structures.

The **Symptoms** of laryngeal syphilis are extremely variable. The patient ay experience nothing but a slight tickling in the throat and a minor gree of hoarseness; or, on the other hand, may suffer the extremest gonies of suffocative dyspnœa. Cough is not usually a prominent mptom, but the vocal function is almost invariably impaired, absolute honia being not uncommonly present. There is seldom much pain hen the parts are at rest; but the movements involved in speaking, and pecially in swallowing, sometimes cause so much distress, as to render e exercise of these functions almost an impossibility. In other cases eglutition is impaired by distortion or loss of substance on the part of the iglottis which, by rendering that valve incompetent to adequately close e laryngeal inlet, allows the food to enter the air-passages.

The **Diagnosis** of syphilitic ulceration of the larynx will be more onveniently treated under that of phthisical ulceration.

Prognosis.—This is favourable so far as life is concerned, the most xtensive ulceration yielding, as a rule, to appropriate treatment; when, owever, the cartilages are involved, the chances of a fatal issue are aterially increased. The great danger of all forms of tertiary syphilitic isease is the narrowing of the glottis from thickening and œdema. The tter subject is treated at p. 688 under "*Contraction of the Larynx,*" but in hese days of the laryngoscope, the cases should be very rare in which lceration cannot be arrested and healed. The prognosis as regards the ocal function will depend on the seat and extent of the ulceration;

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[*] A. Active syphilitic ulceration of the epiglottis and right arytenoid cartilage, with dema and general thickening. A "false excrescence" or cicatricial outgrowth rojects across the left ary-epiglottic fold (View on Jan. 6, 1868).

B. The same larynx, eighteen days later, after treatment by iodide of potassium nd local remedies. The œdema has subsided, the ulcers are healed, and the right ocal cord has come into view. Some general thickening, however, especially of he epiglottis, still remains, and the left vocal cord is still hidden by the swollen entricular band on the same side (View on Jan. 24, 1871).

where the vocal cords are extensively ulcerated, or the cartilages a necrosed, permanent loss of voice is a very probable issue.

Treatment.—*Secondary* laryngeal affections are sometimes difficult cure, but after undergoing certain phases of evolution they may disappe spontaneously. If allowed to run their natural course, condyloma generally disappear at the end of two or three months, but chronic cong tion is more intractable. Stimulating inhalations, and the local applicati of astringents are sometimes of great service. Ulceration of the lary (secondary) is seldom so severe as to require the application of *solid nitr of silver.* In *tertiary* laryngeal syphilis, large doses of *iodide of potassiu* in combination with *ammonia*, and freely diluted with water, are of t greatest use; but local treatment is also of the first importance. T application of *solid nitrate of silver*, or of strong solutions of the same sa (60 gr. ad. fl. oz. i.), or of sulphate of copper (15 gr. ad. fl. oz. i.), attended with the most satisfactory results. For applying the sol nitrate the method of fusing a very small portion of the salt on to curved aluminium wire far surpasses, in simplicity and safety, any oth form of laryngeal *porte-caustique.*

(*b.*) *Phthisical or so-called Tubercular Ulceration of the Larynx.*

Patholgoy.—(*a.*) *Causation.*—"The causes are the same as those whi give rise to other laryngeal affections (such as exposure to cold, function excesses, &c.)—*plus* a special constitutional condition, either inherited acquired, through which cell-germination takes place in the submuco tissues" (MORELL-MACKENZIE; see also article on *Scrofula*, page 1000, Vo I., and *Pulmonary Phthisis* at a subsequent page). It is very doubtf whether laryngeal phthisis is in any way actually *caused* by disease of t lungs, but as a rule the pulmonary disease precedes the throat affection; a though Dr. Mackenzie states that "numerous cases of laryngeal phthis have come under his inspection, where the most experienced stethoscopis have been unable to discover a trace of lung disease," he neverthele confesses that "he has seldom met with a case of laryngeal phthisis in t dead subject without finding pulmonary disease." The course of even is generally—first, hyperæmia; secondly, thickening; thirdly, ulceratio The ulcerations vary in size from that of a pin's point to a threepen piece. One of the common sequelæ of tubercular disease of the larynx caries of the cartilages.

The **Symptoms** are at the outset similar to those of laryngitis. Whe the thickening is great, dysphagia is most prominent—solid foo frequently getting into the larynx, and drink being violently eject through the nares. The voice is affected at a very early stage, general caused by change of structure in the vocal cords; but it may be t result of weakened approximative action of the cords. The cough not as a rule a prominent symptom in the early stages, but as the disea advances it often becomes violent and paroxysmal, and more or le aphonic; occasionally it is but a slight tickling cough, with scanty expe toration. Respiration is always affected in the later stages of the diseas and the dyspnœa is sometimes so extreme as to demand tracheotomy.

With the *laryngoscope* the first thing that strikes the observer is the :tremely pale and almost ashy colour of the mucous membrane. When ickening has commenced, the ary-epiglottic folds are seen to be first fected. There may be only thickening of one of these folds; but later th sides are generally involved, and the posterior portion of the larynx us presents the appearance of one or two pear-shaped semi-transparent dies, according as one or both sides are diseased. Although, for the ke of comparison, the accompanying diagrams represent only one side the larynx as affected, the appearance is far more frequently symme-ical, or nearly symmetrical. (Fig. 55, A, B, and C.)

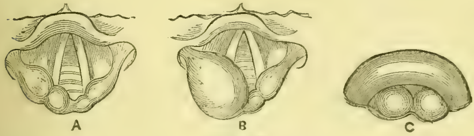

Fig. 55.*

When the epiglottis is attacked, the normal contour of the valve is ften lost, and, in the later stages, a turban-like appearance is produced. The ulceration of phthisis, if it attacks the mucous membrane, is gener-lly multiple, giving the parts a worm-eaten appearance. The edges of e epiglottis are often eroded and the cartilage exposed, and numerous nall ulcers may be found at its root, on a level with the vocal cords. he ulcerative process never commences on the lingual aspect of the piglottis, although it may extend thither from the posterior surface of e valve. The cartilaginous portion of the glottis is the region most mmonly affected; penetrating ulcers, which affect both the vocal cords nd ventricular bands, being almost invariably present in advanced cases, ist behind the posterior extremity of the ventricular orifice. It is in his situation that the arytenoid cartilages become affected, first by ossifi-ation, and subsequently by caries.

Diagnosis.—The thickening of laryngeal phthisis may be distinguished rom acute œdema by the rapid occurrence of the latter disease, and the reater transparency of the simple œdematous swelling. The thickening f syphilis is less symmetrical and more irregular in every respect, and it s rapidly followed by destructive ulceration; or, if this be averted, by esolution and absorption. On the other hand, the thickening which ucceeds extensive syphilitic cicatrisation always gives rise to considerable distortion—a phenomenon which never occurs in laryngeal phthisis. I phthisis, thickening is an early symptom, and generally exists for a lon time before deep ulceration takes place. Dr. Mackenzie considers the pa pyriform swelling of the ary-epiglottic folds and mucous membrar covering the arytenoid cartilages as pathognomonic of phthisis in tl larynx. But although these pyriform swellings are typical of larynge phthisis, it must be clearly understood that the disease may present itse under other forms. Thus, there may be turban-like thickening of tl epiglottis, or scattered ulceration of the general mucous membrane, c diffused deposit.

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* A. *Incipient Laryngeal Phthisis.*—Deposit has taken place around the cartilage of Vrisberg and the Capitulum Santorini, but the normal contour of the parts is not yet ost.

B. *Advanced Laryngeal Phthisis*, affecting the right side of the larynx, and producing characteristic pyriform swelling.

C. *Laryngeal Phthisis*, affecting the epiglottis, and producing the turban-like swelling f the valve. The interior of the larynx is hidden by the thickened epiglottis, but the rytenoid cartilages can be seen as two round balls. The round appearance is due to he ary-epiglottic folds being hidden by the epiglottis. If the whole of the orifice of he larynx were visible, the "pyriform" appearance would be present.

Prognosis is most unfavourable. A patient in whose case there i pyriform swelling of the ary-epiglottic folds very rarely recovers, even i there be no evidence of lung disease. The symptoms may be mitigate and life prolonged by treatment. Those cases generally terminate mos rapidly in which the epiglottis is much affected.

In the **Treatment** of *phthisical ulceration*, remedies similar to thos recommended for chronic laryngitis may be employed with advantag Mineral astringents have a beneficial effect in the early stages, and ar useful in relieving the dysphagia when ulceration of the epiglottis ha taken place. Sedatives, whether in the form of inhalations or as med cines which allay the cough, and so keep the larynx at rest, are also to b recommended. Treatment, however, is at the best but palliative. Ever effort should therefore be made to carry out preventive measure Congestion of the larynx in patients of a phthisical tendency, should b treated with the greatest promptitude—first, by proper local treatment secondly, by complete rest of the voice; thirdly, by a suitable climate— warm, dry, and uniform temperature being most to be commended. Th constitutional treatment required is similar to that which will be foun described under "Pulmonary Phthisis."

ABSCESS OF THE LARYNX AND NECROSIS OF THE CARTILAGES.

LATIN EQ., *Necrosis Cartilaginum;* FRENCH EQ., *Nécrose des Cartilages* GERMAN EQ., *Necrose des Knorpels;* ITALIAN EQ., *Necrosi dell Cartilagina.*

Definition.—*Death of the cartilages, by a molecular or truly carious proces usually preceded by separation of the perichondrium, and sometimes attende with circumscribed collections of pus.*

Pathology.—(*a.*) *Causation.*—This disease, more properly termed carie is in most instances a sequel of *laryngeal phthisis*, *typhoid fever*, or *tertiar syphilis.* It may, however, depend on a gouty diathesis, or it ma originate as a purely local disease—the lungs not being in any wa affected. It is now generally accepted that death of the cartilages of th larynx almost always originates in separation of the perichondrium, ossifica tion being an intermediate stage. Two instructive cases, bearing on thi point, are related by Dr. Mackenzie, in the twenty-first volume of th *Pathological Transactions.* In the first (p. 46), severe pain in the left sid of the throat preceded any swelling in the larynx, or in the tissue ernal to it, for many months. After death, a circumscribed abscess, size of a hen's egg, was found, which almost completely blocked up œsophagus; and at the bottom of the abscess a small portion of the coid cartilage—about the size of a threepenny piece—was found to ve been completely removed by molecular destruction. In the second e (p. 56), there was from the commencement extreme dysphagia; and s was the prominent symptom. After death "a large abscess was nd in the thickened tissues covering the anterior surface of the spinal umn, and extending from the fourth cervical to the second dorsal tebra. The vertebræ themselves were not exposed or diseased. The cess involved both the lateral walls of the œsophagus, the sides of the arynx as high as the upper border of the cricoid cartilage, and the terior wall of the œsophagus, from the middle of the cricoid cartilage wnwards for two inches. The posterior surface of the lower third of cricoid cartilage was completely exposed, and had not undergone ification. At the lower border of the cricoid cartilage was a vertical sure, about half an inch long, communicating between the trachea and ophagus." In this case the disease of the cartilage may fairly be said have been secondary to the abscess.

Whenever the cartilage is exposed to the air, through ulceration of the perjacent tissues, it will be seen to be blackened or greatly discoloured. e cartilages most frequently affected are—firstly, the arytenoid; next, e cricoid; and, lastly, the thyroid.

There is a rare form of disease of the cartilages of the larynx, first scribed by Rühle, in which the cartilages are found to be increased in lume rather than diminished, and in place of ossification they undergo rous degeneration, and become quite soft. A case of this kind is ported by Dr. Morell-Mackenzie in the twenty-first volume of the *Patho-ical Transactions*, p. 58.

The **Symptoms** of necrosis of the cartilages are similar to those of ronic laryngitis and phthisis; but great pain is generally experienced, d there is almost always considerable dysphagia, and often extreme spnœa. *With the laryngoscope*, a slight swelling in the region of the fected cartilage is first seen. This swelling (generally of a semi-œde-atous character) gradually increases, and subsequently either pus slowly udes from its surface, or the pent up matter suddenly escapes. In those ses in which the external cartilages of the larynx are affected, pus forms the tissues of the neck external to the seat of disease. It rarely ppens that the necrosed cartilage is exposed during life. Hunter cords a case in which the cricoid cartilage was expectorated, and the tient subsequently made a good recovery; and several cases are on record which the arytenoid cartilages have been expelled during life.

Treatment.—The practitioner's treatment must be directed to the relief the pain, the dyspnœa, and the dysphagia. For the former, subcutaneous jections of morphia should be administered. The dyspnœa may be such to render tracheotomy necessary; and in cases where the dysphagia is treme (as nothing can be done to actually dilate the narrowed œsophagus) e patient must be fed by enemata or the œsophagus tube. The prefer-ble way, in cases which will admit of it, is to use an œsophageal tube with feeding-bottle attached. By means of an instrument of this kind, in one of the cases referred to, the patient's life was sustained for more than three months; and after death there was a considerable layer of fat covering the body. Of course, great care must be taken in passing the tube; but where practicable, this method of feeding is far more satisfactory in every respect than the use of enemata *per rectum.*

CONTRACTION OF THE LARYNX.

LATIN EQ., *Coarctatio;* FRENCH EQ., *Rétrécissement;* GERMAN EQ., *Verengerung;* ITALIAN EQ., *Contrazione.*

Definition.—*Narrowing of the passage of the larynx, the result of variou lesions.*

Pathology.—(*a.*) *Causation.*—This condition is most frequently due t tertiary syphilitic ulceration, and the contraction is caused by cicatrisatio and puckering of the mucous membrane. Cases have occurred in whic the glottis has become narrowed by adhesion of the vocal cords aft ulceration. In these cases there is generally a sort of web between t two cords, which, when divided, has a great disposition to form aga The contraction may be due to the chronic thickening of laryngeal phthi or to simple chronic ulceration. The canal of the larynx may also narrowed by the pressure of a tumor external to it. (See an interest pamphlet on this subject by Dr. Massei.*)

The **Treatment** of narrowing of the larynx resolves itself sooner later into tracheotomy. In cases of syphilitic cicatricial thickening, *io* *of potassium* does little good, and local treatment is of no avail. In chr tubercular narrowing, treatment is equally futile. In all cases, if dyspnœa is extreme, tracheotomy must be performed at once, and meas can subsequently be taken for dilating the contracted passage by an the mechanical devices which have been invented for the purpose.

BENIGN GROWTHS IN THE LARYNX.

LATIN EQ., *Polypus Laryngis;* FRENCH EQ., *Polype du Larynx;* GER EQ., *Larynxpolyp;* ITALIAN EQ., *Polipo della Laringe.*

Definition.—*New formations of benign character, forming projections mucous membrane of the larynx, generally giving rise to aphonia or dys often to dyspnœa, and occasionally to dysphagia.*

Pathology.—(*a.*) *Causation.*—Chronic congestion of the mucous brane is the most important etiological feature in the production of morbid growths in the larynx. The most common cause of hyper catarrh, which must therefore be looked upon as the great predis The various other influences, such as acute diseases, the inspir irritating vapours, particles of matter, and various occupations, I only act through establishing a condition of hyperæmia. Neither r phthisis, nor any constitutional condition appears to favour the growth these neoplasms. They are most frequent in the middle period of life. ey occur much oftener in the male than in the female sex; and are ecially to be looked for in persons following those occupations in which istant use of the voice is necessary. Benign growths are essentially of al origin, the result of a perverted nutritive process, in which growth is cessive and development imperfect. Dr. Morell-Mackenzie gives the lowing as the various kinds of benign growths in the larynx, enumerated their order of frequency—*Papillomata* and *benign epithelial growths*, *fibro-ta*, *fibro-cellular tumors*, *cystic tumors*, *fasciculated sarcomata*, *lipomata*, and *eiomata*.

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* "Sui Restringimenti Laringei," del Dottor F. MASSEI. Napoli. 187

Papillomata are by far the most common, and occur in about 70 per t. of all cases. They vary in size from a grain of mustard to a wal-, but they do not often attain the latter dimension, their most common being that of a large split pea. They may have a mammillated or liflower configuration. They are generally of a pink colour, but may white or bright red. "In general form and arrangement they have ny points of resemblance, but on an enlarged scale, to the papillæ, ich in various localities constitute natural projections from free surfaces, re especially from the skin and mucous membranes. Their basis-sub-nce is formed of connective tissue, which is continuous with that which mally exists in the part, whilst the free surface is covered by an thelium, which may vary in thickness and its number of layers accord-to the seat of the tumor. Blood-vessels, and even nerves, enter into interior of the papillæ."* Papillomata grow rather quickly, are erally situated on the vocal cords, and show a greater disposition to ur than other growths.

Benign epithelial growths may be considered as a sub-variety of papillo-ta. They constitute a very small proportion of laryngeal neoplasms. them epithelial scales do not clothe papillæ, but form continuous layers more or less undulating character. These growths generally occur in neighbourhood of the cords, and are of a white or pale red colour. In rly all cases of laryngeal growth, the epithelium is of the tesselated iety. Simple epithelial formations are not common, but occur most quently in children.

Fibromata are present in about 11 per cent. of all cases, and are seldom nd before adult life. Their rate of growth is very much slower than t of papillomata. Their structure is that of simple fibrous tissue, and y vary in size from a split pea to an acorn. They are usually of ooth appearance, round or oval in form, and of a bright red colour, and w no disposition to recurrence.

Fibro-cellular growths, or *mucous polypi*, consist of more or less perfectly eloped fibro-cellular tissue, and have serous-like fluid diffused through ir substance. They are semi-transparent in appearance, and resemble al polypi; but differ from them in showing no disposition to recur. The er kinds of growth are too rare to require any detailed description. eir structure is generally that of similar tumors in other parts of body.

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* *Lectures on Surgical Pathology*, by Sir JAMES PAGET, F.R.S., 3rd Ed., p. 591.

Symptoms.—The *subjective signs* of growths in the larynx may be sligh or very severe. A feeling of uneasiness, a desire to clear the throat, an very rarely, a slight pricking pain, are perhaps the only sensations experienced by the patient. In many cases these are altogether absent. O the other hand, there may be extreme dyspnœa, and even suffocatio When the growth is connected with the epiglottis, or with the posterio wall of the larynx, so as to press upon the food tract, dysphagia is th most prominent symptom. The most important *objective signs* are aphoni alteration of the voice, and dyspnœa (for this symptom is objective as we as subjective). There is almost always some modification of the voic Dysphonia is more common than aphonia. Out of 100 cases treated b Dr. Morell-Mackenzie, and analysed by him, there was modification of th voice in ninety-two instances, complete aphonia being present in fifty-fiv cases. Impairment of the voice was the *unique* symptom in no less tha fifty-two per cent. of all the cases (*Essay on Growths in the Laryn* Churchill, 1871, p. 19). *Cough* is not a frequent sign of larynge growths, occurring in only about 8 or 10 per cent. of Dr. Mackenzie' tables of all published cases. It is only in rare cases of a very violent o paroxysmal character. *Difficulty of breathing* depends entirely on the siz and situation of the growth. In the accounts of the specimens collecte in the various hospital museums in pre-laryngoscopic times, almost a the patients died from suffocation. The dyspnœa is not unfrequently o a severe and paroxysmal character; the attacks of suffocation generall occur at night; and it almost invariably happens that inspiration is muc more difficult than expiration. The general health is, as a rule, littl affected by the presence of benign growths, unless the difficulty of breathing has existed so long as to give rise to constitutional disturbance. *Th laryngoscope* at once reveals the presence of a growth in the larynx. The situation can almost always be ascertained by this means; but in a few cases, when the growth is very large the exact seat of origin may be hidden It may also happen that a growth in the upper part of the larynx ma conceal others situated lower down.

These various symptoms may develop themselves slowly, and, after time, remain stationary without causing serious inconvenience, unless a attack of catarrh or some other accidental circumstance arise. Amongs the lower classes patients will suffer from loss of voice for many year without seeking medical aid; but if dyspnœa or dysphagia occur, the will at once apply for relief. Some growths are exceedingly rapid in thei development, and may attain such a size in a few weeks or months as t occupy the larynx almost entirely.

Diagnosis.—Tumors of the larynx cannot well be mistaken for an other disease. In carcinoma the growth is, as a rule, more thoroughl blended with the surrounding tissues, and there is usually ulceratio while in phthisis there is a general thickening, not a defined growth, as the case of a true neoplasm. In both these diseases there is usually son degree of constitutional marasmus. In syphilis "false excrescences," t result of ulceration, are sometimes met with. Eversion of the ventricle a very rare occurrence; but when present it has very much the appearan of a laryngeal polyp.

Prognosis must be considered in relation to life and voice. T ndency to death in all cases of growths in the larynx is by suffocation; ut such a result should never occur in the adult, unless the patient refuses submit to tracheotomy. In children this termination is more likely to ke place, because the difficulties of diagnosis and treatment are much eater. In the young, also, the chances of recovery from the palliative eatment of tracheotomy are considerably less. A few cases are on record which growths in the larynx have been cured spontaneously by becoming parated and expelled by coughing; but it is scarcely necessary to remark at such a fortunate accident cannot be anticipated. A benign growth seldom fatal to life in the adult, if treatment be adopted; but in chil-en a more cautious opinion must be given. The presence of a growth the larynx of a young subject may at any time cause suffocation; and e only treatment which in some cases is expedient—viz., tracheotomy, in itself attended with much greater risk in young patients. With ference to restoration of voice, an opinion must be formed according to e size, situation, and nature of the growth.

Treatment.—In cases where the growth is small, does not appear to crease, and does not give rise to functional derangement, no treatment eed be adopted. Where the opposite conditions, however, exist, means ould at once be taken to eradicate the growth, or to place the patient ut of danger by performing tracheotomy. *Radical treatment* may be ivided into *internal*, that is, removal or destruction of the growth through e mouth with the aid of the laryngoscope; or *external*, by operations rough the neck. Internal treatment may be further subdivided into *echanical* and *Chemical.* Mechanical treatment consists in evulsion, by mmon laryngeal forceps, tube forceps, *écraseurs*, or guillotines. Added this, there are a few cases in which crushing or incision of the base of e growth is effectual. Chemical treatment consists in the application of arious caustic solutions,—a process seldom satisfactory—and in galvanic utery. This last method has been very successful in the hands of some ontinental practitioners; but it cannot be recommended, on account of e complicated and unwieldy apparatus required by the practitioner, and f the great pain often experienced by the patient. *External* treatment ay be divided into thyrotomy, or division of the thyroid cartilage, supra-yroid laryngotomy, or division of the thyro-hyoid membrane, and infra-yroid laryngotomy, or removal of the growth through the crico-thyroid embrane, or by an opening made in the trachea. In addition to these arious surgical procedures, there is the combined method, in which racheotomy having been called for, on account of the urgency of the ymptoms, the neoplasm is afterwards removed *per vias naturales.* All xternal measures are attended with less satisfactory results, both as to fe and restoration of function, than those resulting from laryngoscope reatment; and it should be borne in mind that the more serious surgical ethods *should never be resorted to unless the tumor in the larynx produces ymptoms endangering the life of the patient.*

APHONIA.

Latin Eq., *Aphonia;* French Eq., *Aphonie;* German Eq., *Stimmlosigke*
Italian Eq., *Afonia.*

Loss or at least modification of the voice, is such a prominent sympto in almost every laryngeal disease, that to describe it as a separate affecti would be not less unscientific than inconvenient. In so far as it depen on coarse structural changes within the larynx, aphonia has already be sufficiently dealt with in the preceding pages. But there remains a lar class of cases in which it is associated with some form of larynge neuroses, and for a description of these, the reader is referred to t following sections.

LARYNGEAL NEUROSES.

Neuroses of the larynx may be primarily divided into, (1.) Diseases the motor system; (2.) Diseases of the sensory system.

(1.) *Diseases of the Motor System.*

This division embraces "*Paralysis of the glottis,*" or more properly spea ing, *Paralysis of the muscles of the vocal cords*, and "*Spasm of the glottis,*" *Spasm of the muscles of the vocal cords.* This subject has been investigat by Gerhardt, Türck, Ziemssen, and others; but Dr. Morell-Mackenzie w the first to distinguish and analyse the various paralyses of the voc cords, and to point out the treatment suitable for each variety. The f lowing remarks on this subject are extracted from his *Essay on Ner Muscular Affections of the Larynx,* Churchill, 1868, to which the reader referred for fuller information.

Paralysis of the Muscles of the Vocal Cords.—The paraly affections of the several sets of muscles acting on the vocal cords may subdivided as follows:—(1.) Bilateral paralysis of the adductors. (Unilateral paralysis of the adductors. (3.) Bilateral paralysis of t abductors. (4.) Unilateral paralysis of an abductor. (5.) Paralysis the tensors, either bilateral or unilateral.

Bilateral paralysis of the adductors (*crico-arytenoidei laterales, and a tenoideus proprius*) is a condition in which there is "inaction of the addu tors on to the sides, preventing approximation of the vocal cords attempted phonation, and consequently giving rise to loss of voic This, the most common form of laryngeal paralysis, is known as nervo functional, or hysterical aphonia.

Pathology.—(*a.*) *Causation.*—The disease is essentially one in which t nerve-force is feebly or imperfectly evoked, or is not directed in the pro channel. Debility and hysteria are undoubtedly the most common cau of this disorder. It is often met with in the later stages of pulmon consumption, but occurs much less frequently in association with ute sturbance than is commonly supposed. Women are, however, far more bject to it than men, though it occasionally occurs in the male sex. Loss voice originating in hyperæmia not unfrequently remains as a functional enomenon after the congestion has disappeared.

Symptoms.—The essential symptom is loss of voice; but though the ower of voluntary phonation is lost, the reflex vocal acts are not generally fected, so that the cough, sneeze, and, to a less extent, the laugh, are companied with a distinctly laryngeal sound. With the laryngoscope is seen that on the attempted phonation of a vowel sound, as "*ah*," *a*," "*o*," the vocal cords do not approximate. The laryngeal mucous embrane is generally pale.

The **Prognosis** is very favourable.

Treatment should be directed towards stimulating the larynx so as to roduce a mild reflex spasm. Inhalations of *creasote*, and other pungent plications, may be used; but the most certain method of effecting this bject is faradisation of the larynx by means of the "laryngeal electrode," y which the electric current can be easily passed through the affected uscle. The most obstinate cases yield in the end to this treatment, and equently a few applications of the current are sufficient to restore the oice to its full power. Constitutional treatment of a tonic character is so indicated.

Unilateral paralysis of the adductors depends on "inaction of the dductors on one side preventing the approach of the corresponding vocal ord to the median line, and consequently giving rise to hoarseness or loss f voice."

Pathology.—(*a.*) *Causation.*—The condition may be due to chronic xæmia (lead, arsenic, diphtheria, &c.), may result from cold or muscular rain, or from disease of the nerves or nerve-centres presiding over the ffected muscles. Aneurism of the arch of the aorta, or of the right sublavian or carotid, may cause paralysis of the left or right adductor, but he really important muscular affection in such cases is the associated aralysis of the *abductor*. The disease differs from the bilateral paralysis f the adductors in never being of an emotional character.

(*b.*) *Morbid Anatomy.*—There is probably, in some cases, a special morbid ondition of some filaments of the recurrent laryngeal nerve, but the ctual lesion found after death is merely more or less complete atrophy of he affected muscles. The disease, in many cases, probably consists from he very beginning in idiopathic disease of the muscle; in other cases here is probably an inflammatory exudation, either of simple or dyscrasic haracter.

Symptoms.—The laryngoscope at once reveals the nature of the paraysis. On attempted phonation the affected vocal cord remains at the side f the larynx, whilst the healthy one is well adducted to the median line. ccasionally the mucous membrane is congested. There is aphonia or ysphonia, and the character of the cough, sneeze, and laugh are greatly ltered in tone. *This circumstance enables those not using the laryngoscope to istinguish this affection from functional aphonia or bilateral paralysis of the dductors.* There is not unfrequently slight dysphagia. In cases in which he paralysis is dependent on cerebral disease, there will be loss of power n the same side of the tongue and palate.

Prognosis.—Serious apprehensions need not arise when the conditi is due to local causes, but in those rare cases in which the disease cerebral the prognosis is serious. Cases due to blood-poisoning and cata are favourable for treatment, but the cure is often tedious.

Treatment.—Faradisation, as recommended in bilateral paralysis adductors, is the most appropriate treatment in those cases in which cause is local, but the affection is much more obstinate. The fara current must, in these cases, be applied more frequently, for a longer ti at each sitting, and must be continued for many weeks. Constitutio remedies may be used with advantage in cases of chronic toxæmia.

Bilateral paralysis of the abductors of the vocal cord (*crico-arytenoidei posti* depends on "inaction of the abductors on both sides preventing the o ward movement of the vocal cords on inspiration, and consequently givi rise to dyspnœa and stridulous breathing."

Pathology.—(*a.*) *Causation.*—The *cause* is sometimes cerebral, the affi tion being due to a diseased condition of the nerve-centres in the medu and pons, which preside over the motor functions of the larynx; morbid influences, which affect both pneumogastric or both recurr nerves, may give rise to it. Of these, bronchocele, scrofulous deposits the bronchial and cervical glands, and cancer of the œsophagus, are principal. It sometimes depends on simple degeneration of the musc without there being any evidence of implication of the nerves. T condition is rare. It consists essentially in a loss of power of the *cr arytenoidei postici*, caused either by injury or primary degeneration of muscles themselves, or by interception or non-generation of the ne current. After death the muscles are generally found to be considera atrophied.

Symptoms.—With the laryngoscope it is at once seen that the vo cords, instead of being drawn widely apart on inspiration, remain nea approximated—the opening of the glottis being in proportion to degree of the paralysis. The voice is not generally much affected, but may be slightly hoarse. The least exertion brings on stridor, and sleep the breathing is invariably stridulous. The cough is croupy. T affection may be mistaken for spasm of the adductors; but in spasm vocal cords are constantly varying in the degree of adduction, whilst the case of paralysis, the cords are never drawn back beyond a cert point.

The **Prognosis** is very serious, both on account of the immediate dan of suffocation, implied by the condition of the larynx, as well as on accou of the probable existence of some serious disease in the brain, or in neighbourhood of the pneumogastric nerve or its branches.

Treatment.—Medical treatment is of no avail, but when the breathi is at all seriously embarrassed, tracheotomy should be performed in cases without delay.

Unilateral paralysis of the abductor of a vocal cord may be defined "inaction of an abductor on one side, preventing the outward movem of the corresponding vocal cord on inspiration, and consequently givi rise to more or less dyspnœa and stridulous breathing."

Pathology.—(*a.*) *Causation.*—The causes are the same as those whi produce the bilateral form, but the condition now under consideration re often due to peripheral influences,—that is to say, to pressure on one eumogastric, or one recurrent nerve. Aneurism of the arch of the aorta, l other intrathoracic tumors, cancerous or glandular, not unfrequently volve the left recurrent nerve. Cases in which the right nerve is inved are rare. Cancer of the œsophagus in its last stage often affects recurrent nerve, and the same is true, though less frequently, of umous glands and tumors along the trachea. Unilateral paralysis of an luctor may also, like bilateral paralysis, be due to cerebral disease. here the disease is of any duration, the abductor of the affected side, en examined after death, is always atrophied, and in cases of long nding will often have undergone fatty degeneration.

Symptoms.—With the laryngoscope the affected cord is seen to remain ed in or near the median line when the patient takes a deep inspiran. Stridor, though not so severe as in the bilateral form, is a constant nptom. The voice is shrill. The constitutional symptoms vary accord; to the cause of the paralysis; but when the disease has existed for ne time there is usually more or less irritative fever.

Prognosis.—As this condition generally indicates serious disease elseere, a very unfavourable opinion must, as a rule, be given.

Treatment.—Little can be done curatively, but tracheotomy should performed, if the dyspnœa is at all urgent. In cases of aneurism of arch of the aorta, it is most important to ascertain whether the spnœa is due to direct pressure on the trachea, or to pressure on recurrent nerve. The laryngoscope at once clears up this point. here the symptom is due to nerve pressure, tracheotomy is indicated, ilst, if the dyspnœa be due to pressure on the trachea, it would be less.

In addition to the foregoing more evident and serious forms of parsis, there are others in which the *tensors* of the vocal cords (the cricoyroid and internal thyro-arytenoid muscles) are affected. The loss of wer is generally caused by exposure to draughts, or by a too violent or prolonged use of the voice; it occurs in singers, clergymen, and litary or naval officers. The most suitable treatment is faradisation, th complete functional rest.

Spasm of the tensors of the vocal cords is an affection in which the vocal rds being unduly and irregularly stretched, the voice is feeble, jerky, steady, and constantly rises to a high key.

Pathology.—(*a.*) *Causation.*—The condition appears to be due to connt perverted action of the vocal cords, arising from an artificial and ained use of the voice. The cerebro-spinal system does not appear to at fault; but the affection is simply one of spasm of the expiratory , in which the thoracic and abdominal muscles participate.

The **Symptoms** are those described in the definition. The varying ice distinguishes it from the constantly high pitched tone of paralysis.

Prognosis as to recovery of the natural voice is always unfavourable cases of long standing. There is, however, no danger whatever to e.

Treatment consists in complete rest of the voice for a lengthened riod, soothing inhalations, and the application of belladonna to the ynx externally. When the patient is allowed to use his voice again, he should receive instruction to speak slowly, and take a full breath short intervals, so that the pauses for rest are much more frequent th is usual. He should also be instructed to speak with a “full chest”- that is, with a good volume of air to set the vocal cords in vibration.

(2.) *Diseases of the Sensory System of the Larynx.*

Hyperæsthesia, or increased sensibility, occurring independently inflammatory disease or structural alteration of the tissues of the laryn is undoubtedly a rare morbid condition, but several cases have bee reported. The inhalation of hot sedative vapours and anæsthetics do good in some cases, and narcotics are also indicated. Neuralgic affectio should be treated on the ordinary principles which regulate the ther peutic management of such cases.

Anæsthesia.—Although there is a great difference between the sens bility of the glottis in different people, anæsthesia rarely occurs as distinct morbid affection. Disease affecting the origin, or trunks of tl pneumogastric nerves, or their superior laryngeal branches, would l likely to diminish the sensibility of the larynx in proportion as the fun tion of the nerves was interfered with. The condition is chiefly met wi in connection with diphtheritic and bulbar paralysis; but some degree impaired sensibility of the mucous membrane of the larynx has bee observed by Romberg in cholera.

SECTION III.—DISEASES OF TRACHEA AND BRONCHI.

CROUP.

LATIN EQ., *Angina Trachealis;* FRENCH EQ., *Croup;* GERMAN EQ., *Crou*
—Syn., *Häutige Bräune;* ITALIAN EQ., *Laringitide Membranacea.*

Definition.—*A “non-infectious inflammation of the mucous membrane of tl trachea, occurring in children, differing from other inflammations in like tiss in the presence of plastic exudation”* (BARCLAY); *or it is a disease accompanie by the exudation of a fibrino-albuminous material, which rapidly coagulates upo the mucous membrane of the epiglottis, glottis, larynx, or trachea, and sometim over all of these parts; indicated by accelerated, difficult, wheezing, or shri respiration; short, dry, constant, barking cough; voice altered by hoarsenes with spasm of the interior laryngeal muscles, and pain and constriction abo the sternum; frequently followed, towards the close of the disease, by expector tion of a membranous albuminous substance, or even of a cylindrical cast some portion of the breathing tube. The disease occurs in children, and ma terminate fatally, either in suffocation or exhaustion of the vital powers.*

Pathology.—(*a.*) *Historical.*—It has often excited surprise that a diseas so marked in its symptoms should not have been accurately describe before the middle of the eighteenth century, when Dr. Francis Hom published a treatise on the *suffocatio stridula* or *croup*, as it was observe in Leith, Musselburgh, and the vicinity of Edinburgh in 1765. It ha een described under the name of *cynanche trachealis;* and Dr. Farr pro-osed for it the name of "*trachealia.*" The name of *croup*, by which this isease has hitherto been known in this country, is of Scottish origin. ullen's *cynanche trachealis*, and the more modern *tracheitis*, are objection-ble terms, because they lead to false notions of the pathology of the isease. The "choak," "stuffing," "rising of the lights," and "hives," re all designations by which the disease has been described, and some of hem are still names in vogue amongst the common people of the country. efore the time of Dr. Home, however, there is reason to believe that the isease was confounded with other affections of the throat and chest, esulting simply from exposure to cold. It was certainly described and istinguished by Martin Shisi, in 1749, at Cremona, and by Starr, of iskeard, in Cornwall, in the same year (*Phil. Trans.*, 1750). Many hysicians have described the disease since that time, and none with more inuteness than Dr. Cheyne, of Leith, who observed it for several years, nd illustrated its pathology by careful dissections.

(*b.*) *Causation.*—The disease is peculiar to infancy and childhood. The isposition to it is least during the period of suckling; and after the econd dentition cases are rare. The period of life between the second nd seventh years furnish the greatest number of cases. Vigorous fleshy hildren, with rosy complexions, are frequently those who suffer most. t is more frequent in cold and moist climates than in those which are armer. It is also much more severe in Europe than in America; and ts existence and progress are considerably influenced by changes of season, eather, and temperature. It is prevalent in Switzerland and Savoy, in he eastern counties of England and Scotland, the north-west countries of urope, and in the northern parts of America. The annals of medicine re rich in descriptions of epidemic and endemic croup; but opinions re divided as to the nature of the epidemic influence, and whether or not he disease is contagious or infectious. It seems now, however, evident hat what has been described as epidemic croup is really diphtheria.

(*b.*) *Morbid Anatomy.*—The most remarkable pathological phenomena of roup are to be seen in the exudative process which attends the inflamma-ion in the windpipe, and the formation of a false membrane, almost eculiar to children, but sometimes seen in adults. The disease derives s importance from the tendency of the inflammation to attach itself to he opening of the glottis. In childhood the *trachea* is the chief seat of he inflammation; and when the *larynx* and the *fauces* are involved, they re so secondarily, and to a less degree. The croupous exudation is a *brino-albuminous* one, which rapidly coagulates when it appears upon the ee surface of a mucous membrane, involving the epithelium only, so that hen the croup-membrane is detached, the epithelium is quickly re-roduced. No loss of substance occurs in the mucous membrane itself, nd no scar remains after the membrane is removed or disappears. On the ther hand, as Niemeyer points out, the diphtheritic process is characterised y the production of a similar *fibrino-albuminous* and rapidly coagulable xudation; but differs from croup in the exudation forming not merely *pon* the surface of the mucous membrane, but *within* its substance. It filtrates the mucous and submucous tissue, and this interstitial exudation the swollen elements of tissue, exerts a pressure upon the blood-vessels which results in sloughing (diphtheritic) of a portion of the inflamed mucous membrane. A diphtheritic eschar is the result, and on its separation there is loss of substance and a consequent cicatrix. Microscopically, the membrane of croup consists of amorphous or finely fibrillated fibrine, in which numerous young cells have been entangled during the process of its excretion (NIEMEYER). When death takes place after an illness of four or five days, the windpipe is found to be lined with a white or grey membrane, varying much in thickness and consistency. This false membrane more commonly embraces the entire circumference of the trachea, forming a complete hollow cylinder adapted to the walls of the tube. The membrane is in most instances limited by the larynx above, but in some cases it extends down the trachea to the bifurcation, while in a very few cases it reaches even to the minutest branches of the bronchi. The membrane thus formed is in a few instances removed by the cough; but more generally it adheres with so great tenacity that it can only be detached by a thinner and more serous secretion taking place from the membrane beneath it, which loosens and displaces it. In childhood it is almost exclusively a disease of the trachea, and sometimes of the larynx, and rarely affects the alveoli of the lungs. On the other hand, croupous pneumonia is a common disease of adults, in whom primary croup of the trachea and larynx scarcely ever occurs. The place first and most particularly affected is the upper part of the *trachea*, about an inch below the *glottis*. In that part patients complain of a dull pain. External swelling has been observed there; and the morbid membrane is found spreading thence downwards. The back part of the trachea, where there are no cartilages, seems to be its first and principal seat (HOME). According to Guersent, false membrane is never entirely absent from the larynx. Sometimes it is confined to the glottis, and sometimes lines the whole interior of the larynx, including the ventricles; and not unfrequently it extends throughout the trachea, and, for a greater or less distance, into the bronchial tubes.

Symptoms and Course.—The mildest form of croup differs from an ordinary catarrh only in the addition of spasmodic symptoms. Spasmodic action of the laryngeal muscles is common to both and is characteristic; but the inflammation and exudation are not in general more severe at first than that which attend a common catarrh; and are ushered in by sore throat, by catarrhal symptoms, or by a short dry cough. It may occur without the general health being sensibly impaired. In either case the attack commonly takes place during the night, the sleep of the child, which was perhaps more or less agitated, being interrupted by fits of *hoarse* coughing. These become more frequent, the respiration more difficult, and marked by a peculiar wheezing, which has been described as like the sound of an inspiration forcibly made with a piece of muslin before the mouth, or like to the sound of air passing through a brazen tube. The little patient feels a sense of restriction about the throat, as shown by carrying the hand often to it, and grasping the larynx. After the paroxysm has lasted some hours, there is an interval of ease, which perhaps lasts for some hours. By the end of the second day, sometimes sooner, the tongue becomes white, the heat of the body increased, the pulse frequent, the face flushed, and the countenance distressed. From this point the disease apidly advances, the croupy sound attains its height, and Dr. Home lescribes it as "*vox instar cantus galli;*" others have compared it to the oise which a fowl makes when caught in the hand; while the child often uts its fingers into its mouth, as if to pull away something which bstructs the passage. As the disease draws to a close the paroxysms ecome more frequent, the cough more severe and with less sound, the ulse more rapid, suffocation more imminent, and the extremities cold nd livid. Death may take place by convulsions, sometimes almost etanic.

Sometimes the symptoms are much more moderate; indicated by a uskiness of the voice, till no sound can be heard above a whisper, by a uffled cough, and a wheezing noise which attends inspiration. It is eldom that children expectorate; but mucus tinged with blood is oughed up, and later, perchance, the false membrane is detached and hrown up, and the patient recovers. The croup which has been described s of the most acute kind; but in many cases its course is much more hronic, the symptoms generally milder, and the intervals of ease longer nd more complete, during which the breath is free, the child cheerful, nd the appetite good. In the course of a few days, however, a violent aroxysm seizes the child, and destroys him with every appearance of one trangled. On applying the stethoscope to the larynx, a sort of "*tremblote-ent*" is heard, as if a movable membrane was agitated by the air—an nerring evidence of the existence of a false membrane in the larynx BARTH).

Diagnosis.—It is generally between croup and the following diseases—amely, the different forms of sore throat, as in *scarlet fever* and *measles*, *iphtheria*, *bronchitis*, *chronic laryngeal and tracheal inflammation*, and *hooping-ough;* and the distinguishing symptoms of each of these from croup must e studied by comparing the symptoms and course of each of these diseases, s well as their epidemic characters. In *diphtheria* there is a specific fever, nd the lesion spreads from above downwards, or may commence in the arynx (Vol. I., p. 746). In croup the lesion commences in the trachea, nd spreads upwards. Hence the two diseases have often been and are pt to be confounded together. As to acute laryngitis, that is a disease f adult life, and croup is a disease of childhood. Laryngitis in the adult s marked by the same difficulty of breathing, the same lividity of countenance, the same constriction of the throat, the same paroxysmal attack, nd by the same exemption from any severe constitutional affection. The voice, however, instead of being sharp and shrill, is generally deep nd hoarse, although sometimes altogether lost—differences depending erhaps on the greater size of the glottis, and on the fact of the parts eing the seat of ulceration rather than of the effusion of lymph.

Every now and then discussions arise as to whether or not croup exists s distinct from diphtheria: and the essential duality of the two diseases as of late been again in dispute. Any one who has seen much of croup n children can have no difficulty in recognising it as a disease very lifferent from diphtheria in its attack, its course, and its results. Croup nd diphtheria are distinct diseases. A transition from croup to diphheria is, however, not unfrequent, as Niemeyer also states, when the nalady forms part of, or occurs during the course of, such acute infectious diseases as *measles, small-pox, typhus, scarlet fever*, or during an epidemic of diphtheria.

Prognosis.—"Is never better than doubtful;" and the duration of this disease, when acute, is short. It is to be determined from the violence of the local symptoms, and the frequency of the paroxysms, rather than from the constitutional symptoms. Children, however, seized with croup are said to recover in a smaller proportion in this country than in America. Death tends to occur by *apnœa.* It may terminate in twenty-four hours; more frequently, however, the child lives to the third or fourth day, and in chronic cases much longer. From one day to one or two weeks may be given as the variable periods of the duration of this disease.

Treatment.—*Every case of croup demands the most active, efficient, and energetic treatment.* Great and diametrical as are the pathological differences of croup and diphtheria, the treatment most effectual in either is not the less decided and opposite. While *calomel* in purgative doses, *tartar emetic,* and *blood-letting* may each and all of them be demanded in croup, these agents would be most baneful in diphtheria, which is most successfully combated by the administration of the *mineral acids* (especially the nitric and hydrochloric), *chlorate of potash, quinine,* and *iron* (Vol. I., p. 763). *Bleeding*, especially local, should be employed in most cases to a considerable extent (an ounce of blood for every year of age); and two to twelve leeches, according to the age of the patient, should be applied over the trachea and larynx. The bleeding should be encouraged by the application of a linseed poultice to the throat. This first bleeding often gives great relief, and sometimes averts the danger. If not, the leeching after a few hours may be repeated. As soon as some relief is obtained, a blister should be applied along the lateral aspect of the neck on each side, and not over the trachea. The blistered surface should be dressed with mercurial ointment. Many practitioners prescribe *emetics* also; first, because their emetic effects, and the large evacuations they produce, favour the resolution of the inflammation; and second, because the effort of vomiting may be the means of detaching and of expelling the false membrane, should it have formed. So soon as croupy cough and dyspnœa occur, an emetic of *ipecacuanha* with *tartar emetic* ought at once to be given, in doses suited to age. Four to six grains of *ipecacuanha*, combined with a quarter or a third of a grain of *tartar emetic*, will be sufficient for a child of two or three years of age. The action of the emetic may be aided by a warm bath of 98° to 100° Fahr. If it becomes obvious that the exudation has assumed the form of a membrane, especially if indicated by a diphtheritic coating over the fauces, a solution of the nitrate of silver, varying in strength from forty to one hundred and twenty grains to the fluid ounce of distilled water, should be applied to as much of the fauces and larynx as can be reached. A sponge on the end of a piece of whalebone should be loaded with the weaker solution, and squeezed against the *rima glottidis* two or three times a day. Bleeding has no effect in removing or modifying the false membrane; but the system must be brought as speedily as possible under the influence of mercury. *Two, three,* or *four* grains of *calomel,* with *two* or *three* grains of *James's powder,* may be given at short intervals every two or three hours; and a dose of astor-oil occasionally, till the full effect of the *calomel* as a purgative is btained. Green fæcal stools, like chopped spinach, are characteristic of his result. Mercury appears a powerful resource in these cases; and, ntroduced either internally or by inunction, so as to affect the mouth, but vithout inducing salivation, uniformly gives relief as soon as its contitutional action is established.

Bleeding, *blistering*, and *mercury*, although the rule of treatment in diopathic infantine croup, are, for the most part, entirely inefficient in hose cases in which the affection begins in the fauces, as in the case of nany epidemics, especially after scarlatina, which are really cases of liphtheria. *Expectorant medicines* should be given with the mercurials, nd be continued after them. *Ipecacuanha* and *senega* are most efficient.

Tracheotomy, when not too long delayed, has saved many a life in roup, while it has proved utterly abortive in diphtheria, which is beyond he reach of any such topical remedy. M. Trousseau has saved one-third of his patients by its means; and of twenty cases Bretonneau saved six. Perhaps the experience of the profession generally is somewhat discordant on this point at present. Those who operate early save some of their patients, while those who wait till a case is advanced, and beyond medical reatment, before they resort to this measure, for the most part lose all heir patients. The evidence, however, is daily accumulating which shows *hat tracheotomy ought to be resorted to much oftener as a remedy for croup than it has hitherto been, and that at a much earlier period in the disease,—not as a last resource, when death from asphyxia appears imminent, and after treatment of the most depressing kind.* That this is the secret of success in France and in this country, is shown by the experience of able physicians and good surgeons, of whom the names of M. Trousseau, the late Mr. Jones, of Jersey, Mr. Henry Smith and Dr. Fuller, of London, the late Dr. Cruickshank, of Dalmellington, in Scotland, and Mr. Spence, Professor of Surgery in the University of Edinburgh, Dr. George Buchanan, in Glasgow, and Professor Roser, of Tübingen, may be stated as authorities by experience. In country districts the performance of tracheotomy in a case of croup is almost imperatively called for in the majority of cases, *if some symptoms of amelioration do not follow the steady use of bleeding, emetics, the warm bath, and calomel purgation, pursued for twelve or sixteen hours.* I know from personal knowledge in a wild country district of Scotland, where croup was very common and fatal, that the late Dr. Cruickshank saved eight out of eleven cases during two years. A valuable paper by Mr. Smith, in *The Medical Times and Gazette*, January 26, 1856; another by the late Mr. Jones, of Jersey, in the 8th November of that year; and, lastly, a paper by Dr. Conway Evans, in the *Edinburgh Medical Journal* for January and May, 1860, go to support the same conclusion,—namely, *that an earlier introduction of air, by the operation of tracheotomy, for croup, would not only give a larger percentage of recoveries in this country, but would place the operation in the same favourable light in which it is now regarded in Paris and other parts of France.* Tracheotomy in croup is undoubtedly gaining ground; and it cannot be denied that children perish, in the first instance, almost always from suffocation. *Tracheotomy* is therefore indicated in croup as soon as there are urgent symptoms of obstruction of the glottis. When the respiration is so impeded that the demand for oxygen is only satisfied by difficult forced respirations, dreadful anguish is depicted on the reddened countenance covered with sweat; there is extreme restlessness; the patient tosses from side to side, gets out of bed one minute and into it the next, clutching spasmodically at those around him, as if seeking everywhere for help. *This is the proper period for the operation of tracheotomy in croup,*—the time when success may be expected (ROSER). Should the operation be longer delayed, symptoms of asphyxia appear, overloading of the blood with carbon ensues, the face suddenly becomes blue, with fixed and staring eyes, convulsive exertions are made, and anxious struggles for breath follow the stage of suffocative agony. In some cases the symptoms of asphyxia come on more slowly, and are apt to make considerable progress before the danger is fully appreciated. This insidious form of asphyxia is denoted by symptoms of great weariness and weakness, restlessness, oppression, anxious startings out of short slumbers, loss of consciousness and of feeling, approaching stupor, the face pale, and tending to become œdematous. If tracheotomy is delayed till these symptoms become expressed, it may still enable the child to breathe more freely, and thereby may promote the chances of recovery; but the child has usually no longer power to resist the advancing bronchitis. The causes of death after the operation are mainly pneumonia, bronchitis, or the severity of the constitutional febrile state (*Syden. Society Year-Book*, 1863, p. 278). In diphtheria the fatal result depends on a cause acting generally on the system, which ultimately destroys the patient, and which tracheotomy will not prevent (Vol. I., p. 763). Age influences success to a considerable extent. Under *two* years of age few cases recover; but between the ages of *six* and *twelve* nearly one-half are saved (CONWAY EVANS).

BRONCHIAL CATARRH.

LATIN EQ., *Catarrhus Bronchiorum;* FRENCH EQ., *Catarrhe Bronchique;* GERMAN EQ., *Bronchialcatarrh;* ITALIAN EQ., *Catarro Bronchiale.*

Definition.—*Infiltration and œdema of the mucous membrane of the bronchial tubes, so that tumefaction diminishes their calibre, attended first with dryness of surface, followed by a watery secretion, which subsequently becomes turbid and yellow. The disease is sometimes attended with chilliness and the discomfort of what is called a "common cold."*

Pathology.—(*a.*) *Causation.*—Certain conditions are known to predispose towards bronchial catarrh, namely:—(1.) Childhood and advanced age. (2.) Badly fed flabby individuals, the walls of whose capillaries are probably weak, running through soft and yielding tissues, with a tendency to hyperæmia and increased transudation. The anatomical constitution associated with scrofula, gout, and rickets is of this description. (3.) A previous attack predisposes to others. (4.) Effeminate modes of life, relaxing and enervating habits. The directly exciting causes are—(1.) Impeded evacuation of the bronchial veins, as in cardiac diseases. (2.) Obstruction to the current of blood through the great branches of the aorta below the origin of the bronchial arteries, such as may be caused by the compression of liquid in the peritoneum, accumulation of excrement or of gas in the intestines. (3.) Irritants acting directly on the mucous membrane, such as dust, vapours, hot and cold air. The irritant materials from certain trades, such as that of bakers, millers, and stone-cutters. (4.) Chilling of the skin and the influence of sudden changes of temperature, as sitting in a draught of air when perspiring freely. Children suffer, especially when the legs and lower parts of the body are scantily clothed or left unprotected. (5.) Morbid states of the blood, as when bronchial catarrh is a premonitory symptom of enteric fever, measles, and small-pox, in which the catarrh depends on a specific animal poison, whose material presence circulates in the blood, and whose irritation acts on the mucous membrane, as in gout, rheumatism, and syphilis; and during the administration of certain medicines, such as iodine.

Symptoms are generally combined with catarrh of the *larynx*, of the *nasal mucous membrane* (*coryza*), and of the *frontal sinus* and *conjunctiva*. It rarely spreads to the smaller bronchi. Pains "all over," aching of the joints, limbs, and back, with a sense of tightness across the forehead, and chilliness, usually usher in a "common cold." Then follow discharge from the nostrils, at first watery, with copious flow of tears, hoarseness or rough voice, dryness of throat, furred tongue, thirst, loss of appetite, and quickened pulse.

Treatment.—When the symptoms of a "common cold" first express themselves, when the attack is sudden, attended with marked depression, aching pains in the head, back, and limbs, and febrile disturbance (and even when the sensations have extended to the chest, as indicated by the hoarseness and tendency to cough), the disease may be at once subdued in a healthy person by a full stimulant (but not narcotic) dose of *opium* or *morphia*. "One-third or one-fourth of a grain of *muriate of morphia*, or one grain of *opium*, ought to be taken at bedtime. Its influence will begin to be felt in from twenty minutes to half an hour, by the gradual disappearance of the sense of intense weakness, the relief of the pains, and that peculiar feeling of thorough and evenly distributed warmth of the whole body which is so different from fever on the one hand, or chilliness on the other" (ANSTIE, *op. cit.*, p. 120). An alcoholic diaphoretic drink; or *five* grains of *carbonate of ammonia*, or *ten* to *twenty* grains of *muriate of ammonia;* or, if the appetite is unimpaired, a full supper, followed by a hot alcoholic stimulant, may have a similar effect to opium. Natural sleep ought to supervene, and the morning ought to find the patient well. The following formula has been recommended as an *anticatarrhal olfactory*, originally prescribed by Dr. Hagner; ℞ *Carbolic acid*, 5 parts; *rectified spirit of wine*, 15 parts; *strong solution of ammonia*, 5 parts; *distilled water*, 10 parts. The mixture is kept in a stoppered dark glass bottle. When a catarrh is commencing, a few drops are placed on three or four layers of blotting or filtering paper; the patient holding this in his hand, and closing his eyes, inhales deeply from it as long as any smell is perceptible. The effect is to cut short the acute stage of the cold, to prevent the occurrence of subsequent coryza and bronchial and laryngeal catarrh; while all troublesome symptoms are rendered much milder. The remedy should be applied every two hours (Dr. E. BRAND, *Wiener Med. Wochenschrift*, June 15, 1872). Snuffs composed of *morphia* and *subnitrate* of *bismuth* are also found useful when *coryza* is present—*e. g.*, *muriate of morphia*, gr. i.; *powdered gum arabic*, ʒi.; *subnitrate of bismuth*, ʒiii.; and *oxide of zinc* may be added in the proportion of 40 grains. This compound powder is to be repeatedly and thoroughly snuffed into the nostrils.

If such remedies are delayed too long, the object next to aim at is to induce a copious perspiration and a continued action of the skin and kidneys, by small doses, frequently repeated, of *antimonial* and *ipecacuanha wines*, or *nitrate of potash*, or *acetate of potash*, as well as *bicarbonate of potash* and *aqua potassæ*, combined at a later period with tinctures of *squills* and *hyoscyamus*. In the early stage of the disease the bronchial membrane has its normal moist condition altered to a dry state; and the object of treatment is to bring about a return of the naturally moist condition. This is best effected by the inhalation of the vapour of hot water, the use of *tartar emetic* in doses of *one-twelfth* to *one-sixth* of *a grain*, the use of *ipecacuanha* in *one-quarter or half-grain doses.* From the frequent inhalation of steam great benefit is derived. *Opium* has often been regarded as hurtful in the first stage, because it has been taught that it interferes with the free secretion of mucus, so as to render expectoration difficult. But the onset of the catarrh—*i. e.*, free expectoration, is in consequence of the abatement of the inflammation, which opium tends to subdue, and in this respect it acts as an expectorant. Thus it is often of great service in the first stage of a common cold, as it is in most acute inflammations (FLINT). *The inhalation of iodine vapour* every three or four minutes for an hour is of service, each inhalation lasting one minute, by merely holding a bottle of *tincture of iodine* in the warm hand under the nose (TANNER). Total abstinence from liquids for forty-eight hours, originally recommended by Dr. Richard Lower one hundred and forty years ago, is a practice recently revived by Dr. C. J. B. Williams. *Muriate of ammonia*, ʒss. in solution, is a remedy also much in use, combined with *liquorice* or *simple syrup*, with one grain of *tartar emetic* and one or two drachms of *antimonial wine*, dissolved in six or eight ounces of water. Of this a table-spoonful for a dose, frequently repeated, may induce relief, when the discharge from the nose and trachea is viscid. Copious warm drinks and abundant warm bed-clothing, to induce diaphoresis, are efficient remedies to be used early.

The object aimed at in the treatment of a "common cold" is to establish a free catarrh—to soften the expectoration and make coughing "loose" and easy. Niemeyer lays down some practical rules, based on symptomatic indications, to accomplish these ends:—(1.) In chronic colds or catarrhs, *where cough is incessant and distressing, and out of proportion to the expectoration*, narcotics are indicated—such as ten grains of Dover's powder at bedtime, or the *camphorated tincture of opium* (*paregoric elixir*). External counter-irritants are also of service, in the form of mustard poultice, "mustard leaves," or blistering substances, short of vesication such as stimulating liniments. *Chlorate of potass* is also a useful remedy (2.) *Dyspnœa, with extensive sibillant rhonchus and irritability of the mucou membrane*, are also relieved by narcotics, combined with *ipecacuanha*, o with *tartar emetic.* But narcotics are contraindicated if, owing to th feebleness of the patient, the efforts of expectoration are inadequate t prevent accumulation in the bronchial tubes. *Alkaline carbonates* posses the property of thinning and rendering easier of expectoration the viscid bronchial secretion, and are on that account to be recommended, especially where the condition of the urine suggests such remedies. *Opium* in its many forms is the most useful remedy, so long as the first stage of inflammation exists, if care be taken to watch that nothing contraindicates its administration. Preparations of *conium*, *hyoscyamus*, or *hydrocyanic acid*, must be used in cases where opium is contraindicated.

(3.) When *secretion is profuse, with relaxation of the mucous membrane of the bronchi, when large, soft, moist râles are heard, stimulant expectorants are indicated,—senega, squills, carbonate of ammonia, benzoin, gum ammoniac,* and *oil of cubebs.* Remedies in the form of gas are also of service, such as the *fumes of tar*, by boiling it alone or mixed with water; also *vapour of turpentine*, half a drachm being put into a bottle of hot water, the patient inhaling for a quarter of an hour, four times a day. In all cases absolute rest, vegetable diet, demulcent drinks, and small doses of antimonials will greatly tend to bring about relief. A pint of any demulcent fluid (such as linseed tea), with two drachms of *antimonial wine*, taken in small quantities, so as to be consumed in twenty-four hours, will be found a good standard prescription. *Gum arabic*, in the proportion of *one ounce* to the pint of water, or decoction of *marsh mallow*, or of *dried fruits*, is a good substitute for the *linseed*, and may be flavoured with anything agreeable to the taste of the patient, such as orange or lemon syrup. *If much fever exists*, a very hot foot-bath, just before going to bed, has a soothing effect. The air of the bed-room must be kept at about 60° Fahr., not below, and the patient must remain as quiet as possible.

Generally, in prescribing *cough mixtures*, which ought if possible to be avoided (seeing that they so impair the functions of the stomach), the following conditions ought to be adhered to in devising the mixture:—(1.) That the prescription should be in the form of *fluid* remedies,—solution, or infusion of wines or syrups of the drugs. (2.) That the dose should not exceed one teaspoonful. (3.) That the proportions contained in the mixture should be so arranged that a dose may be given every two, three, four, or six hours, according to the urgency and acuteness of the symptoms. Such a mixture may contain in each dose, for an adult, from ten to thirty minims of *antimonial wine*, or of *ipecacuanha wine*, or of *tincture of sanguinario*, or of *syrup of squills*, or of *vinegar of squills*, with *two minims* to four minims of *hydrocyanic acid*, or thirty to sixty minims of *tincture of hyoscyamus* or of *conium;* or from two to four minims of *tincture of opium;* or from thirty to sixty minims of *camphorated tincture of opium.*

SECTION IV.—DISEASES OF THE LUNGS.

The acute inflammatory diseases of the lungs are more or less defined by the tissues which they implicate and the symptoms to which they give rise. The structures which mainly compose the lung-substance are—(1.) The *bronchial tubes*, terminating in (2.), *the pulmonary air-cells*, *vesicular* or *alveolar structure*, *proper substance*, or *parenchyma of the lung;* and, (3.) *the membrane covering the lungs*, forming a portion of that serous sac interposed between the surface of the lungs and the walls of the thorax.

That portion of the serous sac which immediately invests the lungs is known as the *pulmonary pleura;* that which is applied against and invests the parietes of the thorax is known as the *parietal* or *costal pleura.* One or more of these structures may be concerned in the processes and results of inflammation. The bronchial membrane may be inflamed, when the disease is termed *bronchitis;* or the substance of the lung may be inflamed, the disease being then called *pneumonia;* or the pleura may be inflamed, a condition which is described as *pleuritis.* But it is rare to find that these morbid states are always so completely isolated. More frequently, with *pneumonia* there is associated inflammation of the air-tubes (*bronchitis*), constituting *broncho-pneumonia;* or there co-exists inflammation of the investing pleura (*pleuritis*), constituting *pleuro-pneumonia.* Each of these conditions can now be distinguished by definite symptoms; and by observing the combination of physical phenomena, their association with general symptoms, and the sequence of their occurrence, the physician is able to determine how far any pulmonary affection involves one or more of the structures which compose the lung, and to direct the treatment accordingly. It is therefore necessary to describe the phenomena of inflammation in each of these structures in detail. The College of Physicians has not recognised in their nomenclature the combined lesions just referred to as distinct diseases (see Vol. I., p. 343).

BRONCHITIS.

LATIN EQ., *Bronchitis;* FRENCH EQ., *Bronchite;* GERMAN EQ., *Bronchitis;*
ITALIAN EQ., *Bronchitide.*

Definition.—*Inflammation of the air-passages leading to the pulmonar vesicles, characterised by hoarseness, moderate cough, heat, and soreness of th chest anteriorly—symptoms which are more and more intense according to th severity of the disease, and especially the extensive implication of the smalle tubes. The natural mucous secretion is at first arrested, but subsequently becomes increased in amount and altered in quality, tending to assume th corpuscular character.*

Pathology.—(*a.*) *Causation.*—The causes are similar to those which s up a "common cold," already referred to. But the more severe forn of bronchitis, as distinct from a common cold, acknowledge more vari causes. Especially is bronchitis often a complication or result of variou febrile disorders, such as the exanthemata; but it is also a comm attendant on various diseases of the heart and kidneys, on gout, tuberc losis, and syphilis. It is influenced also by temperature and seas being prevalent and fatal in autumn and winter.

The following are forms and results of bronchitis which may clinically recognised :—(1.) *Acute bronchitis,* (a.) *of the larger and mediu sized tubes;* (b.) *Capillary bronchitis, and of the tubes generally—the "p pneumonia notha" of the older writers;* (2.) *Chronic bronchitis;* (3.) *Pla bronchitis;* (4.) *Dilatation of the bronchi* (*bronchiectasis*); (5.) *Mechan bronchitis,* such as knife-grinder's disease—*carbonaceous bronchitis,* or bl phthisis; (6.) *Bronchitis secondary to general diseases*, such as typhoid fever; (7.) *Bronchitis secondary to blood diseases;* (8.) *Syphilitic bronchitis.*

Capillary Bronchitis has been regarded as most common at the two extremes of life—infancy and old age,—its occurrence in youth or adult age as exceptional, and then only amongst the weakly. It is more common in adults of both sexes than is generally admitted, and is not limited to those of feeble health or the infirm from previous disease. It may occur in those of vigorous and sound constitution. It is sometimes met with as a secondary or intercurrent affection in the course of exanthemata in children, particularly of measles (*Broncho-pneumonia* of Trousseau); and it has been described as lobular pneumonia when coincident with collapse of the lobules.

Chronic Bronchitis, or *Chronic Bronchial Catarrh*, is the result of a frequently recurrent and protracted bronchitis, or the attendant upon chronic heart disease. It may occur habitually once or twice a year (spring, autumn, or both), at first the patient remaining exempt during the summer, till at last the symptoms and the catarrh are more or less constant all the year round.

Plastic Bronchitis, in which false membranes or casts of the bronchial tubes are sometimes expectorated, is most common in middle age, and happens oftener in males than in females. The health in some instances is excellent until the sudden onset of the disorder; but generally it occurs in persons of delicate constitution who have suffered and are suffering from pulmonary disease, *inherited* strumous diathesis, a liability to cutaneous affections, to asthma, or hæmoptysis. It is not unfrequently associated with rheumatism, scurvy, or gout; or been referred to the pressure of tumors, malignant or benign, upon the lung-tissue; or to the presence of aortic aneurism. I dissected a case in which an aneurism pressing on the trachea and lungs proved fatal (but not by rupture), in which the whole ramification of the bronchial tubes were occupied by an exudation of a reddish lymph, which had formed a continuous coagulum of the consistence of jelly. The nature and cause of the disorder are to be sought for, perhaps, in some specific morbid process which still requires investigation.

Bronchiectasis may arise quite independently of all other pulmonary affections; but there are varieties which result from changes in the lungs, caused by *hooping-cough; suffocative capillary bronchitis; stricture of bronchi; long-standing indurations of lung-substance, tubercular or inflammatory; the remains of chronic tubercular cavities,* or *abscesses in the lung-tissue.* It is the result of traction of the thickened interlobular septa upon the bronchial wall (D. J. HAMILTON). It is met with at all ages, but is most frequent about and after forty. Pulmonary emphysema is found in a large proportion of the cases.

(*b.*) *Morbid Anatomy.*—When the chest is opened in cases where the finer or capillary bronchial tubes have been obstructed by mucus, the lungs do not collapse as they ought to do in health. They bulge out of the opened thorax with considerable force, conveying the idea that they were too bulky for the cavity which contained them. They do not collapse, on account of the obstructed bronchi preventing the escape of air from the alveoli. Bronchitis may affect one lung, both lungs, or part of a lung, and the upper lobes are more commonly affected than the lower ones. The larger bronchi are more commonly inflamed than the smaller ones. Hence the more marked primary effects of bronchitis are most obvious in the bronchi towards the roots of the lungs; while the secondary effects (such as *vesicular emphysema*), which are apt to follow a prolonged or severe attack, are mostly developed towards the pleural surfaces of the lungs at the anterior margins. The results of the inflammatory process in the mucous membrane lining the bronchial tubes are peculiar to the texture of the part affected and the dimensions of the tubes. It is attended with changes in their epithelial covering, and in the secretion from the mucous glands. In diffuse bronchitis the inflamed portions of the mucous membrane are of a deep venous red, which may be general or partial, in spots, streaks, or arborescent forms, the result of hyperæmia and of ecchymosis. The more asthenic the inflammation, or the more feeble and cachectic the patient, the more livid, purple, or cyanotic is the redness, and the greater the ecchymosis. The mucous membrane is softened, swollen, and easily torn, the result of an œdema which reduces the calibre of the tubes. The secretion, at first arrested, is eventually increased in quantity and altered in quality. It becomes thin, watery, and frothy, and subsequently thicker and more consistent, assuming the appearance of pus. Young epithelial cells, loaded with serous effusion, and losing their vital cohesion with the basement membrane, are rapidly and easily discharged, constituting the thin, frothy, serous expectoration of bronchitis in its early stage. It subsequently becomes tenacious, more opaque, and pus-like. Occasionally it is of a very fetid character, so as to simulate gangrene of the lungs. An instance of this description (with other analogous cases) was carefully described by the late Dr. Laycock, Professor of the Practice of Medicine in the University of Edinburgh. A chemical analysis of the expectoration demonstrated the presence of *butyric* and *acetic acids;* and the odour was characteristic of the *butyrates of ethyl* (*Med. Times and Gazette*, May, 1857, p. 480). This condition is generally associated with dilatation of the tubes (*Bronchiectasis*).

An important result of bronchitis in the adult is condensation of the vesicular substance of the lung, of a peculiar type, caused by mucus in the air-tubes leading to the condensed portion. This is most apt to occur in patients debilitated by disease, by asthenia, or by age, although in such cases the amount of obstruction may be small. It is produced by collapse of the pulmonary vesicles, the boundaries of the condensation being distinctly mapped out by the interlobular divisions of the pulmonary lobules implicated. When collapse is extensive and sudden, it is not only a frequent cause of death, but, without being fatal, it is a fleeting temporary condition of immense frequency, and important practically to be distinguished from the condensation of lobular pneumonia (Dr. W. T. GAIRDNER The obstruction in the tubes may be due to inspissated mucus, or to soli fibrinous coagula, or to more or less prolonged spasm of the circula muscular fibre. Such obstructions tend to the production of pulmonar collapse; and if the obstruction be considerable and persistent, larg portions of lung may be emptied completely of air in the course of a fe hours. The mechanism by which such an obstruction brings about th collapse, appears sufficiently obvious when it is observed that such obstru tion is never absolutely complete. The air gradually finds its way out by the edges of the obstructing substance, impacted as it is more or less in a series of diminishing tubes, and acting the part of a ball-valve, allowing air to pass in one direction, but preventing it passing in another. The obstruction is thus driven onwards towards the narrower tubes by the force of inspiration, and occlusion is more or less perfect only in the one direction. The expiratory force, so long as there is air in the vesicles, constantly tends to dislodge the obstructing body by pushing it towards the wider end of the tube. While the entrance of the air is constantly and more or less effectually opposed, its exit is always permitted. Ultimately the air-vesicles of the tubes beyond the seat of obstruction become completely emptied, and collapse. The result is a condensation of the tissue—a condition which had previously been ascribed to a limited inflammation of the pulmonary tissue (*lobular pneumonia*), commonly believed to be peculiar to infants. It has been recently pointed out that another agency must be recognised in these results of the forcible expiratory movement—namely, that the blood which is contained in the heart and large blood-vessels is driven outwards, as seen in the distension of the temporal arteries and the venous turgescence which chronic bronchitis exhibits during a paroxysm of coughing (Dr. D. J. HAMILTON, *The Practitioner*, p. 19, Vol. XXIII., 1879). It is an exceedingly common lesion in the adult, also in old persons, in typhus fever and in fatal dysentery. It is always associated with a certain degree of bodily weakness, and is almost invariably found with fatal bronchitis (WEST, W. T. GAIRDNER). When this collapse is complete and permanent, it leads to important results :—(1.) The collapsed portions become atrophied, the proper and special elements of the lung having disappeared, causing a diminution of volume at the place where the collapse occurs. (2.) By a definite law—namely, *That a compensation by increased volume in one or more parts of the thoracic viscera invariably occurs to make up for diminished bulk in another, the internal capacity of the chest remaining the same.* This pulmonary collapse and atrophy lead to vesicular emphysema, and may even tend to dilatation of the heart. The most constant result, however, is emphysema, so much so that the one almost never occurs without the other. (3.) The emphysematous portions are almost invariably free from other diseased appearances, except the dilatation of the air-vesicles, and the consequent stretching and disorganisation of their parietes. (4.) Collapse takes place when a terminal bronchus, opening into a group of air-vesicles, becomes plugged with a mass of muco-purulent secretion. (5.) *Vesicular emphysema*, by *increase of volume* of those portions of the lung to which the air has access, compensates for the *diminished volume* of the collapsed portion. (6.) The *vesicular emphysema* prevails in the opposite parts of the lung to those in which the direct effects of bronchitis are observed. It prevails near the heart and large blood-vessels—the anterior margin of the lungs.

(*a.*) In *Plastic Bronchitis* with casts of the bronchial tubes and their ramifications, there is a lymphy exudation thrown out on their mucous surface, forming false membranes or casts, which are sometimes expectorated, with catarrhal symptoms more or less acute. Dr. Baillie first described and figured these tubular expectorated products; and cases have since been described by many observers, especially by Dr. T. Peacock, of St. Thomas's

Hospital, in the *Transactions of the Pathological Society of London.* Figures of such casts are to be seen published by him in the *Medical Times and Gazette* for 1854, p. 659. It has been described under the various names of *plastic bronchitis, bronchitis crouposa,* or *bronchité pseudo-membraneuse.* It is not common, and ought not to be confounded with cases of diphtheria or of croup.

(*b.*) *Bronchiectasis,* or *dilatation of the bronchi,* occurs in two forms:—(1.) *The general or uniform*—a cylindrical or fusiform dilatation of one or of several tubes throughout considerable lengths of their extent. (2.) *The saccular* or *ampullary*—an abrupt dilatation at a particular point, or at several points. The disease is of interest and importance on account of its alliance with forms of pulmonary consumption in simulating cavities. Wasting of the muscular and elastic coats of the tubes is a usual result, with degeneration of their tissue. These dilated tubes open into each other without any ulcerative process (Dr. T. G. Stewart, *Edin. Med. Journ.* for July, 1867). Copious yellow or inspissated mucus, with casts, often very fetid, exists in the dilated tubes, associated with crystalline fats and fungi. The site of *bronchiectasis* is usually the lower and the middle lobe of the right lung, and sometimes towards the apices. But it may affect many bronchi, and may occur in both lungs. The atrophy of the bronchial walls permits them to yield to the pressure of air, and favours the accumulation of mucus, which undergoes decomposition in the dilated cavities, leading to inflammation and the formation of villous processes, to increase of connective tissue, and to further consolidation of surrounding lung.

General Symptoms and Physical Signs.—(*a.*) *Acute Bronchitis of the Larger Tubes.*—Bronchitis, of whatever kind, is generally preceded by fever, and with symptoms of what is commonly called "a *cold,*" or "a cold in the *chest,*" as already described. It may commence without any previous illness, and the uneasy sensations frequently experienced about the region of the frontal sinuses, gradually pass down the nasal passages, and thence by the trachea and windpipe, and are then felt in the chest, especially over the anterior region. The symptoms are more fully expressed by the hoarse, altered voice, the cough and expectoration. In a few instances the cough is dry and without expectoration; but more generally it is accompanied by sputa, which vary greatly according to the degrees of inflammation, or as it is acute or chronic, sthenic or asthenic In acute cases it is at first a thin mucus, sometimes streaked with blood then more opaque, and lastly purulent. When purulent, it is usuall formed into sputa, but may be thrown up in large quantities unmixed, a from an abscess. The quantity expectorated varies greatly; sometime only a few sputa are brought up in the morning; and half a pint to a pin may be expectorated in the twenty-four hours. Patients may even d suffocated from the immense quantity which is suddenly poured out, causin obstruction of tubes and collapse of the vesicular structure of the lung *The cough* is seldom accompanied by any pain, and has many degrees violence. It may occur in paroxysms, the sputa being discharged after violent effort, at night or in the morning, or at intervals. Or it may incessant, harassing the patient at every instant, causing a sense of so ness or constriction of the chest, and sometimes severe pain at the en form cartilage, in consequence of the mechanical exertion of coughin

The urine varies greatly, as the grades of the disease are almost infinite. When aëration is greatly impeded the *urea* is in large amount, and the pigment increased, while the *chloride of sodium* is sometimes as deficient as in the height of extensive hepatisation. Those cases in which the urinary excreta are in small amount are more commonly those of severe and general capillary bronchitis. The patient (supposing the disease to be unconnected with any morbid poison or organic affection of the substance of the lung) suffers little in general health, and feels he would be well if he could get rid of the cough. In other cases he loses flesh, ejecting every meal from the violence of the coughing, or sinks into a state of marasmus. His pulse is generally natural, although in some cases it is frequent.

In bronchitis the respiratory *bruit* in the adult is changed, and varies, in different cases, from a tolerably sharp sound which, when multiplied from a number of bronchi similarly diseased, resembles a chirping sound, or the bass notes of the violoncello. The sounds embrace a musical scale of considerable compass, the principal and more marked division of which compose the *sonorous* and *sibilant* râles. The cause of the higher notes may be owing to a thickening of the mucous membrane at the orifices of the various bronchial tubes, so that the natural *embouchure* is narrowed, and the sounds resemble those from a musical wind instrument, or the morbid sound may be explained by the varying degrees of contraction of the circular and longitudinal fibres of the bronchi. Besides the alteration of *tone*, its *quality* is affected by the presence of liquid matters within the cavity of the bronchi. Hence the sounds are interrupted and modified by air passing through bubbles of mucus; and as the size of these bubbles and their viscidity vary, so the sounds vary. A scale was established by Laennec, the extremes of which are the "râle muqueux" and the "râle racheal;" the former representing the bursting of small slightly viscid bubbles, the latter larger ones of greater tenacity, yielding a gurgling ound. Sometimes this mucus hardens so as partially to adhere and play s a valve, giving rise to a clicking noise. The danger is denoted by the uantity of fluid effused, and may be judged of by the nature of the sound. The sharp chirping sound is more to be feared than the graver and deeper otes; for grave sonorous notes originate in the larger tubes, chirping histling notes in the smaller; and the danger in bronchitis increases in roportion as the finer bronchial tubes become involved. When the ounds of expiration are also much prolonged, severe bronchial inflamation is indicated. *Percussion* generally returns a healthy sound. A udden disappearance of the respiratory murmur over a definite part or arts of the lung, is due to obstruction of one or more of the bronchial bes; which, as already shown, may lead to *collapse, condensation, atrophy,* d *emphysema.* Percussion sometimes shows that the part still contains r. The respiration may be greatly accelerated, varying from 36 to 50 68 in a minute, especially in cases of capillary bronchitis. The pulse the same time rapidly loses its strength, and becomes excessively quent—120 to 150. The ratio of the respiration to the pulse is there- e greatly altered from the normal standard—that is, about 4 or 4½ ats of the pulse for every respiration; and thus *pulse-respiration* ratios of , 2·5, or 2·25 to 1 are not uncommon.

(*b.*) *Acute Catarrh of the Smaller Bronchi* (*Capillary Bronchitis*).—An attack of capillary bronchitis may begin with shivering, and an initial chill is much less frequent than in pneumonia. Headache is complained of, the tongue is furred, the face pallid, and the expression anxious. There is a feeling and aspect of general debility out of proportion to the objective symptoms, the patient having the appearance of being in the first stage of a continued fever. The breathing soon becomes hurried and somewhat laborious; an incessant hacking dry cough sets in, with the occasional expulsion of one or more greyish, or yellowish-grey, stiff pellets, or there may be scant frothy expectoration. The cough is of far more violent character from the commencement. The pulse-rate is quickened, the skin dry, and its temperature variable. The symptoms become worse. The breathing becomes more accelerated and difficult, and the breathlessness more harassing. The respirations go up to 60 or 70 in the minute, with a pulse-rate of 120 or 130; the pulse-respiration ratio being changed from 4·5 to 2. The supplementary respiratory muscles are brought into vigorous action, and the supra-sternal, mastoid, scaleni, and infra-xyphoid spaces, and the base of the chest along the insertion of the diaphragm, are forcibly sucked in at each inspiratory effort. The seat of pain in the chest is mainly at the points of insertion of the muscles upon the chest and epigastric regions, from the spasmodic jerking movements of the act of coughing, which continues in long paroxysms. The face betokens great suffering. There is cyanosis, the lips are purple, the conjunctiva is turgid, with a bluish-grey tint, and the skin of the extremities, at times cool and moist, is of a dusky hue. There is great exhaustion. In many cases there may be delusory intervals of comparative comfort between the exacerbations. As the disease progresses, the phenomena due to deficient oxygenation of the blood are aggravated; cyanosis deepens, anxiety is extreme and distressing, the surface is livid and damp, words are spoken with great difficulty, dyspnœa is intense, and all the respiratory muscles are forcibly acting; the patient is unable to lie down, though completely worn out. A frequent short, moist cough replaces the ceaseless hack. Expectoration becomes free, the sputa being brought up during or after each fit of coughing. They are frothy and aërated throughout, or spongy at the top, ropy, and adhering together as a single mass when turned out of the vessel. When turned into water the fine filaments of the thread-like exudation can be seen hanging from the frothy mucus floating on the surface. As death approaches the pulse usually becomes more rapid, large and compressible, and then small and thready. The respiratory efforts are less violent, the number of respirations begin to lessen, and the pulse-respiration ratio falls, and approaches to a more natural standard—the deepening asphyxia probably making the patient less sensible of the respiratory wants. Carbonic narcosis may, in some cases, retard the heart's action, and decrease the pulse-rate. Towards the fatal end, both the cough and the expectoration cease, and death happens from apnœa.

The physical signs of capillary bronchitis are—at first, *percussion-sound* natural over the whole chest; subsequently it may be slightly exaggerated over the superior and anterior regions, from the emphysematous state of the air-cells. There may be limited spaces of dulness, owing to collapse of the air-cells of one or more lobules, caused by obstruction of a bronchial tube; towards the close there may be diminished *percussion-resonance* on account of pulmonary œdema and excessive accumulation of morbid products in the air-cells and terminal air-tubes. *Auscultation* shows the respiratory murmur clearer and somewhat exaggerated in the upper portions of the lung, with diffused dry rhonchi over the chest. The sonorous rhonchus is often quite loud and musical, and the sibilant of high pitch; if the respiratory murmur is not masked by the rhonchi, it is in these regions feeble. Proceeding downwards, and as the disease progresses, the subcrepitant rhonchus—distinctive of capillary bronchitis—is heard, particularly in the posterior inferior regions. It is mainly an inspiratory sound, though audible in expiration, and resembles the continuous bursting of innumerable small unequal bubbles, or the sound produced by squeezing a not too wet sponge close to the ear. Heard at first about the base of the lungs, it soon becomes audible over nearly the whole chest, above and below, front and back, moving always upwards. It is from the beginning symmetrical. If the larger air-tubes are more involved, a mucous or submucous rhonchus may replace the dry rhonchi in the middle regions. When the lobules are collapsed, the signs of localised condensation of the lung tissue can rarely if ever be made out, on account of the existing rhonchi. *Vocal resonance* is increased. There is bilateral local expansion of the chest, often of large extent.

(*c*.) *Plastic Bronchitis* commences as a slight catarrh, which does not attract attention till the appearance of the peculiar sputa. Sometimes the symptoms are more acute, like those of pneumonia or bronchitis. The characteristic expectoration may be preceded by—(1.) A sense of constriction about the chest, with short breath, and a dry, hard, paroxysmal cough, which may last for several days. (2.) Breathlessness suddenly becomes urgent and alarming, the lips are blue, and the face swollen and livid, the extremities cold and discoloured. (3.) A fit of coughing, severe and strangling, brings forth the membrane usually along with the white, glairy, adhesive sputa of simple bronchitis, or there may be a little hæmoptysis. Relief is immediate, and lasts till there is a fresh accumulation and another exacerbation, followed by the same phenomena of detachment, expulsion, and relief. Several such paroxysms may occur in the twenty-four hours.

The characteristic expectoration (sometimes called bronchial polypi) was described by Ruysch and Morgagni, and is figured by Cheselden in his *Anatomy* (1722). Two forms are described by Michælis and Cheyne (*Edin. Med. and Surg. Journal*, Vol. IV., 1803)—(1.) *A moulded coagulum of blood;* (2.) *a condensed, lamellated, solid*, or *tubular membrane;* a form of fibrinous exudation where the blood-corpuscles escape along with the *liquor sanguinis*, as in some cases of *pericarditis* and *pleurisy* (WATSON, PEACOCK). It may be expelled in shreds, or in the form of oblong or rounded bodies, sometimes as large as a filbert, which, when macerated in water, gradually unfold into a stem and branches; or they are expelled as cylindrical casts of the bronchi, varying in diameter from one-eighth to three-eighths of an inch at the base of the cast, and from one to three or four inches in length, with small, divergent branches and minute terminal points, resembling some vegetable roots with their radicles or rootlets. As many as ten distinct subdivisions have been seen. They come from as low down as the second, third, and fourth bronchial ramifications. They are of a dull white colour, or occasionally brownish, from the admixture of blood. They seem to have been deposited or exuded in successive layers, and are composed of tough, concentric, fibro-membranous laminæ, between which a fine probe can be passed. The main stem is sometimes solid and sometimes hollow; or portions may be solid and others hollow. The number of casts expectorated may be from one to half a dozen a day for several months. Microscopically, these casts are of uniform structure, of parallel and nearly straight fibres, which are the edges of laminæ concentrically arranged and intermixed with numerous rounded bodies about the size of blood-corpuscles. Acetic acid produces expansion and partial solution of the laminæ, but has no evident action upon the corpuscles. Towards the termination of the minutest ramifications of the casts there are found compound granular cells mixed with irregular clusters of oil globules and ovoid bodies, containing pigmentary matter (BRISTOWE, *Trans. Path. Soc.*, Vol. V., 1864), showing that the lymphy exudation has commenced to undergo degenerative changes.

In some cases the expectoration of plastic membrane ceases after a few days, and recovery is rapid and good. In others, the acute symptoms abate, but false membrane continues to be occasionally expelled for several weeks, with intervals of comfort, and, in some instances, of apparent good health. The coughing paroxysms are often brought on by excitement or exertion. Cases are recorded where this disorder has lasted during one, two, three, or more years, either continuously or at certain seasons. In the chronic form the general appearance is unhealthy; the patients habitually short-breathed and pallid, with livid cheeks and lips, and incurvated nails (CLYMER).

(*d.*) *Dilatation of the Bronchi—Bronchiectasis*—comes on insidiously; but gradually symptoms of bronchitis become well marked. The most characteristic symptoms are *the odour of the breath*, the *characters of the sputum*, and the *cough*. The fetor of the breath is different from that of gangrene, and is not present in all cases, or not till decomposition of the bronchial secretion has taken place. *Blood* may be expectorated with the sputum, sometimes in large quantities, as in phthisis, at other times merely in streaks over the expectoration. *Cough* is frequent, and occurs in paroxysms, but is moist, soft, and usually quite painless. After coughing, large quantities of the sputum are brought up with difficulty. Exertion induces dyspnœa. The *physical signs* depend on the form, size, and extent of the dilatations. *Inspection* may show depression over the affected part of the lung. *The application of the hand* may detect increased fremitus. *Auscultation* gives varied sounds—cavernous moist râles, sometimes gurgling; and the site of these sounds aids to distinguish them from those of phthisis, as well as the history and progress of the case. Perforation of the pleura, empyema, or pneumothorax may prove fatal; or death may result from exhaustion due to the constant discharge of the sputum. A peculiar febrile disturbance, resembling *septicæmia*, may also terminate life. Unfortunately, the tendency is to a fatal result; but recovery may take place—(1.) *From cretification of the contents of the dilated tubes, and the conversion of their walls into a sort of fibrous capsule;* (2.) *from penetration of the pleura and thoracic parietes, and discharge of the contents outwards.* The disease is probably hereditary and constitutional.

Prognosis.—The duration of *acute bronchitis* is uncertain. It may terminate in a few days, ceasing with the cold weather that ushered it in; or its duration is long, and is with difficulty recovered from. It often lays the foundation of other diseases, which may ultimately destroy the patient. In old persons it generally returns every winter, or lasts with intermissions, during the whole year, becoming a chronic ailment.

The course of *capillary bronchitis* is rapid. In children it may last from three to six days; in adults from a few days to two weeks; in old persons it rarely exceeds a week after the acute symptoms have set in. In young children it is apt to come on very insidiously, the symptoms being like those of an ordinary catarrhal affection, exciting little attention, although there may be distinctive signs present from the outset—drooping, pallid face, quickened breathing, and fits of coughing, preceded and followed by wheezing. Dyspnœa suddenly appears, and chest-exploration shows extensive capillary bronchitis. In *pulmonary emphysema* of long standing, with extensive vesicular dilatation, intercurrent localised capillary bronchitis is very apt to happen; but it never becomes general, and though for a time aggravating the symptoms of the original disorder, is not a serious affection. Otherwise the prognosis generally is unfavourable, especially when capillary bronchitis supervenes upon any existing disorder which limits the respiratory area, and when in such cases it is general. In infancy and old age it is commonly fatal.

In *plastic bronchitis* the prognosis depends upon the presence and nature of pulmonary disease. The acute form of plastic bronchitis runs its course rapidly to a happy or fatal issue. In adults, however alarming or urgent the symptoms may be, the ailment usually ends in recovery, particularly when the false membrane is freely expectorated; or it may pass into chronic bronchitis, with occasional exacerbations, which subside after membranous expectoration. When the general and local symptoms are slight at the outset, the disorder is apt to be of longer duration.

Diagnosis is mainly as regards the presence of *capillary bronchitis* and *pneumonia.* From simple *acute bronchitis* it is to be distinguished by the general symptoms and physical signs. Acute bronchitis is nearly always preceded by symptoms of "a cold," and its course is downwards and peripheral, the proximal larger bronchi being first invaded, and afterwards the smaller distal ones. In *capillary bronchitis* the morbid action begins in the minute tubes near the external surface of the lung, and extends in an inverse direction—towards the large bronchial branches. The extreme frequency of the respiration in capillary bronchitis is another distinction. In ordinary bronchitis it is not usually much hurried. The sputa are characteristic. Capillary bronchitis may supervene upon and complicate simple bronchitis. It is of much practical importance to distinguish the two disorders, both as regards prognosis and therapeutics. The initial chill is much less constant in *capillary bronchitis* than in *pneumonia,* rarely amounting in the former to more than shivering, and there is reason to believe that the body-temperature is never so high. In the bronchial disorders the debility is earlier, the physiognomy different—more anxious and livid, the respirations quicker, the feeble reaction greater, and the sputa, when present, distinctive. Pneumonia is commonly unilateral, capillary bronchitis bilateral. The physical signs of the two affections are unlike, especially those shown by auscultation, the characteristic rhonchus of each affection having individual qualities. When collapse of the lobules has taken place in capillary bronchitis, the differential physical diagnosis may be less marked; but even here the rational symptoms and the presence and diffusion of the symmetrical pathognomonic rhonchus should prevent error. Occasionally a case of acute phthisis is met with that at first sight bears some resemblance, in the objective symptoms, to capillary bronchitis; but physical exploration, and a history of tuberculosis, will indicate the real nature of the disease.

In *plastic bronchitis* there is subcrepitant rhonchus, but it is limited, and never general; and the expectoration of plastic membrane in rounded pellets or cylindrical casts is really the only distinctive symptom of this disorder. The distinguishing signs between *plastic* and *capillary bronchitis* are sufficiently marked, the former being generally localised in one lung and one region, while the latter is symmetrical, and shows a great tendency to extend; the subcrepitant rhonchus, when met with in plastic bronchitis, is limited; in capillary bronchitis it is diffused. From pseudo-membranous croup and diphtheria it is distinguished by the local symptoms and physical signs, as well as by the character of the false membranes expelled.

Treatment.—(*a.*) In acute *bronchitis.*—General bleeding uniformly weakens the patient. In severe cases of acute bronchitis some blood may be taken from the chest, with great caution not to take too much, either by cupping between the shoulders or by leeches, and in general from ten to twelve ounces are sufficient. It is only in extensive congestion of venous circulation threatening asphyxia, that general blood-letting is imperative. Next to blood-letting, *tartarised antimony*, administered in solution in doses of *a sixth* or *a quarter of a grain, every three or four hours,* conduces to free secretion, and generally to mitigate the symptoms of the disease. *Digitalis* is a useful adjunct to the antimonial treatment. When the expectoration becomes thicker and less copious, the antimony may be decreased, and *squills*, or *ipecacuanha*, with *paregoric*, given. After this a blister should be applied to the chest; and on its removal a large linseed poultice should be placed over the blistered part, and continued for many hours. The bowels should be freely evacuated by a purgative dose of *calomel*, combined with *compound jalap powder*, and they should subsequently be kept in regular and gentle action by some neutral salt, such as *sulphate of magnesia* in the *liquor ammoniæ acetatis*, combined at the same time *with some nitrate of potass.* The neutral *citrates, tartrates*, or *acetates* of the alkalies are useful eliminating remedies.

After the severe symptoms at the outset have been subdued, expectoration should be promoted by *squills, ipecacuanha*, and *tartar emetic*, combined with *hyoscyamus* or *conium*. *Opium* in narcotic doses (*i. e.*, above a grain) is inadmissible, because its tendency is to paralyse the action of the mucous passages in eliminating secretion. *After secretion has begun to diminish*, in acute cases opium may be prescribed with benefit in *stimulant doses*, in the form of solution of the *salts of morphia*, added at bedtime to the doses o the *cough mixtures* so usually administered. (See under "Cold" o "Catarrh," p. 703, *ante.*) If the disease shows a disposition to pass into the chronic stage, *quinine*, with *squills* and *conium*, may be administered, o a draught containing full doses of *cinchona, with five grains of carbonate of ammonia*, or *ten to twenty grains of the muriate of ammonia, and thirty or forty minims of the compound tincture of benzoin (Friar's balsam), or of the balsam of Peru*, will generally facilitate expectoration and relieve the dyspnœa. If a "*hearty cough*" is not attended by easy and free expectoration, but the chest remains loaded, stimulant expectorants may be given in aid of other remedies, such as the *decoction of senega*, or the *mistura ammoniaci* (FULLER). The patient throughout the treatment should remain in a room where the air is kept moist by the evaporation of boiling water from large flat dishes near the bed, and the temperature of the air should be maintained at 63° to 68° Fahr. Alike in acute and in chronic cases, *iodide of potassium* is of great service. It has a marked effect in reducing the frequency of the respirations. It is best prescribed with *carbonate of ammonia*, and if cough is very troublesome, *tincture of belladonna* and *ipecacuanha wine* may be added.

(*b*.) In chronic *bronchitis*, especially in patients who have made considerable progress in the journey of life, a lower tone of the system prevails. A greater laxity of aërian membrane—particularly with excessive secretion, often muco-purulent—characterises them; and after blistering and poulticing the chest repeatedly with linseed and mustard poultices, the treatment in general should be more tonic. The *camphorated mixture* or *paregoric* and stimulant expectorant remedies are indicated for occasional, but not for constant use. Stimulating embrocations may be rubbed not only over the chest, both before and behind, but along the sides of the neck.

A liniment composed of the following ingredients was extensively employed by the late Drs. Graves and Stokes in the Meath Hospital, Dublin :—

℞. *Spt. Terebinthinæ*, ℥iii.; *Acid. Acet.* ʒiv.; *Vitelli Ovi*, i.; *Aq. Rosæ*, ℥iiss.; *Ol. Limon.*, ʒi.; *misce.*

It is to be applied as a rubefacient, morning and evening, when it generally reddens the skin and produces small pimples. In several cases the secretion of the kidneys is increased during its use (MACLACHLAN). Of *tonic remedies* most reliance may be placed on the influence of *nux vomica, iron, and quinine*, in the form of a syrup composed of the *phosphates of strychnia*, of *iron*, and of *quinia*; so that, in doses of a teaspoonful three times a day, each dose shall contain the *thirty-second part of a grain of phosphate of strychnia*, and *one grain respectively of the phosphates of iron and quinia*. (Formula for the preparation of this compound, see p. 1099, Vol. I.) The inhalation of *slightly* irritant vapours has a beneficial effect, as of *vinegar, turpentine, chlorine, and iodine*. Of the fetid gums, *ammoniac* in particular is a useful remedy. An emulsion of *gum* ammoniac in *diluted nitric* acid, is a combination from which beneficial results are obtained. The following is a formula for its prescription—namely, *an hundred and twenty grains of the gum ammoniac dissolved in two fluid drachms of diluted nitric acid and twelve ounces of water*, compose a mixture of which an ounce may be given in gruel or barley-water three times a day (EASTON, *Glasgow Med. Journ.*, Oct. 1, 1863). It may also be advantageous to administer astringent remedies, and one of the most useful is *tannic acid*, in doses of one to three grains two or three times a day; or the *oil of cubebs* to the extent of ten drops three or four times a day on a piece of sugar (see under treatment of "Phthisis").

Acute bronchitis is apt to be latent in old people, and to be complicated with gastric or gastro-enteric inflammation (MACLACHLAN). The stimulants of food (by *enema* in the form of soup, without salt, if unable to be taken by the mouth) and of *alcohol* are the main remedies necessary from the very beginning. Abstinence cannot be enforced with safety. Leeches must be applied to the pained gastric region. When the disease is associated with a tendency to gout, *colchicum* must be given. "It allays the cough, promotes the flow of urine, keeps up a regular alvine discharge, and can be given much more generally than *squills*, because it does not produce that feverishness which results from the use of the latter remedy, and can therefore be employed where there is considerable fever" (FORBES). It requires to be administered with great caution in the aged and infirm (MACLACHLAN). In the protracted bronchitic affections of the aged, diuretics are of great service. The following formulæ are recommended by Drs. Maclachlan and Stokes.

℞. *Decocti Senegæ*, *f*ʒvij.; *Potassæ Nitratis*, gr. iii.; *Tinct. Camph. Comp.* vel *Tinct. Conii*, ♏xx.; *Spiriti Ætheris Nitr.*, *f*ʒss.; *Oxymellis Scillæ*, *f*ʒss. *Fiaut haustus ter die summendus* (MACLACHLAN).

℞. *Liq. Ammon. Acet.*, ʒiii.; *Potassæ Acetatis*, gr. xx.; *Aceti Scillæ*, *f*ʒss.; *Spirit. Æth. Nitr.*, *f*ʒss.; *Tinct. Camph. Co.*, ♏xx.; *Mist. Camph.*, ʒvi.; *Syrupi Aurant.*, ʒi. (MACLACHLAN). *Fiat haustus ter die summendus.*

℞. *Decocti Senegæ*, ℥v.; *Tinct. Camph. Co. Tinct. Scillæ*, ā ā ʒii.; *Syrupi Tolut.*, ʒiv. *Sumat* ℥ss. vel. ʒi. *ter die* (STOKES, MACLACHLAN).

When gastric irritation prevails, the administration of balsams, gum resins, and terebinthine remedies must be suspended.

(*c.*) *In capillary bronchitis* the treatment must be strictly conservative; spoliative remedies lessen, if they do not destroy, the chances of recovery. The tendency to death is by apnœa, from the imperfect oxygenation of the blood—the area of available respiratory surface being greatly diminished, both from the morbid state of the minute bronchi and the accumulation of diseased products in them. The patient must be properly and adequately nourished. Stimulants are often required, and tonics should be early administered. The chest must be covered with hot poultices, and an oil-silk jacket worn over them. Dry-cupping has seemed to give relief to urgent symptoms. The *muriate of ammonia*—two grains every two hours—either alone or in combination with the chlorate of potash: and the *carbonate of ammonia* has long had a certain reputation in capillary bronchitis, especially in the later stages.

(*d.*) *In bronchiectasis* opiates are required to relieve cough; balsamic remedies (*tolu, tar, benzoin, turpentine, copaiba, cubebs*) and astringents, like *catechu* or *rhatany*, with the use of counter-irritants and the inhalation o variously medicated vapours, are all useful aids in ameliorating the condition of the patient. *Muriate of ammonia* and the alkalies are also to b recommended. Inhalation of disinfectants, capable of being so used, sucl as *creasote*, *carbolic acid*, *sulphur vapour*, *turpentine*, and the like, are of special service (see under treatment of "Phthisis").

(*e.*) *Plastic bronchitis.*—In the acute form, *muriate of ammonia* and the alkalies and *iodide of potassium* may be given, with an occasional emetic, and inhalation of the vapour of hot water. The patient should be carefully protected against damp and sudden changes of weather.

PNEUMONIA.

LATIN EQ., *Peripneumonia;* FRENCH EQ., *Pneumonie;* GERMAN EQ., *Lungenentzündung*—Syn., *Pneumonie;* ITALIAN EQ., *Pneumonitide.*

Definition.—*Inflammation of the lungs, which, in its acute sthenic form, is often complicated by constitutional or specific diseases. It runs a definite course, expressed by severe febrile symptoms, which come on suddenly, attaining in a few hours a great intensity, but which may undergo a no less sudden abatement or improvement between the fifth and tenth day, in proportion to the severity of the disease and the textures implicated, while the local productive results of inflammation in the form of the lung-lesion are yet intense, but which are eventually removed. The natural course of pneumonia is materially modified by constitutional or specific diseases, especially if any organ, such as the kidney, the heart, or the liver, is involved; or it may be modified by the secondary contamination of the blood by absorption of lung-exudation in the later stages of the disease, tending to inflammation of the other lung, to pleurisy, to pericarditis, or to blood-coagula in the cavities of the heart or great vessels.*

Pathology.—(*a.*) *Historical.*—In the continental cities of Europe about *eight* per cent. of the deaths are caused by pneumonia; and while about *three* per cent. of all diseases are due to the same cause, about *two* per cent. of all cases of disease in hospital are referable to pneumonia. It is of general and universal prevalence, subject to periodic fluctuations, appearing as if it were epidemic. The only variety recognised by the College of Physicians is "*lobular;*" and the term "*secondary*" has been applied to pneumonia when it occurs as a complication of other disease. On the other hand, Niemeyer regards inflammation of the lungs as of three kinds, namely:—(1.) *Croupous pneumonia*, in which the air-cells are involved in a morbid process identical with that which attacks the mucous membrane of the larynx in tracheal and laryngeal croup (*acute pneumonia*). (2.) *Catarrhal pneumonia*—a process intimately related to that already described as *acute catarrhal bronchitis.* When inflammation affects the smaller tubes (*capillary bronchitis*), the disease can with difficulty be separated clinically from catarrhal pneumonia, which produces an augmented secretion and active generation of pus-corpuscles, but in which no coagulable exudation is formed. In the forms (1.) and (2.), the products of inflammation are thrown out upon the free surface of the mucous membrane of the air-cells, the tissue of the lung itself suffering no essential change. Dr. Bristowe suggests the names "*diffuse*" and "*patchy*" as more appropriate. (3.) *Interstitial pneumonia*, when the inflammation involves the walls of the air-vesicles and the interlobular connective tissue, which from its chronic course is sometimes termed *chronic pneumonia.*

(*b.*) **Causation.**—Sudden chilling when the body is overheated, and the influence of season, are the most obvious causes. The largest number of cases happen in February, March, April, and May, during protracted winters and the prevalence of north-east winds; but opinions are divided as to the influence of absolute cold in producing pneumonia. It may be associated with an acute dyscrasia, such as *measles* and *typhus fever;* when it has obtained the name of "*secondary pneumonia.*" Its prevalence has often been observed to be coincident with typhus fever. Pneumonia is rare before five years of age. In *typhus* and *enteric fever*, pneumonia occurs as a local lesion, and is sometimes named "typhoid pneumonia." In another class of cases, characterised by *blood-poisoning* or by *embolism*, the lung disease is a secondary result. While in yet another class of cases, as in those where some morbid condition of the constitution prevails—as in the alcoholism of drunkards—a primary pneumonia in them may give rise to blood-poisoning and the phenomena of pyæmia, by the sanious decay of the exudation and its re-absorption during resolution or its passage to the left side of the heart.

(*c.*) *Morbid Anatomy.*—(1.) *Croupous, Acute or Diffuse Pneumonia, Lobar Pneumonia.*—A knowledge of the anatomical characters of the lung is essential to a perfect appreciation of the symptoms, physical signs, prognosis, and treatment of pneumonia. In all forms of diffuse inflammation of the substance of the lungs, their tissue is more loaded with dark venous blood than usual, and the texture is more easily broken down than in health. Air, however, still penetrates the air-cells, and consequently the lung still crepitates, swims in water, and, if washed, the colour is nearly restored; but it is doughy, pits on pressure, is red or livid, and heavier than the normal lung. This is the *first stage of pneumonia—namely, that of simple engorgement*, which may terminate by resolution, or may pass to more complicated and dangerous lesions. When effusion of serum succeeds, the lung is in the same gorged state, and loaded with watery fluid. On cutting into it the serous fluid mixed with blood streams from it as from a sponge. A lung in this condition no longer crepitates, and its bulk is enlarged. It may be seen to have taken the impression of the ribs, and it does not collapse when the chest is opened. It is now technically said to be in a state of *red hepatisation*, or *red softening;* for, although firm, its texture has lost its natural toughness, cohesion, and resistant power. This is the so-called *second stage of pneumonia.* The diseased part readily breaks up by a thrust of the finger into its substance. Up to this point the description of the morbid anatomy is common to the extreme stage of bronchial catarrh, capillary bronchitis, and the commencement of acute, diffuse, or croupous pneumonia. In some cases the productive effects of inflammation (commonly called *exudation*) are very large in quantity, mixed with blood, and the more fluid portion can be readily pressed out of the lung. In other cases the exudation seems to form an integral part of the lung, which then becomes so solid that, if cut, it represents a portion of liver or spleen. It contains in the diseased part little or no air; does not crepitate nor float in water; i cannot be injected, while its texture is easily broken down and penetrated by the finger. It is still enlarged or swollen, and does not collapse whe the chest is opened. It varies in colour from a reddish brown or dee dull red to a violet hue, and is generally darker in the aged than in the adult. When the lung is more acutely inflamed, the bronchial tubes are red, and greatly inflamed (*Broncho-pneumonia*).

The most commonly acute form of pneumonia in adults is that which suddenly attacks an extensive portion of lung, usually commencing at the root, from which it uniformly diffuses itself, first to the lower lobe, passing thence upwards—so that the entire lung may participate in the process. When it extends into the other lung, the condition constitutes a double pneumonia. When the air has disappeared from the air-vesicles, they are seen to be filled by small firm plugs of coagulable fibrine, coloured red by blood (hence *croupous pneumonia*), which extend into the extremities of the bronchi. This new material renders the lung heavy. It sinks in water, does not crepitate, is dense to the touch, but is at the same time tender and friable in texture. When cut into sections the surfaces appear granular, in consequence of the fibrinous plugs swelling out the air-vesicles, to the walls of which they firmly adhere. This firmness of section, rigidity, friability, and granular aspect, with redness and condensation, has obtained the name of *hepatisation of lung*. The microscope shows that a very active formation of new cells takes place in the air-cavities, which, no doubt, spring from the epithelium; and when resolution sets in, fatty disintegration precedes that sanitary process.

When purulent cell-formation and infiltration occurs, pus becomes prominent, and may be either infiltrated or limited, as if contained in an abscess—abscess of the lung. Infiltration is by far the most common; and although this form of lesion may occur suddenly, following serous infiltration in unhealthy persons, or during the progress of general diseases, yet it more generally follows *red hepatisation*. In this latter case the pulmonary tissue, red, dense, compact, and impermeable to air, passes to a grey colour, and is termed *grey hepatisation*, or *grey softening*. The structure in other respects of either form of hepatisation appears to be the same; for the same granulations, white or grey or red, are found. There are instances, however, in which these granulations are wanting, when a grey smooth surface only is observed. Hence the *granular* and *non-granular* forms of hepatisation described by some authors. In aged persons the granulations of a pneumonic lung are much larger than in the adult, depending on the increased size of the air-cells in persons of advanced life (MM. HOURMANN, DECHAMBRE, and Dr. MACLACHLAN). Hepatisation in old people, with granulation, is nearly four times more common than non-granular or planiform hepatisation (MACLACHLAN, l. c., p. 279). In the *grey*, as in the *red hepatisation*, the tissue is easily torn, and the quantity of pus infiltrated is sometimes so great that, on cutting into the lung, pus readily flows from it: at other times the pus will not flow on a simple incision, but exudes by compression. The lung is solid and impermeable to air. It sinks in water, and has ceased to crepitate. The interspersion of red and dark points gives a granite-like appearance to the section of a lung which is in a state of grey hepatisation; and it is then more friable than in red hepatisation. The finger sinks into the grey portions upon the least pressure, and when squeezed it breaks into a pulp.

(2.) *Catarrhal, Patchy, or Lobular Pneumonia—Broncho-pneumonia*—is always preceded by catarrhal bronchitis, and may be a form of capillary bronchitis extending to the air-cells; hence, many of its features are similar and undistinguishable from capillary bronchitis (*q. v.*, p. 712). It is often a complication of measles and of hooping-cough. It is the form to which the name or variety of *lobular pneumonia* has been applied, because the lesions are limited to lobules, and are disseminated or insulated in patches in contra-distinction to the acute or croupous form, which is diffuse and affects whole lobes. The lesion is widely spread, and is recognised by the scattered, condensed foci of inflamed tissue, chiefly abounding towards the periphery, where they are more or less wedge-shaped. On section, these condensed portions have a smooth homogeneous appearance, without the granular characteristics of acute or *croupous* pneumonia. They are impervious to air; and a dilated bronchole, filled with tenacious secretion, may be seen in the centre of them.

(3.) *Chronic or Interstitial Pneumonia, Cirrhosis of Lung, Fibroid Phthisis.*—The little connective tissue which pervades the lung is the principal texture involved, so that a hyperplasia or growth of its substance takes place (*cirrhosis*), but the whole parenchyma is extensively implicated—resulting (1.) in augmentation of the substance of the lung, with compression of the air spaces, and (2.) contraction of the new material. Interstitial pneumonia scarcely ever occurs as an independent lesion. It is usually the result of prolonged irritation, as from the inhalation of iron or steel filings, fine dust of coal or other substances, which establishes a bronchitis, followed by the induration peculiar to this chronic form of pneumonia. Before contraction of the new material commences, the lung is solidified, void of air, hyperæmic and red, or of a pale bluish-grey colour. After contraction has begun, bands or condensed masses of tissue run through the pulmonary substance, which contain much pigment, and grate like cartilage under the knife. It is the material which forms the walls of old cavities and of abscesses of the lung. It is usually an affection of middle and advanced life, and is frequently met with among spirit-drinkers. It has sometimes been described in England and America under the name of "*fibroid degeneration of the lung.*" It has been regarded—(1.) As a variety of phthisis (BAYLEY); (2.) as a result of unresolved pneumonia (ADDISON); (3.) as a special form (interstitial) of pneumoni (ROKITANSKY, NIEMEYER); (4.) as a peculiar growth or hypertroph (WILKS); (5.) as a pulmonary cirrhosis (FELTZ); (6.) as fibroi degeneration of the lung (CLARK, CLYMER, SULTAN). In general bot lungs are affected; and if only one, generally it is the left that suffer commencing in the upper lobes. It occurs as a nodular growth, or as diffused fibrous tissue development. Cavities are apt to form, whi constant cough, with expectoration and gradual waste, brings the disea under *one form* of pulmonary phthisis or destructive lung disease, to afterwards considered, the progress of which is very slow, extending ov several months, and even years.

The weight of pneumonic lungs is generally in proportion to the ext of the pulmonary tissue involved. Grisolle has recorded the weight o hepatised lung as high as 5½ lbs. An average excess of 2 lbs. over weight of the opposite sound lung has been found, and the weight of new material has been found as much as 4 lbs. 3 oz. The right lung s more frequently attacked first than the left; and pneumonia of the ri t lung is about twice as frequent as pneumonia of the left, and this holds good at all ages. The inferior lobe is oftener affected than the superior, in the proportion of four to three; while pneumonia of the apex is two and a half times more common in the right than in the left (GRISOLLE). Double primary pneumonia is very rare. The formation of one or more abscesses is a rare termination; but the rarefied condition of the lung in the aged "seems to favour the formation of small abscesses, which are occasionally seen interspersed through the red and grey consolidated tissues, as if certain air-cells had broken down and coalesced during the plastic or suppurative process, and formed so many sacs for the reception of the effused or secreted matter."

Symptoms.—The characteristic phenomena of pneumonia in the majority of cases are evolved in a regular and consecutive manner.

1. *Acute, Diffuse, or Croupous Pneumonia.—The Course of the Pyrexia as Measured by the Thermometer and the Pulse.*—The commencement of primary croupous (acute) pneumonia is marked by a rigor in almost all cases, which lasts from half an hour to several hours before giving place to a sensation of heat. Nevertheless body-temperature is appreciably elevated during this algide stage; and except in *ague* and *pyæmia* rigors of equal violence to those in pneumonia are not met with in any other disease. In ague and pyæmia the rigors are repeated; in pneumonia the rigor is not repeated; and hence it marks the commencement whence the duration of the disease is counted. In children, convulsions may take the place of rigor. The commencement dates from the *shivering*, and four hours after the initial rigor the temperature has been noted as high as 102·5° Fahr. in the axilla; and eight hours later, *i. e.*, twelve hours after the commencement or invasion, it has risen to 104° Fahr. (ZIEMSSEN). The elevation of temperature rises on the first day even to 103° or 105° Fahr. (rarely higher), and is accompanied by acceleration of pulse, and by increase of thirst. The face is flushed, pain is felt in the back and loins, with soreness of limbs and joints. By the third day the patient is intensely febrile, with flushed cheeks, constant cough, viscid bloody expectoration, characteristic of pneumonia, hurried breathing, with crepitation and bronchial respiration. The fever is never a continued one, but remittent or sub-remittent—*i. e.*, the daily exacerbations and remissions may be considerable, varying from 7·5° Fahr. to 1·80° Fahr.; or may be so slight as not to exceed from 0·4° Fahr. to 0·5° Fahr. (THOMAS of Leipsic). The temperature is lower during the early morning hours; and the exacerbation for the day begins in the course of he forenoon, attaining its height usually in the afternoon, when in bad ases it may rise to 105·8° Fahr., or even to 107·7° Fahr. In most cases day or two before the crisis the remission increases. The progressive ncrease of temperature rarely continues beyond the second day; for enerally about that time, or about 48 hours after the initial rigor, the aximum of temperature is reached. During the *third* to *fifth days* the mperature is uniformly high. Towards the end of the *fifth day*, and uring the commencement of the *sixth*, the thermometer falls slightly; nd during the early part of the *sixth day* of the disease, it makes a great scent. The fever usually ends by crisis (a sudden and rapid deferscence), usually accompanied by a marked action of some eliminating

organ, such as the skin, the kidneys, or the bowels. Several observations carefully made at Netley have shown an elevation of temperature in the axilla of the affected side greater than that of the opposite side. The fever of pneumonia has the following peculiarities, namely:—(1.) The occurrence of isolated *brusque* elevations and intercurrent falls of temperature, the latter of which are in direct contrast to the elevations. (2.) The continuous high temperature lasts as long as the process set up in the lungs progresses, which is seldom less than three days, and seldom longer than seven. (3.) Defervescence in the majority of cases begins late in the evening, and takes place rapidly.

The Correlation of the Pulse and Temperature.—In a pneumonia of average severity, the pulse usually ranges between 100 and 120 beats in a minute, and then falls to 90, 80, or 70; but in severe cases, when the temperature is very high, it may attain a frequency of 130, or even 150, or more. At the outset it is generally large and full; but as the disease progresses, it becomes small and soft, from depression of the heart's action consequent on the blood-heat and secondary blood-poisoning. There is a connection between the variations of the pulse and temperature, so that either simultaneously, or often a little before or after, a fall or rise in the thermometer, a fall or rise in the number of the pulse occurs. The respirations average 38 to 50 in the minute during the first four days, according to the amount of lung whose function is suspended. They do not fall nearly so much as the *temperature and the pulse*, and are not nearly so good an indication of the course of the pyrexia.

2. *The Course of the Local Lung-Symptoms.*—There is considerable crepitation, and some bronchial respiration in the pneumonic lung. The hepatisation increases, and may be considerable on the fifth and sixth days. Its greatest amount is either at the period of *defervescence* or subsequent to it—the number of respirations being even greater after the temperature and pulse commence to fall than before, so that they may appear rather to run *parallel with the amount of the hepatisation than with the general fever.* The sputa are most bloody during the *third and fourth days* of the disease—less florid and more rusty on the *fifth* and *sixth*—after which they become less viscid and free from blood or hæmatine. Pain in the side seldom exists unless the pleura is affected. It consequently indicates a additional danger (pleuro-pneumonia), unless it disappears by the *fourth* o *fifth day.* After the *seventh* or *eighth day* the bronchial respiration begin to lessen, and ceases by about the *twelfth* to the *sixteenth day.* Harsh respiration, some *redux* crepitation, and a little sonorous *râle* may be lef for some days more.

3. *The Condition of other Organs.*—There is usually supplementar breathing in the other lung, in which there may be some slight suspiciou bronchial respiration, if it too is about to be attacked. *The liver* may enlarged or tender, and the conjunctivæ slightly yellow, and the yello ness may disappear at convalescence. There is thirst, loss of appetite, a dry furred tongue. *The skin* may act profusely throughout, both before a at the period of defervescence; the bowels are usually confined. A litt albumen may appear in the urine at the subsidence of the intense pyrex

The observations of the late Drs. Parkes, Wunderlich, and others, ha shown that the so-called physical signs of pneumonia are inferior in val to those afforded by accurate observation of the correlations of temperature, pulse, condition of the urine, and tendency to crisis. The crisis may occur on any day of the disease, but the usual dates are the *fourth, sixth,* or *seventh day.*

The Urine in Pneumonia.—As to normal products, the amount of water is lessened by a third or a half, most marked during the early days; and during the stage of resolution the amount of water increases. *The urea* is greatly increased during the height of the disease, more abundant during the day than during the night; and in larger quantity *before* than during resolution. The general result is that *the greatest amount of urea coincides with the highest temperature, and that it arises from general increased metamorphosis. The specific gravity* is high (1025–1035), partly from deficiency of water, and partly from excess of urea; and as the chloride of sodium is absent, the specific gravity indicates pretty accurately the amount of urea. *The uric acid* is increased, and its free excretion is a favourable sign. *The urine-pigment* is increased two or three fold, or altered. It tints the urates, when they fall, brown, red, or carmine; generally the colour of the urine is of a deep yellow-red hue, or flame colour. *The chloride of sodium* is diminished, or is entirely absent during the early period, or at the commencement of hepatisation. Even when given by the mouth at this period it will not pass off as in health, but is retained, and will not appear in the urine. It reappears during, or rather after resolution, but it does not always reappear directly resolution commences. It may be retained for some days (*eight* or *ten*), after which it is poured out in such quantities as to raise the specific gravity. This occurs in cases where there is little expectoration and no purging, although the water of the urine is increasing and the urea is diminishing. *Fifteen* to *twenty-five grammes* may be thus passed in the urine during twenty-four hours; and the period of its increase is some time after the excretion of urea has reached its acme and is declining. There is thus ample evidence that the chlorine has been retained, and not excreted in excess through the skin; and even when *hydrochloric acid* is given, no chlorine appears in the urine. Beale's observations also prove that the lung-exudation is very rich in chlorides, which are often largely excreted in the sputa. The retention of the chlorides seems to be intimately connected with the development of the lung-lesion or exudation; and their excretion is increased during its absorption and disappearance. *The sulphuric acid* is increased by about a third. *The phosphoric acid* and the free acidity are lessened.

As to abnormal products in the urine.—They are chiefly, (1.) *Bile-acids.* 2.) Bile-pigment, with or without jaundice; and, (3.) Albumen is present n a large proportion of cases. The period of its occurrence is variable; ut both Weller and Parkes agree that it is most common at the ommencement of and just before resolution—*i.e.*, at the height of the isease. In some cases, instead of being increased, the urinary solids are ssened, even in intensely febrile cases, with a heightened temperature of *ve or six degrees*—a result of retention, and not of diminished excretion ee Vol. I., p. 264, *et seq.*) In such cases some one or other of the llowing events happen :—(1.) At a later period a large amount of some gredient may be poured out by the urine, although the febrile symptoms ive almost or entirely gone—*e.g.*, uric acid—the crisis by the urine of the older physicians. (2.) Towards convalescence spontaneous diarrhœa may come on. (3.) The recoveries are not so rapid as in cases in which the urinary excretion is large. The amount of the urates, indeed, is a good guide for prognosis. As in *typhoid fever* and other acute affections, it is probable that the products of metamorphosis are retained, and poison the blood.

The leading **General Smyptoms** of the several forms of pneumonia may be stated as follow:—

(1.) *Symptoms of Acute, Diffuse, or Croupous Pneumonia.*—In about a fourth of all the cases, the usual initial symptoms of a threatening illness prevail, such as febrile phenomena or bronchitis. The actual invasion is generally marked by a rigor, the nature of which has been already described. In old people it is invariable. Sharp lancinating pain at or near the nipple of the affected side, is an early and most common symptom. Some describe the seat of pain as at the point where the inflamed lung comes in contact with the thorax; but opinions are expressed of the most opposite kind with regard to pain in pneumonia. It may indicate implication of the pleura (*pleuro-pneumonia*). Simultaneously with the rigors, shortness of breath sets in, and hurried respiration is an early and constant symptom—varying from thirty to sixty in a minute (which latter number is rarely exceeded), in proportion to the amount of lung disabled. In children the breathing attains the greatest frequency. The countenance, tongue, and lips are more or less livid, in proportion to the imperfect aëration of the blood. The nostrils are dilated. The tongue is coated with a white or yellow fur. The patient inclines to lie on his back well supported by pillows. *Cough*, hardly ever absent, and often present from the very beginning, is short, dry, ringing, and harsh. At an early period the peculiar viscid adhesive sputum, at first scanty, semifluid, grey and frothy, begins to be ejected. It maintains sometimes a catarrhal, yellow, or white appearance, and increases in viscidity. It almost always contains blood, from rupture of capillaries and extravasation of their contents. So viscid are the sputa, that the containing vessel may be everted without the sputa falling out, being highly charged with albumen and mucin. Fibrinous casts of the minuter bronchi may be found in the expectoration. *Headache* usually continues throughout the attack, frontal, acute, lancinating, or constrictive. It is generally combined with sleeplessness, and sometimes slight delirium. It is at its worst about the fourth day, and generally begins to subside as the fever subsides, about the seventh day.

Physical signs are to be appreciated by *inspection, palpation, mensuration, percussion,* and *auscultation. The physical signs* in the adult and in the aged have been already described, to some extent, in connection and in correlation with the morbid anatomy of the disease. During the *first stage, inspection of the chest* shows that costal movements are not materially diminished, unless the motions of respiration are restrained by pain. The natural sounds heard on percussion of the chest are altered (page 543, *ante*). They are rendered duller than natural, while the bronchial respiration undergoes still more remarkable alterations. It is only by *percussion and auscultation combined* that the existence of pneumonia can be determined. By *percussion* a peculiar resonance may be emitted from that portion of the chest at which pneumonia is beginning, which ceases after the commencement of exudation, to be replaced by dulness more or less marked according to the extent of the lung involved (FULLER). During the period of invasion in the aged, the chest continues to sound clear over the seat of the impending inflammation; and as congestion increases, the lung becoming more dense and less permeable, the sound emitted on percussion may be somewhat duller; but in general there is no appreciable alteration till the *second stage* has set in (MACLACHLAN), when the signs elicited by *percussion* depend on the consolidation of the tissues, the amount and position of the consolidated part in relation to the surface of the chest, and the amount of intervening healthy lung. If the consolidation is near the surface, the sound is decidedly dull, and the sense of resistance to the extremities of the percussing finger is considerable in proportion to the amount of consolidation. For the chest to emit this unequivocal dull sound of hepatisation in middle age the disease must be extensive, and without intervening healthy lung; but in some cases, if hepatisation be central, the air in the more superficial portions of healthy lung may prevent a dull sound from being heard. In such cases percussion elicits a clear but shallow resonance. A slight diminution in the resonance of the chest, immediately over the condensed portion of the lung, is usually all that percussion elicits in old age. By *auscultation* during the second stage, tubular breathing is found to be a constant accompaniment alike in the aged and adult. The respiration is of a hollow character, diffused throughout the hepatised portion—tubular towards the centre, but harsh and blowing towards the periphery of the affected part. Where tubular breathing prevails, râles and rhonchi are absent; while vocal resonance is intense, usually of a metallic ringing character. This is most marked in the aged. *Œgophony* in them is more decided, the shrill, acute, tremulous voice of old age being more favourable to *œgophony* than to *bronchophony*. When fibrinous exudation is great and complete, blocking up vesicles and tubes completely, absolute dulness on percussion, with little or no vocal resonance, can be heard. The heart's sounds, greatly intensified, are then frequently transmitted hrough the consolidated lung. *Auscultation* during the early period of ngorgement, before exudation has taken place, finds the breathing weak n the affected parts, and exaggerated in their immediate vicinity. Both weak and harsh respiration may be heard at the very beginning of pneunonic engorgement, according as the bronchial or vesicular element of the reath-sound predominates. If the morbid process is limited to the ir sacs, the soft, breezy, vesicular sound will be feebler, while the natural und in the minute bronchial tubes, more or less marked in healthy spiration, becomes more audible, without increased intensity. Those unds may alone reach the ear (Drs. CLYMER, A. FLINT, and J. R. EAMING). In the aged, tubular breathing is sometimes audible at the ot of the lung at a very early period, if respiration is weak. But as on as fluid exudes into the affected parts, the respiratory sounds are scured or replaced by the *small crepitation* characteristic of pneumonia. is sound is made up "of a multitude of minute crackles, which occur a volley towards the end of inspiration, and are unaffected by coughing expectoration" (FULLER). It is the *crepitating râle* of Laennec, and has been variously compared "to the crackling of salt thrown upon the fire," or to "the sound produced by rubbing a lock of hair between the finger and thumb close to one's own ear" (WILLIAMS). It usually, but not invariably, accompanies the accession of pneumonia. It is very seldom present in advanced life. In subjects beyond the age of fifty, and particularly in still more advanced age—as between sixty and eighty—the bubbles composing the pneumonic crepitation are very generally larger, more hurried, and less numerous. The râle in the aged essentially resembles the *subcrepitation of capillary bronchitis*, and is very often speedily marked by copious accumulation in the larger bronchi (MACLACHLAN, GRISOLLE, CAZNEUVE, HOURMANN, DECHAMBRE). When the air-cells are completely filled by the exudation, and the minute bronchial ramifications obstructed, crepitation ceases; and when capillary bronchitis commences, small bubbling râles are heard accompanying both expiration and inspiration. When acute pneumonia arises in connection with *acute rheumatism*, crepitation may never occur (FULLER). Vocal resonance is generally intensified. The return of the respiratory murmur, and the gradual disappearance of crepitation, indicate the resolution of the disease—a process generally more tedious and less perfect in the aged than in the adult, often followed in elderly persons by symptoms of continued irritation of the lung or bronchi. In proportion as crepitation masks the respiratory murmur, and replaces the sound of pulmonary expansion, the supervention of consolidation may be anticipated. Beyond the limits of dulness and consolidation, *the extension of fine crepitation* denotes the extension of the process of *hepatisation;* but when resolution has commenced, crepitation again returns to the part which was dull and consolidated before; or moderate-sized râles are heard, denoting the passage of air to and fro amongst the fluid which occupies the air-cells and smaller bronchial ramifications; and very soon the percussion note assumes its normal character.

During the *second stage, inspection of the thorax* shows the costal movements diminished on the affected side, and they may be somewhat increased on the unaffected side; but the movement of elevation is less affected than that of expansion. By *palpation* we learn that *vocal fremitus* is usually above the average. But vocal fremitus may be entirely abrogated when the bronchi are plugged with exudation. The *fremitus* produced by a deep-toned powerful voice is seldon observed in the aged, and even *bronchophony* is occasionally absent (MACLACHLAN).

In the third stage of the disease the physical signs do not differ from those already described; and neither in the adult nor in the aged are any certain phenomena appreciable by which the passage from the second to the third stage can be determined. A persistence of the sounds already described in place of those denoting resolution, and especially when th expression of the phenomena diminishes with increasing prostration of th patient, and diminished energy of voice and respiration, betokens *suppuration of the lung.*

Fine crepitation of the lung cannot always be regarded as pathognomon of acute pneumonia. It may be absent throughout the attack, alike in the adult and in the aged. "It is only by the concurrence of differen signs, that it is possible to arrive at a trustworthy conclusion. Fin crepitation occurring coincidently with intense heat of skin and rusty-coloured expectoration, warrants the strongest suspicion of pneumonia, and justifies the adoption of active treatment. If it be accompanied by marked alteration in the ratio of the pulse and respiration, speedily followed by dulness on percussion and tubular breathing, the existence of acute pneumonia cannot be doubted. But crepitation, however fine, if not attended by an alteration in the ratio naturally subsisting between the pulse, the respiration, and the temperature, and not speedily followed by tubular breathing, cannot be relied upon as indicative of pneumonia. If it occurs without these symptoms, it is commonly indicative of capillary bronchitis with scanty secretion; whereas, if under the same conditions, it is accompanied by dulness on percussion, it is probably due to rapid œdema, or to congestion of the lungs, connected with some febrile hæmic disorder, cardiac disease, or the deposit of tubercle (Dr. FULLER, l. c., p. 229).

(2.) *Symptoms of Patchy or Catarrhal Pneumonia* generally supervene on catarrhal bronchitis, and may thus run an acute course, or pass on to excessive emaciation and wasting. The cough is painful; and the temperature of the body always becomes elevated. In simple capillary bronchitis the temperature rarely exceeds 102° Fahr. (ZIEMSSEN); but when patchy or *catarrhal pneumonia* supervenes, it may soon reach 104° or 105° Fahr., with frequent pulse and flushed face. Collapse of the lung extends in proportion to the foci of tissue involved, and progress to a fatal end may be more or less rapid. When resolution sets in, it commences gradually, and progresses slowly but steadily, with a range of 2° to 3° Fahr., after about the sixth or seventh day.

(3.) *The Symptoms of Interstitial Pneumonia* are obscure. It is apt to follow an acute bronchitis which has become chronic, a pleurisy, or the tardy resolution of acute or croupous pneumonia, as an induration. The contraction of the indurated part is rendered conspicuous by the sinking in of the thoracic wall, or by flattening of the chest over the site of lesion, and different degrees of pulmonary condensation complete the physical signs. Constant cough and breathlessness are complained of. There is great loss of flesh at first, which continues to progress, although more slowly than at first. Profuse hæmoptysis is sometimes an early and occasional symptom. The expectoration is that of chronic bronchitis, sometimes streaked with blood. There may be concomitant symptoms of hyperplasia of the connective tissue in other organs, such as *granular kidney*, *fibroid degeneration of the heart* or *pylorus*, or *cirrhotic liver*.

Diagnosis.—Pneumonia is distinguished from phthisis by the previous good health of the patient, by the more acute nature of the disease, and, in some degree, by a difference of its seat, the lower lobes being more particularly affected in inflammation, the upper lobes in phthisis. The two diseases, however, are often combined. Many cases of capillary bronchitis are reported as pneumonia. In determining distinctly the further diagnosis of pneumonia, from the phenomena of condensation and respiratory murmurs, it is necessary to bear in mind the various conditions which may produce condensation of the lung, either in the child or in the adult. The conditions which lead to consolidation of the lung are:—(1.) A partial unexpanded state in a new-born child, termed *atelectasis* (JÖRG).

(2.) The accumulation of mucus in the bronchi and air-cells—*bronchitis* in the infant, followed by *lobular pneumonia* of children, the *état fœtal* of the French (FUCHS, WEST, ZENKER). (3.) Collapse of the pulmonary air-cells, causing *lobular* or *more patchy* forms of pulmonary condensation in adults, as well as in children, due to bronchial obstruction (GAIRDNER). (4.) It is probable that *hypostatic pneumonia* (PIORRY), and *péripneumonia des agonisans* (LAENNEC), and *some* of the so-called *latent pneumonias*, are forms of condensation due to *pulmonary collapse*, combined with serous effusion or vascular congestion. (5.) The coagulation of inflammatory lymph in or about the pulmonary vesicles, so that they are imbedded in solid material, "as the stones of a wall are in mortar." (6.) The pressure of pleuritic effusion. (7.) Extravasation of blood (apoplexy of the lung), or tubercular or cancerous deposits, or enlarged bronchial lymphatic glands, arranged along the sides of the air-tubes in their passage through the substance of the lungs. No disease shows more forcibly than cases of pneumonia, especially as regards diagnosis and treatment, that *every individual case requires to be made a special study as regards its individual history, progress, combination, and sequence of symptoms.*

Prognosis.—Whatever be the facts as to the order of events in pneumonia, whether the lung affection is a mere localisation of a blood-disease, or whether the undoubted blood-disease of the developed stage is merely produced by absorption from the inflamed lung, it is certain that the usual course of pneumonia is, that—(1.) There is an early period of intense fever, ceasing of itself at a tolerably determinate time if no complication be present. (2.) There is a later period of lung hepatisation, which softens down during a period of moderate fever, and is expectorated or absorbed. There are therefore two periods in pneumonia, both having their dangers. The intense early fever may kill by its intensity; the exudation may kill subsequently, by apnœa; or it may contaminate the blood, during softening, to such an extent as to lead to renewal or increase of the fever and inflammation of other parts, or to coagulation of the blood in the heart or great vessels. Each period has its own dangers, and must have its own treatment (PARKES). The duration of a case of pneumonia varies from seven to twenty-one days. In simple uncomplicated cases the crisis is reached about the fourth or sixth day (WUNDERLICH, PARKES, METZGER). The progress of pneumonia is more rapid in the aged than in the adult. More generally, however, taking all its forms, cases of pneumonia terminate between the seventh and the twentieth day. Defervescence may begin between the third and fifth day, and from that to the seventh or ninth: but the belief in *certain* critical days is getting less and less. The death-rate from pneumonia is much higher in armies than in civil life. Between the ages of fifteen and forty, acute, *single, or croupous pneumonia, uncomplicated,* is not a dangerous disease. Recovery is the rule; and it is often rapid. *Secondary pneumonia,* on the other hand, or pneumonia as an intercurrent affection, particularly to measles, chronic camp diarrhœa, and malarial toxæmia, is a dangerous lesion, and one apt to be fatal. Pregnancy is an unfavourable condition, especially during the first six months. After fifty years of age the danger increases. Between forty and fifty years about one in five die; after sixty years one in two. Double pneumonia is always dangerous. A knowledge of the range of body-heat in a case o pneumonia indicates,—(1.) The degree of the affection and its danger; (2.) improvement or relapses; (3.) the completion of the disease-process; (4.) the certainty of convalescence and recovery; (5.) the continuance of unresolved or lurking lesions, and the supervention of sequelæ. The intensity of cases of pneumonia is to be judged of by the occurrence of the phenomena of temperature, pulse, and respiration, so as to call all cases slight that fall *below*, and all cases severe that are *above* those averages in which *the temperature records* 104°, *the pulse more than* 120, *and the respirations more than* 40 *in the minute during the height of the disease.* It is only by a rigorous application of some such rule to the cases of pneumonia which are grouped together to furnish statistical data, as a guide to treatment, that anything like trustworthy results can ever be obtained. Such results have not yet been obtained. The disease is attended with extreme danger to aged persons and to drunkards. In advanced life the mortality is between sixty and seventy per cent. Absence of all sputa is an unfavourable sign; so also is the appearance of a very dark brownish-red, commonly called *prune juice*, expectoration. It signifies a poor state of nutrition, fragility of the pulmonary capillaries, and a general cachectic condition of the individual. Copious liquid serous sputa are also significant of evil. Scanty expectoration during resolution is not specially significant of any danger if the dulness continues to disappear; but if expectoration fails while gurgling sounds are heard with each respiration in the chest, palsy of the bronchial tubes, with *œdema* of the lungs, are indicated, and the approach of death. Albuminuria is an unfavourable sign. While the mortality of all the cases recorded by Dr. Parkes was *four* in *thirteen*, or 30·7 per cent., those with albuminous urine were 50 per cent., and with non-albuminous urine 14 per cent.; "*therefore albuminuria occurring before resolution is an unfavourable sign;* and the very frequent occurrence of albuminuria in sthenic pneumonia is one of the most interesting facts in its clinical history. In no disease seen in this country, except, perhaps, in *maculated typhus*, is albuminuria so common as in *pneumonia. Bronchitis* of the large tubes, chronic phthisis, and emphysema present a most marked contrast to pneumonia in this respect." Common pneumonias terminate by—(1.) Restoration of the consolidated texture to a healthy state (*resolution*); (2.) desiccation and caseation of the new material, followed by softening and breaking up of the mass, with the involved tissue forming a cavity; or, (3.) smaller circumscribed patches forming abscesses; (4.) direct death of all the parts involved, as in gangrene of the lung.

Treatment.—One of the most discordant topics in the Science of Medicine seems still to be with some—the treatment of pneumonia. At one time (about fifty years ago) large bleedings were the rule. But Laennec and Louis were early convinced that large bleedings were by no means successful, and that in some cases they were absolutely injurious. The same difference of opinion was held with respect to large doses of the *tartrate of antimony.* At the same time we have it in our power materially to modify the course and to shorten the duration of pneumonia by the judicious employment of *blood-letting, leeches, tartar emetic, certain salines,* and *opium* (Parkes). The correlation of the body-temperature, the number of the pulse, and the respirations *per minute*, furnish the best guide as to the course to be pursued in the treatment of pneumonia. If the pneumonia is not intercurrent to some other disease, but happens idiopathically in a person otherwise healthy; if the mean body-temperature remains below 104° Fahr., the pulse does not exceed 120 beats in the minute; and if the respirations never exceed forty in the same time, the case must be regarded as a typically favourable case, which will certainly begin to get well when the cycle of the pneumonic process is complete—*i.e.*, in from three, four, seven or eight, to twelve days *without any special medicinal treatment;* but with the careful management implied by *good nursing, absolute rest in the horizontal position,* and *a strictly antiphlogistic diet.* In such cases it is sufficient to keep the patient in bed, in a well ventilated room, whose temperature should be kept as near 60° Fahr. as possible, to relieve thoracic pains by *leeches,* or *fomentations, linseed and mustard poultices;* to relieve thirst by *alkaline* or *acidulated* drinks; and to give frequently repeated small quantities of *nutritious fluid food* in the form of *milk, milk and eggs, oatmeal gruel,* or even *meat soups.* The natural history of an uncomplicated pneumonia shows that it has a definite cyclical course; that if left to itself in a vigorous patient, uncomplicated by any other ailment, and if it be of the typical intensity as here indicated, it almost always ends in recovery. The expectant mode of treatment of the Vienna School confirms this view, as the statistics collected by Balfour, Magendie, Skoda, Niemeyer, Schmidt, and Legendre fully prove. In cases of average severity there is a decided tendency to amelioration of all the symptoms by the *eighth day.* The results obtained by the *Homœopaths* have also shown this to be the case. Unless warranted, by certain special indications, active interference has an unfavourable effect upon the course of pneumonia (Niemeyer); and when treated by blood-letting, as a rule, it more often terminates fatally than where no venesection has been practised (Diett). Blood-letting is no specific. It does not cut short a pneumonia; and in epidemic pneumonia it is hardly possible to bleed the patient without rendering him worse. But much must be left to the discretion of the practitioner. That there are cases in which the patient can only be saved by general blood-letting, everybody must admit. The cases of Louis and of Dr. Alison of Edinburgh establish the propriety of bleeding early, if blood-letting is to be used at all. As a general rule, the earlier the inflammatory state is detected (if possible before the third day—Alison) the more likely will bleeding be followed by well-marked beneficial results, the disease will be sooner cured, and the convalescence more rapid and perfect. Blood-letting in pneumonia should only be had recourse to when the following special indications for it arise:—(1.) When the pneumonia attacks a vigorous and hitherto healthy person, is of recent occurrence (not exceeding fifty or sixty hours after the rigor), the temperature being at or exceeding 105° Fahr., and the frequency of the pulse as much as 120 per minute. The object of such venesection is to reduce temperature, lessen the frequency of the pulse, and so lessen the danger from the violence of the fever. (2.) When collateral œdema in the portions of the lung unaffected by pneumonia is causing danger to life, the presence of the blood is reduced by bleeding, and further transudation of serum into the air-vesicles is prevented. When the frequency of respiration is due to the pneumonic process alone—not to fever or to pain—and as soon as a serous foamy expectoration appears, together with a respiration of more than forty or fifty breaths a minute at the commencement of pneumonia; and when the rattles in the chest do not cease for a time, after the patient has coughed as well as he can, copious blood-letting is indicated to reduce the mass of blood, and to moderate collateral pressure. (3.) When there are symptoms of cerebral pressure indicated by stupor or transient paralysis.

Antimony, in the form of *tartar emetic*, is another remedy which has gone through several extremes of administration in the treatment of pneumonia. Rasori introduced the practice of giving it in large doses, and Laennec was so dissatisfied with his own results of bleeding that he adopted Rasori's method. Few, if any, now believe in the usefulness of such large doses of antimony; and such a use of *tartar emetic* has justly fallen into discredit. When *antimony* is cautiously administered as a sedative, or diaphoretic and expectorant, in doses of *the eighth* or *twelfth of a grain*, every *two* or *three* hours, and in doses of a *sixth* or a *quarter of a grain*, in the more sthenic attacks, when the patient is strong and plethoric, it is a valuable remedy. It may be combined with *compound tincture of camphor*, or *dilute hydrocyanic acid*. It is contra-indicated in gastric or gastro-enteric irritation. There are cases where the remedy produces severe depression and nausea—cases in which the stomach will retain *no* food, not even water when given alone. Under such circumstances the administration of *alcohol* in stimulant doses often repeated is called for, and may require to be combined with *ammonia*, *chloric ether*, and *camphor*. *Alcohol* is especially indicated by the occurrence of delirium (if vascular excitement does not exist); by a weak, rapid, or dicrotic pulse; by signs of collapse; by age and general debility of the patient; or in cases of pneumonia secondary to those diseases which it frequently complicates.

A rational treatment of pneumonia is one which must secure to each ase its own individuality and consideration. *Bleeding*, *antimony*, and *listers* may be demanded in one case; *quinine*, *opium*, and *wine* in the ext; a third may require but little interference beyond *a well-regulated iet*, *moderate stimulants*, and *absolute rest*. It is the proper *combination* of emedies, not a single agent or mode of practice, which must be sought or in its treatment (LAWSON). The so-called "rational" or "restorative" ode of treatment is the one to be adopted in cases of *sthenic pneumonia* haracterised by heat, *above* 104° Fahr., dryness of the skin, a full esistant pulse, *over* 120, rusty-coloured expectoration, *great oppression of e breathing*, and to the extent of *beyond* forty respirations per minute, lood-letting, had recourse to at the beginning of the attack, in the age of congestion before the *fastigium* of the pyrexia (not later than rty or fifty hours after the initial rigor), not only affords immediate lief to the breathing, but removes the extreme tension of the vascular stem, and promotes secretion. The most suitable time is that during e evening exacerbation (HUSS). These indications for blood-letting ould be decidedly marked before it is undertaken; and the amount blood to be lost varies in each case, according to the oppression of eathing, the type of the disorder, and the constitution of the patient; well as to whether or not there is any prevalent epidemic tendency associated with the pneumonia. It is seldom necessary to draw more than from *ten* to *sixteen* ounces; and *eight* or *ten* ounces will usually suffice. After bleeding, the pain in many instances ceases, expectoration takes place more easily, and alters in character; the skin becomes moister, and evidence is afforded of the action of remedies which before proved inoperative (Fuller). But in many cases it is either unnecessary or inexpedient to let blood—inexpedient chiefly because of the constitution of the patient being shattered by excesses, constitutional disease, anxiety, and mental distress. Under such circumstances, it impairs the strength, leads to greater impoverishment of blood, arrests the actions on which the absorption of exudation-matter depends, and induces a tardy convalescence. Bleeding ought not to be employed *after* extensive consolidation of the lung has taken place.

The cases in which *tartar emetic*, in small doses (one-eighth of a grain) fails to exercise a curative action are those in which hepatisation proceeds with extreme rapidity; in which crepitation either does not exist at all, or is of very short duration, giving place after a few hours to intense tubular breathing; and those which are marked by extreme depression almost from the first. *Salines* and *stimulants* with *opium*, if necessary, are found to be the most efficient means of treatment in such cases. Of other proposed remedies, the following may be noticed, namely:—(1.) *Aconite* is only to be given at the very outset, or first stage of pneumonia, from ♏iii. to ♏v. *of the tincture* of B. Ph. every four hours (Wilks); or from ♏ii. to ♏iii. every three hours at the outset of the congestive stage (Reith, *Edin. Med. Journ.*, Oct., 1868, p. 905). Half a drop to one drop *of the tincture* in a teaspoonful of water every ten or fifteen minutes for two hours, and afterwards to be continued every hour, has been useful in *febrile catarrh* and *catarrhal croup*, and may be useful in some of the phases of pneumonia, especially to subdue increased action of the heart and circulation, and to reduce the abnormally high temperature. Care must be taken that the remedy is not combined with alkalies (Ringer). (2.) *Carbonate of ammonia.*—A combination of *ammonia* and *chloroform* (where antimony in small doses is not indicated) may be taken every three or four hours. Four grains of *carbonate of ammonia*, twenty minims of the *spirits of chloroform*, in five drachms of *camphor water*, combine the beneficial properties of both stimulants (Waters). It is indicated when the symptoms generally resemble those of *delirium tremens*, when the pulse is small and weak and quick, or if delirium is present. (3.) *Digitalis* has of late years been employed by Oppolzer, Traube, Schneider, Niemeyer, and others, as one of the best agents for the relief of the febrile symptoms. It lowers the temperature and diminishes the frequency of the pulse, without exercising so weakening and depressing an effect upon the system as bleeding. A pulse of 100 to 120 in frequency is an indication for its employment—a less frequent pulse doe not require it. It is usual to give it in the form of *infusion*, combined wit the neutral salts of *nitrate of potass* and *soda*. *Digitalis* may be advantageously combined with *ergot*, in the proportion of four drachms of th *fluid extract of ergot* to one drachm of *digitalis*, with six grains of the *aceta of lead* in two ounces of *cinnamon water*, of which a table-spoonful is to b given every two hours till the blood disappears from the sputa (Well

New York Med. Record, Vol. XV., No. 17). (4.) *Opium* is of great service. It may relieve the pain of the side, and diminish the distressing cough. It is also of use in warding off delirium, combined with stimulants and nourishment. It ought to be given when there is want of sleep, restlessness, and staring of the eyes, with slight tremors of the hands. It may be used hypodermically. (5.) *Hydrochlorate of ammonia* is of use in congestion of the lungs, in the advanced stages of *pneumonia*, and in the cough of old age. One drachm is to be combined with one drachm of *extract of liquorice;* two drachms of *spirit of sulphuric ether*, and four ounces of *water*, of which a table-spoonful is to be given for a dose every two or three hours (WARING). Or it may be given in *one* or *two* grain doses every hour or two, mixed with equal parts of *glycerine* and *water.* (6.) *Iodide of potassium*, in the advanced stages of pneumonia, appears to promote the absorption of effusions and indurations. In severe cases of broncho-pneumonia, combined with *tincture of hyoscyamus* and *ammonia* it may reduce the frequency of the respiration in a marked degree. (7.) *Quinine* is of use chiefly in the advanced stages when the patient is old, the constitution debilitated, and typhoid symptoms prevail. It is best given in combination with *sulphuric acid*, in doses of *five grains* every third hour. The pulse ought to become slower and steadier under its use, and the respiration freer. It should be given in doses of *two grains* every two hours, or in two or three *ten grain* doses within a few hours, whenever danger threatens from *excessive elevation of the temperature of the body.* (8.) *Alkalies.—Bicarbonate of potash*, largely diluted in mucilaginous liquids, five grains up to thirty grains in each dose,—four, six, or even eight doses being given in the twenty-four hours (to the extent of two to three drachms of the salt per day), alters the character of the expectoration by the second or third day of its use. The viscid sputa become resolved; the fine bubbles become coarse and large; the red colour of the expectoration disappears; its tenacity of adhesion lessens, so that it is brought up easily, and the cough becomes moist, soft, and expulsive. Thus the alkali acts as a sedative, allaying the cough and abating the congestive state on which it depends. The white pasty fur over the tongue dissolves away in an increased flow of saliva. The urine becomes alkaline, and the physical signs of pneumonia become resolved. It has also a marked action as a nerve sedative. A drachm dose in water produces an agreeable, tingling, numbing sensation, in the lips, cheeks, and other parts, extending gradually to the lower limbs. It is especially indicated where the pneumonia is associated with rheumatism. "Flying blisters," applied for four or six hours, so as to redden the skin (not for suppuration), is a valuable auxiliary. Suppuration from blistering is to be avoided as exhaustive and prejudicial (Dr. JOHN POPHAM, *Brit. Med. Journ.*, Dec. 28, 1867, p. 586). (9.) *Stimulants.*—The treatment by repeated alcoholic stimulants alone is but the opposite extreme of practice to excessive blood-letting. It has now found its level and appropriate place as a valuable aid to other remedies, especially in patients whose constitutions are depressed under the influence of life imposed upon them by the "*great town system.*" When pneumonia tends to death by exhaustion, as the disease advances; when exudation has been excessive, the fever prolonged, or the constitution debilitated before the attack, such a state of complete adynamia requires the use of stimulants, in the form of large doses of *camphor, mint, benzoic acid, alcoholic drinks;* and in addition, concentrated soups and milk are called for in small quantities, given often. In old persons, stimulants, with generous diet, preparations of *quinine* and *iron,* are indicated from the very commencement. *Alcoholic stimulants* are rarely required in young persons, or in previously healthy adults.

Some local applications require special notice, namely:—

(1.) *Cold Applications.*—The chest of the patient (the affected side in particular) is to be covered with cloths that have been dipped in cold water and well wrung out. Compresses thus made must be repeated every *five* minutes. After a few hours the patients experience a material relief. The pain, dyspnœa, and frequency of the pulse are reduced, and the body-temperature may go down an entire degree (NIEMEYER, SMOLER). The treatment is not merely palliative, but tends to shorten the duration of the illness. Few cases delay to improve beyond the seventh day, many improve on the fifth, and a very large number as early as the third day. The remedy is justly held in repute for *meningitis* and *peritonitis,* and hence it may fairly claim to have a similar good effect in *pneumonia.*

(2.) *Linseed Meal Poultices.*—On the other hand, heat combined with moisture in this form, made as light and soft as possible, and sufficiently large as thoroughly to inclose the affected side, and changed as often as it gets unpleasant, not only affords present relief, but exercises a favourable influence on the course of the disease. The skin may be at the same time stimulated by *mustard,* or *terebinthine applications* added to the poultice.

(3.) *Turpentine.*—Hot turpentine fomentations may be applied over the affected side also with advantage.

(4.) In children it is sometimes of importance to be able to induce cough and expectoration. This can be facilitated by using a spray of a weak warm solution of *carbonate of soda,* to which a little *glycerine of carbolic acid* is added. It is a valuable resource in promoting the expulsion of the plugs of tough mucus which tend to accumulate in the bronchia tubes of children with bronchopneumonia (Dr. BURNEY YEO, *Brit. Med Journ.,* Dec. 7, 1878).

Time is an important element in the cure. As particular phases in th course of the pneumonic cycle call for special treatment, so much mor successful will the management of the case be if the indications for in terference are understood and acted upon. The natural course of tl disease is to go on rapidly to a spontaneous favourable termination, by process of resolution through which the solidified lung-tissue regains i natural and healthy state, independently of remedies. Those remedi that have been specially noticed may each be of service in safely a surely controlling the circulation when the indications for treatment a short of requiring the agency of blood-letting. Their effects must watched so that they do not lower the vital powers nor disturb t digestive functions. The indications for nourishment, properly adjust to the circumstances of each case, and each stage of the case, are of pa mount importance from the first; and the happy issue of a case of pn monia depends quite as much upon early and properly feeding the pati as a case of typhus or enteric fever does. A milk diet should be gi from the commencement, to be followed by beef-tea and meat-stock so s as the disease advances and the pulse softens. Stimulants cannot be regarded as the equivalent of food (CLYMER).

GANGRENE OF THE LUNG.

LATIN EQ., *Gangræna;* FRENCH EQ., *Gangrène;* GERMAN EQ., *Brand—* Syn., *Gangrän;* ITALIAN EQ., *Gangrena.*

Definition.—*Disintegration and breaking up of the tissue of the lung. Shreds of lung-tissue may here and there be distinguished; but the whole mass becomes converted into a heap of amorphous granular matter, of a yellowish-brown or black colour, mingled with drops of oil. The tissue becomes soft and flaccid, in some parts perfectly liquescent, and generally emits a fetid smell.*

Pathology.—(*a.*) *Causation.*—Diseases of the lung going on to gangrene, may result from cerebral disease; and a tendency of pneumonia to pass into gangrene is promoted by the existence of disease in the brain. The frequency of gangrene of the lung in epileptic cases is recorded by Cruveilhier. The insane are prone to the disease when the bodily health has suffered, or where, as in maniacs, a greatly depressed state of the animal functions succeeds to inordinate nervous excitement.

The following conditions are those under which gangrene of the lung has occurred:—(1.) Pulmonary diseases, as a result of *Pneumonia,* acute and chronic; tuberculisation; cancer; hæmorrhage; hydatids (WALSHE); apoplectic foci in the lung; acute and chronic dilatation of the bronchial tubes; pleuritis; bronchitis (SILFVERBERG). (2.) Other thoracic diseases —*Cardiac:* the result of acute endocarditis of the right side of the heart; *Mediastinal:* tumor; *Aortic:* aneurism; *Animal venoms:* stings of certain insects. (3.) Diseases in which the blood is materially changed—*Morbid poisons:* Glanders; exanthemata; typhoid fever; Bright's disease; alcoholism; scurvy; purpura; septicæmia; pyæmia. *Other poisonous agents:* poisonous gases; mercurialism. (4.) Chronic abdominal disease (SILFVERBERG). (5.) Perverted innervation, as in epilepsy; insanity; organic cerebral disease. (6.) Traumatic.

The termination of acute sthenic pneumonia, in an otherwise healthy person, by gangrene, is one of the least frequent terminations of that disease.

(*b.*) *Morbid Anatomy.*—The extent to which death of lung-tissue takes place varies much. Nearly the whole of a lung may pass at once into a gangrenous mass; or only a small portion in the centre of an exudation will die. In every case there is the formation of a slough, its liquefaction, and the formation of a cavity. Sometimes a line of separation between dead and living parts is attempted to be formed; in other cases no vidence exists of any limit to the extent of destruction. This diffuse orm of gangrene is generally associated with cases in which the blood is reatly changed, as in typhus fever, Bright's disease, mercurialism; or in which the nervous functions are impaired, as in maniacs, or in the paralysis f the insane.

The **Symptoms** vary according as the lesion is *diffused* or *circumscribed:*— .) In the diffused form the progress of the case is extremely rapid. tter prostration of strength; oppressed breathing; a small, weak, and frequent pulse; pallor and anxiety of countenance; and all the general appearances of intense adynamia, characterise the disease. The expression of the countenance becomes small, pinched, contracted, haggard, ghastly, miserable, and death-like; eyes sunk and void of lustre; patient squeamish and languid, with occasional vomiting, and a feeling of indifference to all external objects. There is frequent but feeble cough, with profuse, frothy, and diffluent expectoration, of a peculiar greenish colour and intensely fetid gangrenous odour. The power to expectorate is soon lost. The vital energy seems utterly oppressed or exhausted; the pulse fails, the features collapse, and the patient rapidly sinks—death generally occurring from apnœa. (2.) In the *circumscribed* forms of gangrene the train of symptoms varies at different periods of the disease. At first, signs of pulmonary congestion are coupled with an amount of prostration quite out of proportion with the apparent extent of local lesion—a prostration which is sometimes the only prominent feature in the case. After a time expectoration commences; at first, muco-purulent, rarely bloody in adults, frequently so in infants and children (if they expectorate at all), and emitting a disagreeable odour the moment a communication is established between the bronchial tubes and the dead tissue. The sputa then lose their muco-purulent character, and become extremely liquid, sero-purulent, and of a dirty greenish, yellowish-brown, or ash-grey colour. On standing they separate into layers, and a thick sediment subsides, in which elastic fibres may be detected. They exhale an odour distinctly gangrenous, while the breath acquires the same offensive putrid smell. Its fetor is one *sui generis.* The pulse becomes feeble and rapid, with evidence of great and increasing prostration. Emboli may be carried from the district of pulmonic lesion, and so originate septicæmic abscess elsewhere. The patient passes rapidly into a state of collapse, and sinks in a few hours or days, without the occurrence of any other change, unless profuse hæmorrhage occurs, which terminates in death (WALSHE, FULLER).

Treatment.—The chief reliance is to be placed in all forms of stimulants, combined with such tonics as *bark* or *quinine* in full and repeated doses. Repeated doses of an ounce of *yeast,* as well as the influence of *chlorate of potass,* are suggested by Dr. Walshe. *Ammonia* has not been found so useful as the *mineral acids.* Inhalation of the vapour of *turpentine,* poure upon boiling water, exercises a distinctly remedial power (SKODA) Gargles of Condy's fluid should be freely used, and antiseptic inhalation as described under "Phthisis." The *mineral acids,* especially *nitro-muriati* combined with *quinine,* are the main remedies in the chronic state this disease. A generous diet, easily digestible, with as much malt liquo as can be taken, are also recommended.

CONGESTION OF THE LUNGS (ACTIVE AND PASSIVE), ŒDEMA, HÆMORRHAGE, AND PULMONARY EXTRAVASATION.

Definition.—*An hyperæmia or over-fulness of blood in the capillaries of lung, resulting in the discharge of blood by expectoration (hæmoptysis); or an extravasation of blood from capillary rupture into the air-cells, termi bronchi and the interstices of elastic tissue (pulmonary apoplexy).*

Pathology.—(*a.*) *Causation.*—*Active congestion* may result from increased cardiac action, and generally from those causes which have been stated to bring about bronchitis or pneumonia. Mitral disease, or pressure of tumors, may give rise to *mechanical congestion;* while *passive congestion* occurs during the course of specific febrile affections and depressing conditions of the heart's action. *Hæmoptyses* are hæmorrhages occurring through the air-passages. The occurrence of *hæmoptysis* is always suggestive of organic disease, such as engorgement of the pulmonary vessels, the giving way of pulmonary tissue, indicating disease of the heart, or pulmonary phthisis, or pneumonia, or thoracic aneurism, or cancer of the lung. Consequently *hæmoptyses* have attracted the attention of medical men ever since medicine as a science began to attract attention. It may be of so-called idiopathic occurrence, as from variations (sudden) of atmospheric pressure, ascending high mountains, or descending in diving-bells, violent straining efforts, or from plethora. In such cases "there is usually some latent mischief in the chest—some local cause of pulmonary congestion—some mechanical interference with the capillary circulation through the lungs" (FULLER, WALSHE). Spitting of blood may precede for years the fatal development of pulmonary phthisis, with which it is often connected. Hæmoptysis is often hereditary.

(*b.*) *Morbid Anatomy.*—Observations on the morbid anatomy of the lung have led to the rejection of the belief that large vessels are the usual source of hæmoptyses, and that the vessels in the condensed tissue surrounding the cavity are obliterated. The anatomical sources of *hæmoptyses* are:—(*a.*) Rupture of pulmonary arteries, or branches of pulmonary veins; or bursting of an aortic aneurism into the pulmonary passages; or exudation of blood through mucous membrane of the bronchi; or violent hæmorrhages from pulmonary apoplexy (LAENNEC). (*b.*) From bronchial membrane without co-existent tubercle; from pulmonary apoplexy; from the eroded blood-vessel in a trabecula of a cavity (ANDRAL). (*c.*) Altered collateral circulation surrounding tubercle-growths, so that vessels remaining distended readily burst, while others are compressed by condensed portions (ENGELSTEDT). (*d.*) Molecular rupture of the capillaries of the parenchyma (WALSHE). (*e.*) From branches of the pulmonary artery situated in the condensed walls of cavities, especially in dilated bronchial cavities, either through a slit or an aneurismal dilatation. They are due partly to ruptures of small sac-like aneurisms developed on branches of the pulmonary artery running in the walls of cavities; partly to small dilatations (*ectasias*) of similar vessels (ROKITANSKY, RASMUSSEN). (*f.*) Ruptures of small capillary vessels in the walls of recent cavities from little excrescences on them (HERARD and CORNIL). (*g.*) Rupture of capillaries, either from over-distension or morbid delicacy of their walls—a result of perverted nutrition (NIEMEYER). Every cavity in the lungs whose walls are formed by condensed pulmonary tissue, containing non-obliterated blood-vessels, may be the seat of aneurisms or aneurismatic dilatations, with consecutive ruptures (RASMUSSEN). Aneurisms may occur in the largest as well as in the smallest cavities; hence they are often easily overlooked. Profuse and fatal hæmoptysis almost always proceeds from cavities irrespective of their size.

Another source of *hæmoptysis* has been traced to systemic aneurisms, (as of the aorta), bursting into a bronchus or into pulmonary tissue. It has been noticed as an early symptom of thoracic aneurism, either as evidence of actual rupture, or as producing by pressure such disturbances of the circulation in the lungs as leads to hæmoptysis. Mr. Liston, the celebrated English surgeon, during the space of eight months before his death, brought up small quantities of blood under the influence of such disturbance of the circulation in the lungs by pressure, which first led to its being recognised as a symptom of aneurism. It may also result, not only immediately after penetrating gun-shot wounds of the chest, in proportion to the greater or less degree of direct violence to the lung-tissue, but may occur long after the external wound caused by a gun-shot penetrating the lungs has healed up. Such may be called secondary hæmorrhages of the lung after gun-shot injury.

Pulmonary Extravasation, or Apoplexy of the Lung.—(*a.*) *Causation.*—The worst cases are generally connected with extensive disease of the heart, especially cases of disease of the mitral valve, in which clots (embolic) are found in the arteries leading to the infarctions (ROKITANSKY, GERHARDT, NIEMEYER). The *emboli* which block the artery in disease of the heart do not come from the greater circulation, like the emboli of metastatic infarction, but from the right side of the heart, and especially from the right auricle, in which clots usually exist, firmly entangled in the *trabeculæ* formed there as a result of the slowness of circulation. It is the particles of such clots which obstruct the pulmonary artery. The *débris* from these cardiac clots is generally much greater than from the aortic or systemic circulation, and the resulting infarction from *embolism* is also much more extensive than those from *metastatic* sources. The infarctions of heart disease are usually found at or towards the roots of the lungs, while the *infarctions* of metastasis are generally found near the periphery, and generally involve the pleura in a local pleuritis. They are often also mixed together in heart disease. Why the obstruction of a branch of the pulmonary artery by *embolism* should produce capillary hæmorrhage within a certain area of the obstructed vessel has received several explanations by Rokitansky, Virchow, Ludwig, and Niemeyer; but the explanations are not conclusive (see NIEMEYER, p. 156, Vol. I.)

(*b.*) *Morbid Anatomy.*—It occurs as an induration caused by an incorporation of the infiltrated blood with its tissue. It is confined to minute and sharply-defined section of the lung at one or many point often bounded by the limits of a single lobule. The blood is extravasate partly within the cavity of the vesicles and terminal bronchi, and partl in the interstices of the lung. It generally proceeds from the capillarie pertaining to a single twig of the pulmonary artery. The induration may be black, brown, or red; and if scraped half-coagulated blood escape while the surrounding tissues are healthy, or more or less congeste The lesion has received the name of "*hæmorrhagic infarction*" or "*infarct* occurring either in the interior of the lung, when they are large, towards the periphery, when they are small in size and of the shape of t superficial lobule. The blood is generally coagulated, the liquid pa being absorbed. These *infarcts* vary in size from a hazel nut to a hen egg, of a blackish-red or dark chocolate colour, inelastic, and void of a On handling the lungs, the extravasations may be felt as hard knobs their substance. On section, they show a surface which is irregular in outline, coarse, and granulated, but sharply defined. When the infarction has existed for some time, it looks more pale and yellow than when recent, the colouring matter of the blood having become decomposed and absorbed, and at last the only trace of its existence may be a localised black induration.

Some modern views regarding pulmonary extravasation are embraced in the doctrines of *thrombosis* and *embolism*, which have been already stated. When the branch of the pulmonary artery is examined, within whose range a pulmonary infarction has formed, a clot is usually found by which its calibre has been more or less obstructed; but it is difficult to demonstrate this in small vessels. Such clots, from which the infarction arises, are now believed to be *embolic*—*i. e.*, they come from some region of the body. Being detached in whole, or bit by bit, from the place where they formed, and so swept into the current of the blood, they become impacted into a branch of the pulmonary artery too narrow to admit of farther passage (VIRCHOW, NIEMEYER). Thus, the so-called metastatic infarctions of the lungs arise from disintegrating *thrombi* found in peripheral veins.

Symptoms.—Hæmoptysis may take place suddenly, or be preceded for two or three days by a sense of heat, or a feeling of weight at the chest. The patient may suffer pain between the back and shoulders, or may labour under dyspnœa, palpitation, cough, or coldness of the extremities. At length a fit of coughing, or a tickling of the throat, is followed by the expectoration of blood. As much as ten pints of blood have been thus thrown up in forty-eight hours, and as much as thirty pints in a fortnight (LAENNEC). If the quantity thrown up be large, the patient feels oppressed at the præcordia, breathes with difficulty and with a gurgling sound, caused by the air passing through the viscid blood retained in the bronchi. This is shortly followed by increasing weakness, even to complete prostration. In still more severe cases, as the blood flows the patient turns pale, his countenance becomes anxious and strongly expressive of terror; or he falls into a complete syncope, which sometimes has a curative effect. After the patient has lain for a time in a state of depression, a reaction takes place. In some instances the effusion is so sudden and so considerable that the patient dies suffocated. Usually, hæmoptysis recurs after a time. It is then remittent, each attack being ushered in by a violent fit of coughing.

Hæmoptysis due to *aneurism*, or to *ectasia* of a branch of the pulmonary rtery, usually occurs suddenly, without either the patient or the physician having the slightest suspicion of its approach. During a fit of coughing r violent bodily effort, blood rushes from the nose and mouth, and the atient may die asphyxiated in the course of a few minutes.

The symptoms of pulmonary apoplexy are mainly those of chronic heart isease, and especially of the valves. But there occurs a sudden difficulty f breathing which threatens suffocation, some expectoration of blood, ome mucous rhonchus and cough, and a total inability for a time to lie own. The formation of one or more hæmorrhagic infarctions may then e inferred. On percussion that portion of lung which corresponds to e seat of the disease returns a dull sound—a sign of circumscribed condensation, not unfrequently followed by signs of pneumonia or of pleurisy. In the most severe cases the patient is more oppressed in his breathing; he is obliged to be supported by pillows, and his head may fall forward, while his face is purple, his pulse small and frequent. If the extravasation is very extensive, the patient appears to be almost instantaneously destroyed. There are signs of cardiac thrombosis of the right side of the heart which are also characteristic of hæmorrhagic infarction. These are, "sudden irregularity of the pulse, a sudden widening of cardiac dulness, and sudden cessation of cardiac murmurs previously heard" (GERHARDT, NIEMEYER).

Diagnosis.—It is important to distinguish *hæmoptysis* from *hæmatemesis.* The diagnosis between them is difficult, because while the contents of the stomach are always rejected in *hæmatemesis*, they are frequently rejected in *hæmoptysis* also. The stethoscope, however, greatly assists in determining the seat of the disease. Blood is generally found in the stools in cases of *hæmatemesis,* while it is, for the most part, wanting in *hæmoptysis.*

Apoplexy of the lung may be distinguished from *hæmoptysis* by the dulness on percussion, by the tubular breathing, and by the subsequent fever and pneumonia.

Prognosis.—*Pulmonary apoplexy* is always a grave affection; but should the patient survive for a few days, and the effusion be inconsiderable, and the subsequent inflammation slight, he may recover. The prognosis in *hæmoptysis* is unfavourable eventually, so far as freedom from organic disease is concerned; and danger is more or less imminent in proportion to the amount of blood lost, and the frequency of its recurrence. *Pulmonary congestion* is serious in proportion to its extent, and to how far it complicates other morbid conditions, as that of typhus or other fevers.

Treatment.—The medicines most useful in *hæmoptysis* are, the *bitartrate of potash, in doses of a drachm,* repeated every four or six hours, to each dose of which may be added a quarter to half a grain of *opium.* The *mineral acids*, as the *infusion of roses*, with diluted *sulphuric acid,* in doses of from three to five drops, combined with *opium* or *morphia*, every four or six hours. One to three grain doses of the *acetate of lead,* every four or six hours, may be given, with *half a grain of opium* to each dose, or combined with *dilute acetic acid* and *laudanum* (A. T. THOMPSON). The *muriate of soda,* in doses of half a drachm to a drachm, is in estimation with some practitioners on the Continent.

In *atonic hæmoptysis, ergot* is said to be of service, especially in the following combination:—

℞ *Ext. Ergotæ Liq.*, ʒii.; *Tinct. Digitalis*, ʒii.; *Acidi Gallici.*, ʒi.; *Magnes. Sulph.*, ʒvi.; *Acid. Sulph. Dil.*, ʒi.; *Infus. Rosæ Acid.* ad ℥viii.; *misce.* A sixth part of this mixture to be taken every three hours, till hæmorrhage ceases (DOBELL, WARRING-CURRAN). Wunderlich also recommends the *secale cornutum*, in doses of from five to ten grains, to be pushe until a numb sensation is experienced in the fingers and toes.

The hypodermic injection of *ergotine* is by far the most efficacious o styptics in hæmoptysis. A grain of (Bonjean's) *ergotine* should be injecte on the back of the side of the thorax whence the bleeding comes, an repeated every two to four hours; and fifteen minims of tincture of *digitalis* should be given every three or four hours, its effect on the pulse being watched (Coghill).

When hæmoptysis is connected with amenorrhœa, preparations of iron often succeed when other remedies have failed. Two grains of the *sulphate of iron*, with one drachm doses of the *sulphate of magnesia*, three times a day, often restore the menstrual secretion, and cure the hæmoptysis. It is in this form of amenorrhœa that iron is most successful. When hæmoptysis depends on disease of the heart, cupping from the chest, or moderate bleeding from the arm, combined with the use of the *bitartrate of potash*, or the mineral acids, to which should be added five to ten mimins of the *tincture of digitalis*, to give steadiness to the irregular, turbulent, or rolling action of the heart. Repeated *dry-cupping*, aided by the application of *ice* down the spine, and by the internal administration of *full* doses of *digitalis*—half a drachm of the tincture, or a grain and a half of the powder (Fuller). It is chiefly as an adjunct to other means, when hæmoptysis is attended with much vascular excitement, that digitalis is of service. Full and frequent doses of *gallic acid*, or *lead and opium*, may be given, if the circulation is much accelerated; and of *spirits of turpentine*, in half-drachm doses, if the bleeding is unattended with vascular excitement. The *gallic acid* is more adapted for chronic cases, and should be given every hour, in *eight* or *ten grain* doses, until the hæmorrhage is subdued, or till a dark-green colour in the sputa indicates its action on the system (Fuller, *op. cit.*, p. 265). Its efficacy appears to be increased by combination with *sulphuric acid*, and may be conjoined with *opium* or *digitalis*.

The following formula is a useful one:—

℞ *Acid. Gallic.*, gr. xxx.; *Acid. Sulph. Dil.*, ʒi.; *Liquor. Opii. Sedat.*, ♏ xxx.; *Infus. Rosar. Co.*, *f*℥vi.; *misce.* Two table-spoonfuls for a dose every three or four hours (Dr. L. Earle).

Absolute bodily and mental rest must be insisted on throughout, in cases of *pulmonary infarction.* Stimulation must be resorted to in the first instance; after which the *mineral acids*, or *super-acid salts*, appear to offer the best hopes of recovery. If the apoplexy be secondary, and depends on disease of the heart, *digitalis* may tranquillise its excitement.

Dietetic and General Treatment.—The patient should be placed in bed, with his head and shoulders raised; the window should be partly open, so as to keep the room cool. It has been recommended that the air respired should pass through a respirator containing ice; and it is common to place a bowl of ice immediately before the patient, so that he may suck small portions as often as he can. A large pad of *spongiopiline*, soaked in *chloroform*, should be laid on the chest, over the locality of bleeding. The bedclothes should be light but sufficiently warm. The diet should be slops, and these slops cold; and if cooled to a low temperature by ice, so much the better. No red meat, nor beef-tea or stimulants, are to be given; but milk, chicken broth, jelly, and iced lemonade.

EMPHYSEMA.

LATIN EQ., *Emphysema;* FRENCH EQ., *Emphyseme;* GERMAN EQ., *Emphysem;* ITALIAN EQ., *Enfisema.*

Definition.—(1.) Vesicular Emphysema.—*Relative increase of air in few or many air-cells of the lungs, causing a misshapen enlargement of them, by dilatation or rupture of septa, and the blending of several into one or many great cysts. It is attended with gradual effacement of the functional blood-vessels distributed over their walls, anæmia of the lung in the affected parts tending to dilatation of the right side of the heart, with anasarca.* (2.) Interlobular Emphysema.—*Air infiltrating the meshes of the sub-pleural and interstitial connective tissue of the lungs.*

Pathology.—(*a.*) *Causation.*—Some confusion has arisen from the use of the term "*Emphysema*" (which means the presence of air in the connective tissue) to designate dilatation of the air-cells of the lungs. Here the air is where it ought to be; but the air-cells are large and misshapen. Limited in this way to the vesicles of the lung so misshapen, this form of disease was named by Laennec "*vesicular emphysema.*"

The disease forms one of the most serious complications of *bronchitis;* and the tendency to vesicular emphysema appears to be hereditary—60·4 per cent. acknowledging hereditary transmission (FULLER). Several theories have been put forward to explain the mechanism which produces vesicular emphysema. Whatever prevents any one part of the lungs from expanding when the thorax expands—whether it be a material obstruction of the bronchial ramifications or a compression of them, or whatever else—it will occasion those parts which remain dilatable to keep dilated in a correspondingly increased degree, in order to fill up the vacuum which the expansion of the chest occasions. If there be any part of the lung that will not dilate when inspirations are made, then other parts are over-dilated to fill up the vacuum. Those parts are *over*-distended, in order to compensate for the want of distension in other parts; and when once *over*-distended they are unable to recover themselves (ELLIOTSON, *Practice of Medicine*, p. 851). In this way the secretions, or the thickening of the tubes in bronchitis, may set up emphysema, by preventing air entering the corresponding vesicles, whilst those which are adjacent and having free tubes communicating with them receive an excessive amount of air (Dr. C. J. B. WILLIAMS). The explanation of the occurrence of emphysema by Professor W. T. Gairdner, has been stated at p. 709. Sir William Jenner considers it to result from violent respiratory efforts, with partial closure of the glottis, as in the act of coughing, lifting weights, and playing wind instruments. For emphysema to happen in this way there must be a want of compression and support of the lung tissue in the parts which become emphysematous. This applies especially to the apices, the anterior margins and edges of the bases. Emphysema may also be due to a primary degeneration of the septa and walls of air-vesicles. Bronchitis in old persons may also bring about degenerative pulmonary changes of an atrophic kind, so that septa waste and disappear. Hooping-cough is a common cause. Sir Thomas Watson follows Laennec in believing that the dilatation is mainly due to th imprisonment of air within the cells under the influence of imperfectly obstructed tubes, so that air enters the vesicles more readily than it can escape from them. More and more air thus accumulates, and is incarcerated in the terminal air-cells of the obstructed tubes, which, yielding to the distending force, lose their elasticity and become permanently large. To confirm this view, it ought to be shown that the dilated air-cells are those belonging to the tubes in which the obstruction exists.

(*b.*) *Morbid Anatomy.*—The dilated air-vesicles vary from the size of millet seeds to that of Barcelona nuts, or larger. When they form a great expansion, it is probable that many air-vesicles are dilated into one common cavity, by rupture or degeneration of the partitions which separate them from each other. The dilated vesicles may be seen through the pulmonary pleura; they also protrude from the surface of the lung, pale, and sometimes quite white. The tissue is drier than normal; it cannot be easily emptied of air, resembling the lungs of a reptile rather than those of a human being. It possesses fewer capillary vessels. They become obliterated by distension of the air-cells; hence the lung is anæmic, and contains less moisture than the normal lung. Such lungs do not collapse, and they may even bulge out of the chest when it is opened. The apices and anterior margins are the parts most affected.

Symptoms.—In typical cases the patient is short-winded and distressed by a constant sense of fulness and oppression at the chest, and generally seeks advice after suffering and discomfort have become too great for him to bear any longer. The difficulty of breathing is often aggravated by spasm, as in *asthma;* and emphysema is a frequent consequence of that disease. The physical signs are, incompleteness of the act of expiration, the thorax remaining prominent and round over the emphysematous lung. In spare persons the clavicles are not well defined. Percussion over the bulging parts yields a peculiarly clear and resonant sound. There is thus shown to be abundance of air underneath the part which yields such a sound; but the *vesicular murmur* of breathing is extremely indistinct, showing that the air is not in motion there. It is shut up in the enlarged air-ells (WATSON). The disease tends to impede the circulation through the ungs, and so to produce hypertrophy, with dilatation of the right side of he heart, nervous congestion of the head and face, attacks of palpitation, aroxysms of cough and dyspnœa, œdema of the feet and legs, general nasarca; and dropsical effusion is a frequent termination.

Treatment.—Apart from the management of the bronchial congestion, n the principles already given under *Bronchitis,* little can be done for the pecial treatment of *emphysema.* If bronchial spasm prevail, the *spiritus theris* of the British Pharmacopœia may give relief, of which *thirty* to *xty minims* may be prescribed in *camphor* water, or in *spiritus ammoniæ romaticus,* or in *volatile tincture of valerian;* or it may be combined with imulant doses (*one grain*) of *opium;* or with *twenty minims* of the *æthereal ncture of lobelia, belladonna, conium;* or the *æthereal tincture of Indian hemp d hydrocyanic acid* may each in turn be found of service (FULLER). *Dry-pping* between the shoulders often relieves passive pulmonary congestion; d if an attack is imminent, an emetic, or unloading the bowels by a se of the *compound jalap powder,* may prevent its accession or moderate e paroxysm.

PULMONARY PHTHISIS, OR DESTRUCTIVE LUNG DISEASE.

Latin Eq., *Phthisis;* French Eq., *Tuberculeux;* German Eq., *Phthisis;* —Syn., *Schwindsucht;* Italian Eq., *Tise Cronica Polmonale.*

Definition.—*Lesions commencing with induration of a lobe or lobule of the lungs, ending in disintegration of the new material and of the textures involved in the induration; the formation of caverns, and the accumulation and constant discharge of purulent secretion, with or without the growth of miliary tubercles.*

Pathology.—(*a.*) *Causation.*—The several forms of pulmonary lesions about to be described must be recognised as tending to lung destruction; and in the development and propagation of pulmonary consumption, or destructive lung disease in man, there are several agencies at work, especially—(1.) *An ill-defined agency, to which the name of constitutional tendency has been given;* exemplified in pulmonary phthisis so frequently attacking in succession several members of a large family shortly after adult life. An hereditary predisposition to *pulmonary phthisis* is not only certain but frequent; and is chiefly due to congenital or inherent weakness and vulnerability of the constitution. Such vulnerability is evidenced by repeated illnesses during early life. That the tuberculous or scrofulous constitution is actually transmitted from parent to child has long been a popular belief, and regarded as one of the best established points in etiology (see "Scrofula," p. 1002, Vol. I.) On the other hand, while the general statement may be made that some cases of phthisis may be traced to hereditary influence, it is undoubted that *much phthisis is, in each generation, non-hereditary* (Walshe). The final conclusion seems to be,—That *phthisis in the adult hospital population of this country is, to a slight amount only, a disease demonstrably derived from parents;* and there is no reason to believe that the law differs among the middle and higher classes of society; and we have the tuberculous or scrofulous cachexia communicated, both as regards the parents and the generation following. But there is still a class of cases to be inquired into—namely, such as show how far parents labouring under *scrofulous cachexia* entail on their offspring *a disposition* merely to tuberculous affections. It is a well-known fact, emphatically insisted upon by the late Sir James Clark, that in families of consumptive parents there are constantly to be met with instances of ill-health characteristic of the scrofulous constitution. The mere fact of the parents being unhealthy, and not necessarily tuberculous, appears in some instances sufficient to entail tuberculous diseases upon their children.

(2.) *The agency of irritation.*—A common bronchial catarrh, not differing in any respect, except in its result, from other bronchial catarrhs which terminate favourably, may gradually become an agent in destruction of the lungs. Sometimes an obvious source of irritation is to be found in the nature of the work done by those who suffer thus and die. A striking demonstration of this was afforded by an observation long ago recorded by Dr. Home. In a stonemason who died of phthisis an earthy nucleus was found at the centre of many of the pulmonary condensations. This earthy nucleus turned out to be of precisely the same character as the stone of Craigleith Quarry, near Edinburgh, where the man had been employed (HOME, *Edin. Med. and Surg. Journal*, January, 1838). Thus there are certain classes of men more predisposed to phthisis than others by the irritant nature of their occupations. Dust is undoubtedly a most deleterious agent, and when combined with great vicissitudes of temperature in the surrounding of those who breathe an *air loaded with particles of dust,* the combined agencies are extremely deleterious. To show how irritant substances may induce the local lesions, Professor Alfonso Corradi, of Venice, wrote an essay in 1870 on the spread of pulmonary phthisis in Italian towns, in which he mentions that of the stonemasons who work at granite near the *Lago di Garda*, nearly all of them died towards fifty years of age; and the same is true regarding those who work in slate in the caverns near Chiavari, and who suffer from the *Mal di Chiapperolo.* Those who do the same work in the open air are but little subject to the disorder; neither is the dust found in their lungs. In Edinburgh, in the experience of the late Professor Alison, a stonemason scarcely ever reached fifty years without becoming consumptive. Thus, irritating particles come to be a fertile cause of phthisis among those who are engaged in hackling flax, carding cotton and wool, grinding steel *(grinder's rot)*; also, workers in porcelain, makers of mother-of-pearl buttons and of mattresses, chaff-cutters, bakers, stonemasons, quarrymen, needle-grinders and saddlers, furriers, glovers (HANNOVER), who inhale dust from animal tissues,[*] and coal-miners and bricklayers. The potter's consumption is chiefly caused by portions of clay being trampled into dust on the floors of the work-rooms. Few men who enter certain rooms in cotton factories ever live to thirty-eight years; and out of twenty-seven men in a flax factory, twenty-three had pulmonary disease (GREENHOW). The noxious influence of varnishes, turpentine, and drying oils, in developing destructive lung disease, was long ago pointed out by Lombard (*Annales de Hygiéne*, t. xi., 1834), and it is said to be frequent among artisans who use solder, such as tinmen, coppersmiths, and goldsmiths (CLYMER). In miners it assumes the form of *fibroid phthisis*, in which a chronic interstitial pneumonia leads to fibroid deposits with cheesy degeneration (tyrosis) of imprisoned portions of lung, to be afterwards more fully described.

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[*] The sorting of alpaca and mohair has long been known to be attended with considerable risk to life; also, the sorting of all wools and hairs which are dry, dusty, and more or less filthy from contamination with decomposing animal matters, particularly if "fallen fleeces" are present; and all kinds of goats' hair, camels' hair, low class East Indian, Persian, and such like wools, are injurious to health,—(1.) From the dust and fine short hairs which arise from them exciting chronic disease of the lungs; (2.) from the amount and virulence of poison from decomposing animal matter that is liberated, producing a low form of pneumonia (*septic pneumonia*); (3.) from a specific blood-poison, derived from the fleeces of animals which have died from *anthrax*, producing the rapidly fatal disease now known as "*wool-sorter's disease.*" This disease has prevailed in Bradford and its neighbourhood for more than forty years, and still continues as severe and frequent as ever it was, overclouding the life of the "*sorter*" with a mysterious shadow, and threatening him daily with death (Dr. BELL, *Lancet*, June 5, 1880). In December, 1879, a case of this disease occurred in Bradford, in which an examination of the blood showed the disease to be *anthrax*, and during April and May, 1880, five more cases occurred in the same neighbourhood, four of which were rapidly fatal—*i.e.*, within three or four days. A malignant pustule formed in three of these cases. The men had been "sorting" what is known as "Van mohair."

(3.) *The agency of infection,* which means that whenever a chronic induration, due to hyperplasia, exists in any organ, it is apt to give rise to similar processes elsewhere. Buhl showed more than twelve years ago, in regard to miliary tubercle (to which he limited his statements), that in persons who die of fever characterised by the general dissemination throughout the body of miliary granulation (*acute miliary tuberculosis*), masses of induration which have remained long enough in the body to become caseous, are in the great majority of cases to be found; and that in those cases in which miliary tubercles are sown over a much more limited region—as, *e.g.*, when they are confined to a single organ—they spring from old lesions in it. The signification of this infective process must now be extended to extensive indurations, as well as to miliary deposits; since by inoculation we obtain miliary granulation in the serous membranes and in the choroid, but interstitial and diffused lesions in the liver, lungs, and other massive organs.

(4.) *The agency of extreme bodily exertion* (which so disturbs the circulation), and *the influence of hæmoptysis,* itself an evidence of an abnormal and disturbed condition of the bronchial circulation. Blood remaining in the bronchi has been regarded since the time of Hippocrates as a frequent cause of *pulmonary phthisis.* It is followed by an irritation of the lung and pleura—*consecutive pleuro-pneumonia*—in which, on the second or third day after hæmorrhage, an increased frequency of the pulse occurs, with heightened temperature and general *malaise,* lancinating pains in the lateral regions of the chest, fine bubbling rhonchi, friction sounds, or slight percussive dulness, with weakened vesicular murmur, or bronchial breathing. Such lesions may end in resolution, or gradually go on to caseation (*tyrosis*), and perhaps the growth of tubercle, as the direct result of infective absorption of the decomposed blood (*Phthisis ab hæmoptöe*).

(5.) *Influence of Climate.*—Where the civilised inhabitants of the globe are located in the temperate zone, it is calculated that *one-tenth* of the population die of destructive lung disease. In the mining districts of Cornwall and Devonshire (although those counties are considered among the most healthy portions of Great Britain), one-half of the whole number of the miners, deprived of fresh air and light, die of phthisis. But destructive lung disease, or phthisis, is not a product merely of climate. It is more a social disease; the climate under which it thrives being one made for it under the influences of modern life and work. The more people become crowded together, the more industries are developed, the more does destructive lung disease show itself. Crowding together of families in small rooms, the inhalation of foul air, or of deleterious particles of dust in manufactories, and a too sedentary life, are the frequent causes of pulmonary consumption by destructive lung disease. The great centres of industry are the places which yield the highest mortality from phthisis—*e.g.*, London, Manchester, Liverpool, New York, Philadelphia, New Orleans, Berlin, Munich, Vienna. England has been called the home of industry and of phthisis. It seems now to be generally admitted that the number of cases of destructive lung disease diminishes as the elevation increases, a fact which may be accounted for under the following considerations—(1.) Many of the factors of destructive lung disease are absent at certain elevations—all those especially that are concerned with social life aggregated and deleterious trades. (2.) The alternations of temperature are less marked in the mountains than in the plains (HIRSCH). (3.) The air is more tonic and favourable to nutrition (BREHMER). (4.) On account of the deficient supply of oxygen, for as the higher layers of air have less density, the quantity of oxygen in them for each inspiration is less—at 15,000 feet about one-half of what it is in the plains. Hence, in order to get the necessary supply of oxygen, the inspirations become deeper and more complete, and the thoracic cavity increases in capacity. (5.) The tonic action of pure air shows its beneficial influence in promoting appetite and digestion. (6.) A certain excess of carbon in the system, consequent on the diminished supply of oxygen (LIMBARD). (7.) The comparative absence of organic particles—of dust—in pure mountain air. The transparency of the air, which is dependent on its greater dryness; and, the more powerful action of light, dependent on the same cause, aid materially the operation of the purity of the air (GOURAUD, MACPHERSON, *London Med. Record*, July 15, 1876, p. 294). (8.) It has also been determined that a greater quantity of carbonic acid is formed in the body and exhaled at the higher than at the lower altitudes, and the moisture exhaled increases as a person rises above the sea (Dr. W. MARCET, F.R.S., *Proceed. Roy. Soc.*, No. 195, p. 498).

The disease appears to be more fatal to soldiers than civilians, producing nearly *one-half* of the whole mortality among the Dragoon Guards in this country. In the Foot Guards it has hitherto been nearly double of what takes place in the Dragoon Guards. The habits of a soldier's life, his liability to febrile and inflammatory attacks, the frequency with which he contracts venereal diseases, and the climate in which he lives, have hitherto been favourable to the induction of the more aggravated forms of pulmonary indurations and destructive lung disease. The history of phthisis in armies shows how materially its prevalence influences the health, the wealth, and the military strength of a nation. In Prussia, phthisis caused 27 per cent. of the total mortality; in Austria, 25 per cent.; in France, 22·9; in Hanover, 39·4; in Belgium, 30; in Portugal, 22 per cent. In all these armies the same causes are in action, and the predominance of the disease is mainly to be sought for in the impure barrack air (PARKES'S *Practical Hygiene*, p. 492). Some are inclined to ascribe this excessive amount of pulmonary disease and mortality to the night duties of the soldier—a statement supported by the large amount of mortality from consumption amongst night-watchmen generally. But when one looks to the age and height of the men enlisted for the regiments of the Guards, and compares them with the physiological records regarding the stature and growth of the human frame, it will be seen how, sometimes, the combination of requirements for enlistment in the regiments of Guards have been little calculated to secure a hardy and efficient body of men.* They are constitutionally vulnerable to an extreme degree. There can be no doubt that there still is an excessive prevalence of destructive lung disease in the army; and the astonishing disproportionate number of cases in the Foot Guards (18 to 20·6 per 1,000 of strength) is still as remarkable as it was twenty years ago (Parkes, l. c., p. 495). The tables prepared by the late Dr. Parkes, and given in his work on *Practical Hygiene,* show that there must be a large amount of phthisis *generated* in the army; and in the Foot Guards nearly *four* times as much as among the civil male population of twenty-five to forty-five years of age. In the American army (1861 to 1863) the ratio varied from 8 to 9 per 1,000, and the deaths from one in every 2·7 to one in 4·5 cases. In the British army, during 1861-62, the cases of phthisis were from 9 to 10 per 1,000 of mean strength, and the deaths one in three. But neither in the American nor in the British army is it shown how many soldiers are *annually invalided* on account of pulmonary consumption, the greater number of whom die after their discharge, and swell the mortuary return of both countries (Clymer). The deaths from phthisis in the British navy during three years averaged 2·6 per 1,000 of strength, and the invaliding 3·9 per 1,000 (Milroy).

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* On this subject the reader is referred to a little book, by the Author, *On the Growth of the Recruit and Young Soldier.* Charles Griffin & Co., London.

It has long been a prevalent belief that consumption was limited by latitude, and that it never appeared in warm countries—for instance, south of the Mediterranean. This is proved not to be the case. The returns of the army, prepared by the late Sir Alexander Tulloch have shown that phthisis is more frequent in the West Indies than in this country—a statement first made by Sir James Clark, in his work *On Climate*, in illustration of the injurious effects of that climate on consumptive patients sent there from this country. Great heat appears to have a powerful effect in predisposing to tuberculous diseases (probably by diminishing the exercise in the open air). But it is more than probable that crowded barrack-rooms, a restriction to salt diet, and drinking spirits, may have produced the result, rather than climate alone, seeing that officers were attacked in infinitely smaller proportions than private soldiers. The influence of climate shows that phthisis is most frequent in low and damp situations; while it is far less so in the mountainous districts of all countries.

(6.) *Influence of Contagion.*—Regarding the possible propagation of pulmonary phthisis by contagion, the late Dr. Parkes thus expressed himself (having regard to the fact that purulent and epithelial cells have been demonstrated as floating about in the air where numbers of persons are together):—"Considering that the pleuro-pneumonia of cattle is probably propagated through the pus and epithelium cells of the sputa passing into the air-cells of other cattle; that even in man there is some evidence of a pneumonic phthisical disease being contagious (Bryson, *Cases in Mediterranean Fleet*), the floating of these cells in the air is worthy of all attention. It may explain some of those curious instances of phthisis being apparently communicated" (*Prac. Hygiene*, p. 74). So also Professor Virchow at the International Congress for Hygiene at Brussels in 1877, directed attention to this question—namely, "in how far one of those diseases which we have hitherto regarded as a spontaneous disease of the ruminant animals—bovine tuberculosis (*Perlsucht*)—is a communicable disease, and communicable to man." The disease can be communicated exactly in the same way as in the inoculation of tuberculosis. A further question is, "how far, by the partaking of substances coming from a tuberculous (*Perlsüchtig*) animal, similar disease, and in fact tuberculosis may be induced in man, from partaking of their flesh, or through the use of their milk?" It is in the juices of the tainted animal that we must suppose the contagion to reside. Dr. Charles Creighton, Demonstrator of Anatomy in the University of Cambridge, has described an infective variety of tuberculosis in man identical with bovine tuberculosis (*Perlsucht*), (*Lancet*, June 19, 1880, p. 943). His cases show the distinctive and specific characters of that disease in their pathological anatomy, and how they are related to it in their causation. They have precisely that relation to bovine tuberculosis which glanders in the human subject has to equine glanders; and bovine tuberculosis (*Perlsucht, pommelière*) is a disease *per se* as much as glanders is. It has well-marked distinctive characters, which appeared to Dr. Creighton to be reproduced in the cases related by him. In 1869, Dr. Julius Petersen, District Physician in Copenhagen, showed that a belief in the contagiousness of phthisis has a very ancient historical foundation. "Morgagni said *phthisicorum cadavera fugi adolescens, fugio etiam senex.* In Italy consumption has been, and still is, looked upon as a communicable disorder. A consumptive is shunned, and the vessels he may use in eating and drinking are avoided or destroyed. His clothes are burnt or buried. The opinion that long and continuous exposure to the body-effluvia of a tuberculotic patient puts a previously non-tuberculous person to the risk of the disease, by a predisposition to it, has been held by Jos. Franks, Laennec, Sir James Clark, and others. Andral went so far as to say that under certain conditions these tubercular emanations become a source of true contagion. Most medical practitioners of long experience have seen examples of the apparent contagion of consumption—a tuberculous husband infecting a wife, and the reverse. Some facts of this kind are related in the *Gazette Hébdomadaire,* 1868, and a case is related in the *Lancet,* June 22, 1880. Breeders of stall-fed cattle are now very careful as regards the prevention of bovine tuberculosis. They will not suffer a stall-feeding animal to resort to the same water-trough or to the same stall which an infected animal has used. Although unable to produce any absolute proof of the existence of contagion, Dr. Petersen sums up the result of his inquiries as follows:—(1.) That a contagious origin of some cases of phthisis cannot on sufficient grounds be denied. (2.) That phthisis caused by contagion is in general of a very dangerous and inflammatory character; that it must justly be considered hazardous to sleep in the non-disinfected bed of a phthisical patient, and to be habitually in too close contact with such a person; that this danger in Denmark seems to be greatest in the warm period of the year. Besides direct transmission, Dr. Villemin suggests that consumption may be contracted through indirect means, by clothes, bed-linen, water-closets, the vitiated air of rooms lived in by tuberculous persons, and the like. The possible transmissibility of the disease in this manner merits, he thinks, the attention of medical officers of the army. He is satisfied that the barrack is to the soldier in the production of consumption what the regimental stable is to the horse in the development of farcy, the contagion and transmissibility of which are at length accepted. Fournet, who in his work is a non-contagionist, still gives some weight to the possibility of infection from an atmosphere constantly breathed, and necessarily poisoned, by the consumptive. "In protracted debates upon phthisis in the French Academy of Medicine, the subject was fully discussed; the views of two of those to whose opinion we are disposed to attach value will be quoted. Dr. Jules Guérin believed that crude tubercle can never be contagious; but that when it is softened, and the ulcerated lung-surfaces are exposed to the air, the patient may become a source of infection to those about him, just as the pulmonary lesions he has may infect his own organism by the resorption of purulent and putrid products. Dr. Bouillaud's ideas would seem to be nearly the same. He writes,—'During the course of pulmonary tuberculosis, when pus or other septic products are formed in parts which are accessible to the atmosphere, phthisis, like so many other affections in which similar purulent foci happen, becomes indirectly a cause of septic infection.'"

(*b.*) *Morbid Anatomy.*—The rapid advance of more accurate knowledge has not been more marked in any direction than in demonstrating the nature of the lesions which lead to destruction of the lung, the formation of miliary tubercles in them, and the relation of such tubercles to the processes of disintegration, softening, and absorption of pneumonic products, and other products in various parts of the body. While the observations and writings of eminent pathologists and physicians on the Continent have done much of late to rectify erroneous views regarding the subject of destructive lung disease, it must be acknowledged that the late Dr. Addison, of Guy's Hospital, did more than any one else in England to advance the doctrine of a pneumonic phthisis. For a long time his labours had remained entirely unknown on the Continent; and having been mainly brought to light by the New Sydenham Society, they show that already, when Laennec's teaching had just commenced to dominate over the pathology of lung diseases, Addison had arrived at and firmly held the opinion that "*inflammation constitutes the great agent of destruction in every form of phthisis.*" On the other hand, the formation of *miliary tubercles* in the lungs and other parts, and their relation to the softening of cheese-like products and to phthisis, have been placed in a new light by the practical experiments and clear expositions of Drs. Villemin, Burdon-Sanderson, Wilson-Fox, Andrew Clark, Colin, Chauveau, Cohnheim, Buhl, Waldenburg, and Gasquet (*Scrofula*, p. 1014, Vol. I.) "Phthisis," as implying a wasting of the body with a condition of lung ready to disintegrate and to have cavities formed in its substance, must no longer be regarded as having always a basis of tubercle-formation as its starting point; and the doctrine that pulmonary phthisis is always a constitutional disease—in the sense of Laennec—the result of a specific morbid product, arising from some peculiar diathesis or constitutional fault, and caused solely by the deposition of tubercle, can no longe be entertained. A comprehensive view must be taken of the agencie concerned in destructive lung disease, or pulmonary phthisis, such as tha which Dr. Andrew Clark has embodied in the following provisiona classification of the pulmonary lesions, namely:—

(1.) *Tubercular, granular, or specific phthisis*, the chief anatomica characters of which are, the true grey granulations, pigmentary tubercl fibrous tubercle, cellular tubercle.

(2.) *Scrofulous or epithelial phthisis*, composed of primitive yellow masse of (so-called) tubercle; accumulation, cheesy degeneration, and disintegration of epithelium-like cells.

(3.) *Catarrhal or bronchial phthisis,* in which there is ulceration of bronchi, with adjacent fibroid and cellular deposits, and cheesy degeneration of the same.

(4.) *Pneumonic phthisis,* the chief anatomical characters of which are, disintegration of recent or old deposits, occurring in vesicular, lobular, or lobar pneumonia, primary or secondary, common or scrofulous.

(5.) *Fibrous phthisis, cirrhosis, chronic or interstitial pneumonia,* in which there are fibroid growths with cheesy degeneration of imprisoned portions of lung, due to—(*a.*) Mechanical irritation (as in grinders, masons, miners, &c.); (*b.*) rheumatic inflammation of interlobular tissue; (*c.*) chronic pleurisy; (*d.*) constitutional states, as in granular kidney and liver.

(6.) *Lardaceous phthisis,* characterised by circumscribed or diffuse cellular formations composed of lardaceous material.

(7.) *Syphilitic phthisis,* in which there are cheesy disintegration and diffuse infiltrations of nodules of nucleo-fibrous tissue.

(8.) *Hæmorrhagic phthisis.* Cheesy degeneration and disintegration of nodules of extravasated blood.

(9.) *Embolic phthisis, including pyæmic deposits and suppurations,* in which cheesy degeneration and disintegration of grey or yellow deposits arise, directly or indirectly, from pulmonary emboli coming from the liver, lympathics, or veins.

Softening and decomposition of any of these may, through an infective process, disseminate the growth of *true miliary* tubercle.

The nomenclature of the College of Physicians regards this subject from two points of view, namely:—(1.) As allied to scrofula—a general constitutional disease, with or without tubercle (p. 1000, Vol. I.); and, (2.) As destructive disintegration of the pulmonary tissue, having its source in *acute* or in *chronic* pneumonia.

It will already appear, from the definition, that miliary tubercle, as a cause of phthisis, plays a very insignificant part in the process of lung destruction, while many lesions tending to induration of the lungs, in the course of their subsequent disintegration, give rise to phenomena which have been confounded together under the common name of "tubercular phthisis." The records of medicine have shown that in a large number of cases in which death results from destructive lung disease, *true miliary tubercles* are not present in the lungs (Addison, Niemeyer). In the aged it has also been shown by the late Dr. Maclachlan (*Diseases of Advanced Life*, p. 333), that "consumption may exist independent of tubercular development, that tubercles are not the essential anatomical character of senile phthisis; and that the most extensive destruction of the lungs not infrequently occurs accompanied with the usual symptoms without a trace of tubercle." Destructive lung disease is usually a sequence of chronic bronchitis, terminating in indolent inflammation and partial nduration of the lung. These indurated parts at last break down, leaving caverns and burrowing sinuses, which are characterised by a dark nd sloughy aspect of their inner surface, and by the absence of any membranous lining (Armstrong, Graves, Stokes, Maclachlan). Such a condition constitutes the *ulcerous phthisis* of Bayle, the *pneumonic* *phthisis* of Addison." In *senile pulmonary consumption*, when tubercles are found, the tubercular growths are generally confined to the lungs —commonly limited to the upper and back portion of one lung only —tubercles existing in the very apex or apices, the remainder of the lung being healthy (MACLACHLAN, *op. cit.*, p. 334). There are many formations and lesions constantly mistaken for tubercle; and no appearance is more often so regarded than the cut sections of bronchial tubes, thickened, softened, dilated, and containing a muco-purulent fluid in their lumen, as Addison long ago figured and described. Every condensation of lung-tissue, every cheesy metamorphosis of a previous growth, be it pus or more solid exudation, have severally been regarded as tubercle; and the fact of their having become yellow and cheese-like (tyromatous) was regarded as evidence of their having been originally tubercle, more especially if the yellow cheese-like masses happened to co-exist with true miliary tubercle. They were then regarded as a diffuse growth of tubercle, or as a tubercular infiltration, like the infiltration of a cancer. But it is now a well-established fact, that many consolidations, such as inspissated pus, old cancer masses, lymphatic glands enlarged by hyperplasia of their cell-elements, hæmorrhagic deposit, as well as the clear miliary tubercle, may each and all become yellow and cheese-like, quite independent of the *scrofulous diathesis.* To this condition Dr. Craigie, of Edinburgh, proposed the names "*tyrosis*," "*tyroma*," "*tyromatous*" (from τυρος, cheese), to signify the caseous aspect of such masses or accumulations, and without reference to the origin or causes of the lesion; but these names have never come into general use (*Pathological Anatomy*, p. 1008). Some o these caseous transformations constitute the "tuberculisation" of Virchow. They are to be looked upon as forms of degeneration, to which the nam *tyrosis* would be much more applicable, or at least less objectionable. Al the morbid products in the lungs which lead to destructive lung diseas whether of pneumonic origin or true miliary tubercle, tend to underg this caseous transformation (*tyrosis*).

There is no special or peculiar form of pneumonia which leads t lung destruction; but the lesion is invariably a more or less prolonge lobular induration of the lung which undergoes the process of *tyrosi* disintegration and breaking down into cavities (ADDISON, NIEMEYE BURDON-SANDERSON). Every form of pneumonia (as Addison has show may leave a residue which, under certain circumstances, terminates *tyrosis;* but some cases of pneumonia are more liable than others leave a *residuum*, and instead of the material of consolidation bei liquefied and reabsorbed, it becomes more dense, and finally is transform into a caseous substance (*tyrosis*), which finally softens and breaks into a cavity. Such a termination is rare in cases of common acu (croupous) pneumonia in a healthy person. It is more frequently m with in catarrhal, patchy, or lobular pneumonia; and in chronic catarr pneumonia to find it is almost the rule (NIEMEYER). On this po Addison has written that when the matter thrown out into the lu is of the more plastic or organisable kind, it fails to be entirely absorb part of it remains forming deposits, indiscriminately called "tubercl or "tubercular infiltration." Such forms of induration are altoget independent of true miliary tubercle, and may result from a sim pneumonia, or broncho-pneumonia, incident to all ages, in which there is little cough or expectoration, and therefore it is often overlooked. Dr. Addison recognised two forms—namely, *pneumonic phthisis* and *tuberculo-pneumonic phthisis;* they are now recognised as *acute pneumonic phthisis,* and *chronic pneumonic phthisis,* to which now may be added *bovine tuberculosis.*

A. Acute Pneumonic Phthisis.—How far a recent acute pneumonic lesion must proceed to be beyond the limits of complete restoration to health is not determined. The condition of the patient as to soundness of constitution influences the result. Hale and otherwise healthy patients recover from an extensive pneumonic lesion, which persons of a scrofulous or cachectic constitution would not recover from. How far lungs in a stage of grey hepatisation can be recovered from is not known. When there is actual induration and obliteration of the lobules, perfect repair and restoration cannot be expected. All that can be hoped for is that the residuum may remain quiescent, without retrogression towards cheesy degeneration (*tyrosis*). Cases are constantly seen, *post-mortem,* in which gradual changes are obviously passing from red to grey hepatisation, with well-marked granulation on the surface of a section, or to cheesy degeneration (*tyrosis*). Dr. Addison distinguished two varieties of acute pneumonic lesion tending to pulmonary destruction,—(1.) Inflammation, more or less acute, but slow and insidious in its course, manifesting some attempts at repair, as indicated by various stages and degrees of induration. The pulmonary tissue continues friable, and within a few weeks or months softens down and gives rise to cavities, generally by a slow ulcerative process, rarely by actual slough or death of the indurated but friable tissue. (2.) Inflammation supervening upon, or around ancient induration, leading to disorganisation of the newly inflamed tissue, of the old induration, or of both at the same time. The material accumulated in an *acute catarrhal pneumonia,* when it passes on to the cheesy *degeneration* (*tyrosis*), commences by catarrh of the smallest bronchi—extending to the alveoli, which become filled with young indifferent round cells, so that a whole lung, or parts of it, may become disseminated with these minute translucent nodules of lobular or catarrhal pneumonia. In phthisical cases, the mass *caseates* at the centre (Sanderson)—*i. e.,* becomes opaque and soft, and fatty metamorphosis remains incomplete. The cells lose their round form, and by losing water, shrink into irregularly shaped corpuscles. To the naked eye such consolidation of lung appears of a dull grey, or reddish-grey homogeneous appearance, gradually passing into a cheesy-like substance (*tyrosis*). From this point, disintegration may advance till a vomica is formed. Such terminations of lobular pneumonia are common in the course of measles and hooping-cough. It is a result frequently met with in weakly and delicate individuals possessing but feeble powers of resistance against injurious influences—a delicate vulnerable constitution. There is a tendency in such persons to the cellular products of inflammation.

B. Chronic Pneumonic Phthisis; Fibroid Phthisis; Cirrhosis of Lung.—Of this form, two varieties are described, namely—(1.) That in which the old indurations undergo a slow process of disintegration. (2.) That form in which an insidious inflammation proceeds very slowly to convert a considerable portion of pulmonary tissue into grey induration or *cirrhosis* (ADDISON). Generally, the lesion may be considered as the result of *chronic catarrhal pneumonia,* where the induration is of a gelatinous material, to which the names of "*tuberculous infiltration,*" "*tubercular,*" or "*cheesy pneumonia,*" have been given (NIEMEYER). Such an *infiltration of the tissues* by an albuminous fluid of a thick synovial-like character gradually degenerates into a firm greyish-red granular softened mass, inclosing living tissue. It was first described by Baillie in his *Morbid Anatomy.* "In cutting into the lungs, a considerable portion of their structure sometimes appears to be changed into a whitish soft matter, somewhat intermediate between a solid and a fluid, like a scrofulous gland just beginning to suppurate." Chronic pneumonic phthisis not only involves lobules, but whole lobes. The infiltration is not unlike frogs' spawn in colour and appearance. It is homogeneous and smooth on section. It occurs in previously healthy lungs, as well as in those which already contain tubercles, induration, old cheesy deposits, or cavities; and it may be one step towards pulmonary phthisis. Occurring in lungs already diseased, it contributes essentially to the further extension of their destruction. The cells which accumulate in this variety of pneumonia tend, as a rule, to undergo the caseous change; but cavities are not invariably produced. More often the contents are partially absorbed; and *cretaceous masses* are left behind.

One mode of cavity-formation in chronic phthisis occurs where the absorbed material is replaced by a growth of connective tissue. The lung-tissue does not again become permeable to air, but is transformed into a tough indurated substance. The new connective tissue, by gradually shrinking, occupies less space, the thorax contracts, and the bronchi dilate into oblong round cavities (*bronchiectasis*). A common form of pneumonic lesion, which terminates in this way, is the fibroid o sclerosed lung, a form of chronic or interstitial pneumonia (see p. 722 *ante*). It may present a smooth or a granulated surface. The granulations resemble minute hard semi-transparent tubercles, each set in th midst of a hyperplasia of connective tissue. This tissue is very distinc between the lobules. It divides and subdivides by innumerable ramifications, closing in minute polygonal spaces containing air-cells, an interspersed with much pigment. The cut section presents a bluish-gre colour, different shades of iron-grey, a granite-like appearance, or nearl black. Microscopically the indurated part is made up of cell-elements i various stages of elongation on to highly organised fibrous tissue. Th granules inclosed consist of imperfect cell-elements—the possible remai of catarrhal inflammation. In some cases the whole lung, or the great part, is converted into a tough fibrous tissue, hard and contracted, whic looks striated, as if interwoven with fibrous filaments. Cavities are ap to form in the indurated texture, generally at the apex, irregular in shap and containing an offensive dark-red fluid. Their walls are formed the condensed lung-tissue. Smaller cavities are generally scatter through the indurated tissue formed from the dilated bronchial tub having a thin membranous lining continuous with that of the bronc *Tyrosis,* or caseation of portions, may be interspersed among the conso dation. There is evidence of long existing disease of the bronchi, whi are enlarged and thickened. The pleura is thickened over the indurated parts, sometimes to the extent of one-fourth or one-half of an inch; and fibrous bands pass from the pleura into the texture of the lung. The bronchial glands are enlarged and hard. There is generally granular degeneration or cirrhosis of other organs, such as the kidney, liver, heart, pylorus, capsule of the spleen, bronchial glands, and skin (SUTTON, CLYMER).

"Microscopical examination shows—(1.) great increase by a production of new tissue-elements, connective or fibroid tissue; (2.) that this new formation invades and destroys, in part or wholly, the lung-tissue, which undergoes fibroid change; (3.) that these fibroid elements are found most highly developed in the connective tissue surrounding the minute bronchial tubes and lobules, as well as in the neighbourhood of the thickened pleura; (4.) that the new formation begins in the connective tissue of the lung, and extends in every direction amidst the elastic fibres of the air-sacs, until these degenerate and are obliterated; (5.) the yellow or cheesy matter found amidst the grey fibrous induration, consists of granules, having bright sparkling centres, and of irregularly shaped, shrivelled or wasted cells, filled with fat-granules. In fibroid growths elsewhere, as in the uterus, circumscribed fibroid deposits in the kidneys, syphilitic tumors in the lungs and liver, and in cancer of the lung, similar yellow cheesy centres occur (CLYMER). As to the nature of this fibroid change, or *cirrhosis of the lung*, it has been described by many British and French authorities as a chronic pneumonia. In chronic pulmonary inflammation, the lung is not shrivelled or contracted as is the lung in cirrhosis, and although rather hard and elastic, it is comparatively readily broken down, which is not the case in the fibroid lung. The lung-tissue in cirrhosis is firmer and drier than in chronic hepatisation, scarcely yielding a trace of moisture when scraped, and often creaking under the knife. Dr. C. J. B. Williams, who published two cases of the disease thirty-four years ago, looks upon the fibroid lung as a modification of ordinary phthisis, its distinctive character being its tendency to hronicity, to which it owes its comparative curability (*Lancet*, Vol. I., 868). By Dr. Andrew Clark the lesion is regarded as a local expression f a constitutional affection, such as rheumatism, syphilis, chronic alcoholsm. Its tendency is to hyperplasia; and it is a frequent form of senile hthisis (STOKES, MACLACHLAN, CLYMER). Irritation, prolonged from ome cause, is generally a constantly acting cause, and a previous history f chronic catarrh, combined with excessive spirit-drinking, are usual ntecedents. It is a frequent form of lesion in the consumption of stoneasons, grinders, and the like.

C. TUBERCULO-PNEUMONIC PHTHISIS AND TUBERCULAR PHTHISIS.—uberculo-pneumonic phthisis is generally a rapidly fatal and common rm of destructive lung disease, in which, although true miliary tubercles e present, the really efficient cause of the mischief is the pulmonic flammation and its subsequent results in softening and cavern formaon. "*The greatest danger to most phthisical patients is the development of ercles*" (NIEMEYER); and this statement leads to a consideration of the ation that may be traced between *tubercles* and *phthisis.*

Generally, it may be stated, that the more frequently the products of any inflammation (whatever its form or kind) undergo softening and decomposition, the greater will be the chance of the deposit of miliary tubercle; and when tubercles are formed in a lung previously healthy and free from cheesy deposits and cavities, cheesy products (*tyrosis*) in stages of disintegration, softening, or decomposition, are to be sought for, and may be found in other organs. They have been found in the urinary and genital organs, in the lymphatic glands, and among the products of suppurative peritonitis. From this point of view, Niemeyer regards *tuberculosis* "*as in most cases a secondary disease, originating, in some unknown way to us, in the action of cheesy morbid products on the organism*"—perhaps, also, directly producible by the absorption of decomposed blood (GASQUET); or, by the absorption of any sufficiently softened down or decomposed matter as to admit of absorption through veins or lymphatics. In what way does tubercle thus originate? has been answered, to some extent, by the experiments of inoculation, especially and directly made by Drs. Villemin, Burdon-Sanderson, Wilson Fox, Cohnheim, Chauveau, and Waldenburg, on rabbits, guinea-pigs, hedgehogs, goats, horses, and cattle. The result is the production of miliary tubercles in different parts of the animal's body (Vol. I., p. 1015).

The next pathological question of importance concerns the nature of the tubercle when so developed by inoculation. There is the evidence of Virchow to prove that the miliary formations which so arise are identical with tubercle. There is the evidence of Sanderson, Fox, and other experimenters, which shows that the result of inoculation is to produce a definite disease, affecting almost all the internal organs by producing in them nodules of new growth having a peculiar structure. This structure is common to all those diseased products which Virchow calls *lymphomas*, because they resemble certain organs of the lymphatic system. Sanderson prefers the word "*adenoid*" to characterise this tissue. He regards the tubercles which are produced artificially as overgrowths of adenoid tissue, and not new growths; and the parts most apt to be infected by tubercle are those in which the adenoid structure exists naturally in marked abundance.

In the lung the inoculated tubercle is disseminated with minute nodules of lobular catarrhal pneumonia. Each nodule is extremely translucent, and consists of two materials entirely different from each other anatomically. The alveoli are choked with the ordinary roundish epithelial cells always found there, and the alveolar walls are thickened by the growth in them of the adenoid tissue. These masses of lobular pneumonia coalesce. Each *caseates* at the centre—*i.e.*, becomes opaque and soft; and disintegration goes on till a vomica is formed. The disease progresses not by continuous growth, but by the distribution or dispersion of infective material from one point—the focus of infection (SANDERSON). The communication of the disease from a part primarily affected to the rest of the body takes place by such distribution or dispersion of infective matter so that we have to do with primary and secondary lesions in such a form of destructive lung disease. It is the nodule of induration produced by inoculation which is the primary lesion; and from this the infective agent proceeds as matter in a state of extremely fine division (WALDENBURG, BURDON-SANDERSON).

Thus, it appears that recent researches tend to bring tuberculosis into the category of infective diseases; and lead us to believe that the infective material from which it proceeds is infinitely more common, and the conditions for its production of much more frequent occurrence, than those which generate other morbid poisons. *Tuberculosis* thus implies a certain degree of malignancy; and when an organ or part is *tuberculous*, it means that it is in a state of destructive induration, and softened—the remnants of a process like pneumonia. Such remains are found either in enlarged lymphatic glands from hyperplasia, which afterwards undergo cheesy degeneration (*tyrosis*); or they are the cheesy remnants of pleuritic, pericardial, or peritoneal effusions; or the products of chronic inflammations of bones and joints in states of softening and decomposition.

True miliary tubercles in the lungs are thus developed in a slow and insidious manner, and their structure has been minutely described at p. 1015, Vol. I. The course of such cases is generally acute; and when subjects in a state of scrofulous cachexia are exposed to violent irritation of the lungs, these granulations grow so rapidly and in such numbers throughout the lungs as to give rise to most alarming dyspnœa (Sir JAMES CLARK). There are few cases in which the destructive progress of pulmonary phthisis is uniform. It is continually being checked, and for a time slumbers. Even in the worst cases of destructive lung disease numerous cicatrices and evidences of attempts to heal may be recognised; but as one portion cicatrises another becomes the seat of further induration, softening, and cavities. Cicatrices of healed lesions present different appearances, according as the cavities have been superficial or deep-seated. When superficial, the pleuræ are more or less adherent and thickened, frequently thus forming an external boundary to the cavity. When the walls of the cavern contract, the pleural surface of the lung is drawn inwards; and thus the irregular puckerings on its surface are produced. Occasionally no traces of scrofulous matter or tubercle are discovered either within or in the vicinity of these cicatrices; but more generally the contraction and puckering of parenchyma occur round indurations which have undergone various transformations, and sometimes a cyst incloses the mass. The inclosed growth may be found to have undergone any of the processes of transformation already noticed; and the cretaceous or calcareous concretions may remain an indefinite time in the parenchymatous substance of the lungs, or they may be evacuated through the bronchi with the sputa. Thus, masses of new material are sometimes absorbed or thrown off, and the evidence of this may be summed up under the following points:—(1.) Small puckered cicatrices, with loss of substance, after subsidence of the systemic disturbance characteristic of destructive lung disease. Strumous growths in the lungs of children seem capable of absorption without undergoing any change, such as cretification or softening. Most frequently it seems in them to affect mainly the small lymphatic glands along the outer walls of the air tubes. (2.) The conversion of the indurated lesions into cretaceous or horny masses, which are either expectorated or remain latent. (3.) Isolation of the growth by so-called plastic material; adhesion and thickening of the pleura; adhesions and lymph thickenings round cavities, analogous to cysts round clots of blood or musket balls.

D. Infective Bovine Tuberculosis in Man is not identical with the ordinary autochthonous tuberculosis of man; but a disease *per se*, as much as human glanders is contracted from that disease in the horse (Vol. I., p. 779). It has well-marked distinctive characters, the salient points of which are:—(1.) *Embolic infarcts in the lungs,* due to blocking up of terminal branches of the pulmonary artery, composed of a definite new growth from emboli, charged with specific (infective) properties. (2.) *A condition of the lymphatic glands,* in which their diffluent or medullary state resembles a soft lympho-sarcoma. This condition obtains in the bronchial and portal glands, unlike scrofulosis, and unlike lympho-sarcoma. Some individual glands are of great size, joined by connective tissue with large hollows between, forming potato-like clusters of nodules, whether growing from serous membranes or lymphatic glands. Each enlarged gland contains several distinct round masses; and the implication of the abdominal and thoracic lymphatic glands is a distinctive character of bovine tuberculosis. (3.) *The character of the new formations in the glands* shows a chronic formation in their brown coloration. (4.) *An eruption on the serous membranes,* especially the peritoneum and pleura. It consists of large, flat, confluent nodules on the diaphragm, pedunculated on the pleura (no doubt due to the movement of respiration during their growth, hence varying in form in man and animals). (5.) *Microscopic characters* show *giant cells* in enormous numbers and in most exquisite forms. It was in the bovine variety of tuberculosis that these giant cells were first described by Virchow. They seem to be the agents for extending the vascular supply in the formation of blood-vessels. The structure may caseate; but it is more vascular, more stable, and more like vigorous connective tissue than is the indigenous product in man (Crichton, *Lancet*, Jan. 19, p. 981).

The General Symptoms on which the diagnosis of destructive lung disease may be made are the following:—(1.) *Increased frequency of respiration,* not always attended with *dyspnœa,* without dulness on percussion, and without bronchial breathing. *Dyspnœa* may be felt only at times, as when the respiratory wants are increased by fever, and therefore by increased tissue change. In some cases the increased frequency of respiration (48 to 54 per minute), the pulse being 104 to 112, with dyspnœa and pyrexia, indicated by a temperature of 100° to 102° Fahr., may be the most marked symptoms. There is rarely pain on respiration. Dyspnœa is only present when these phenomena are present together. Miliary tubercles *per se* escape detection by physical examination. (2.) *Pains in the chest and shoulders* may be absent, or occur as dull, aching, "flying" pains about the collar-bones, or under one or both shoulder-blades. (3.) *Cough and expectoration precede lung destruction* by a longer or shorter time (two or three weeks), indicating catarrh, which spreads to the alveoli, as catarrhal pneumonia, the tyrosis and disintegration of which commences the destructive process, as after measles, hooping-cough, and the exanthemata. (4.) *Characters of the sputa expectorated* are finely and sharply marked deep yellow streaks of mucus, which indicate catarrh of the smaller bronchi, the inflammatory product being rich in cells—a form likely to pass into the air-cells. Intimate admixture with blood indicates the beginning of a pneumonic process. (5.) *A tedious and troublesome, chronic, hoarse or aphonic cough,* with expectoration, *and with constant pyrexia,* are suspicious symptoms of a pneumonic process with the growth of tubercles. (6.) *Increase of temperature* indicates extension of catarrh from bronchioli to air-cells; and its persistent increase testifies to the continuance of the pneumonic process. The difference of morning and evening temperature amounts, as a rule, to 1·8° to 3·6° Fahr., rarely less—very often more. The characteristic range of temperature in the pyrexia of phthisis is a low morning and high evening variation. Dr. Coghill, Physician to the Consumption Hospital at Ventnor, has found the usual extremes to be from 97° to 103° Fahr., and when these extremes are exceeded, the tendency is towards a lower morning range rather than a higher evening one. In such cases the patients complain of rigors and chilliness. In the morning temperature may be normal, while in the afternoon or evening it may be 102° Fahr., or higher; but when tubercle complicates pneumonic pulmonary phthisis, the differences between morning and evening temperature are much less. (7.) *Impoverishment of blood and emaciation* are constant, and in relative proportion to the height and persistence of the fever. (8.) *The physical signs* indicate either (*a.*) *tuberculosis;* or (*b.*) *inflammatory processes* which have led to infiltration, cheesy degeneration (tyrosis), disintegration, with shrinking or destruction of lung-tissue; or, (*c.*) both these conditions combined. (9.) Physical examination of the apices yields negative results so long as peri-bronchitic and pneumonic deposits are not massed together into extensive consolidations, so as to produce dulness and bronchial breathing. But a protracted catarrhal affection of the apices must always be regarded as a grave sign, because it tends to such consolidations. It is chiefly from pyrexia being persistent, impairment of general health, loss of flesh, and pallor of the skin, that a diagnosis is made as to the presence of catarrhal inflammation of the bronchioles and air-cells; but from which, by careful management, recovery may yet be made. Catarrh of the apex of the lung is not always a certain sign of pulmonary *tubercles.* It is a sign *that the patient is in danger of suffering from destructive lung disease.* (10.) *Dulness on percussion, bronchial breathing, and consonant râles in the upper part of the thorax,* are probably never caused, in the first instance, by tubercles alone, but indicate consolidation, the *residuum* of a pneumonic process. (11.) *Flattening of the supra- and infra-clavicular regions, on one or both sides, and lower situation of the upper edge of the lung, with diminished respiratory movement,* are certain indications of induration and shrinking of the apex of the lung. Thus far it may be a curative process, only apt to be compromised by a fresh pneumonic attack, or a subsequent deposit of ubercle.

(*a.*) *Symptoms of Acute Pneumonic Phthisis.*—The inflammation of the ung which precedes a destructive process may be either acute or chronic, ssociated with hæmoptysis or extensive bronchial catarrh. If the fever of an acute pneumonia does not cease at the end of the first, or beginning f the second week; if in the evening hours the fever rises considerably, when it only remits in the morning with considerable sweating; when percussion-dulness persists, and liquid clanging rhonchi are still heard ver the affected part, then acute pulmonic destruction may set in. The puta become muco-purulent. Microscopically, the characteristic elastic bres of the lung are to be seen in them. Induration of the lung is oftening and breaking up. The diagnosis of acute pulmonary destruction is no longer doubtful, which may prove fatal in a few weeks if the fever is violent. Sometimes the fever ceases, expectoration diminishes, and the patient improves, retaining signs of induration and retraction of the parietes of the chest over the affected part. There may be signs of a cavern (generally from bronchiectasis) in the pulmonary substance. When an acute catarrh is likely to terminate in progressive destruction of the lung, that result is generally indicated by the comparative gravity and endurance of the symptoms, rendered more certain by the sputa becoming rusty, and by physical signs of pleurisy or consolidation of the lung.

The chronic form of pneumonia which precedes acute pulmonary phthisis may begin very insidiously. A slight catarrh is usually the first indication of its commencement. The general health suffers, the patient losing his appetite, becoming thin, and then comes increased body-heat, with physical signs of consolidation and softening of the lung. It may tend, on the one hand, to recovery or to extension, more or less rapid, of the induration. The typical cases are such as find their way into hospital one winter after another, improving under the careful nursing, food, and medicines for the time; but each attack spreads the induration and subsequent softening over a greater extent of lung, till at last they die from a severe pneumonic process or extensive infective growth of tubercle in the lung. This latter event is the greatest danger to most consumptive patients. The deposit may occur at any stage; and no form of destructive lung disease can ever warrant an unconditionally favourable prognosis.

(*b.*) *Symptoms of Tuberculo-pneumonic Phthisis.*—These cases are chronic, and the tubercular element is indicated in the commencement by slight cough of a dry hacking character, mostly induced on rising in the morning and going to bed at night. The cough seems simply intended to clear the throat. The irritation is mainly referred to the pharynx, which is red, rough, and coated with tenacious mucus. As the cough increases a scanty expectoration occurs, of ropy or glairy mucus, hardly discoloured, or slightly stained by a trace of blood. Dyspepsia, sick headache, biliousness, and loss of appetite prevail. The patient is feeble, easily fatigued, feels unequal to his usual work, has burning heat of the soles of the feet at night, and some perspiration in the morning. His nights are restless, so that he rises in the morning weary and unrefreshed by sleep. He is irritable, often depressed in spirits, his appetite capricious, with dyspepsia (a constant, important, and early symptom), and there is a sensible loss of flesh. The muscles become flabby, the countenance pale, the conjunctiva pearly-white, and the pupil dilated. These symptoms are accompanied by a permanently accelerated pulse, from 90 to 140. The cough begins to recur at intervals during the day, especially after the least exertion. He feels "short-winded,"—active exercise exhausts him, so that he must rest at intervals if he walks fast or goes up a stair. The number of respirations per minute is increased, the pulse accelerated, especially towards evening, and deficient in force. Febrile paroxysms ar of daily occurrence; and elevation of temperature is a constant phenomeno capable of being appreciated for many weeks before physical signs becom decided (DAVY, RINGER). There is also correspondence between th elevation of temperature and the activity of the growth of tubercles in th lungs; such that when it goes on rapidly the elevation of temperature high, and *vice versa.* This elevation of temperature is persistent, varying from 103° to 104° Fahr., for several weeks before diminished weight or physical signs indicate the undoubted presence of tubercle (RINGER). This state of things may last a few weeks or a few months. The patient often revives, and seems, to an unpractised eye, for a short time to have recovered his good general health, were it not for a sense of weakness and undue exhaustion after such exercise as he has been daily accustomed to. But the disease silently proceeds. All the symptoms are gradually but sensibly aggravated. A large amount of material may have grown during the earlier stage; and after a time the intervening portions of lung become so congested that bronchial irritation, with intercurrent attacks of pneumonia, are of frequent occurrence. Rapid softening takes place in those in whom constitutional weakness or vulnerability is intense; and with the continued discharge of pus from the lungs, hectic fever becomes permanently established. The sweat from the head and chest towards morning is often so profuse that the patient lies bathed in perspiration. The cough becomes more distressing, the sputa purulent, and the pulse more frequent—90 to 110. Emaciation is well marked. The duration of these phenomena is indefinite—a few weeks to many months may end in a fatal issue. The disease occasionally intermits and becomes latent, so that there may be a marked amendment. The patient may regain strength—a most important issue, to be sedulously taken advantage of to promote the tendency to cure. But fresh tubercles may continue to grow during these intervals, so that the lung-tissue is further encroached upon; fresh local irritation is set up, all the general symptoms are aggravated; sickness and rejection of food are then excited—the expectoration becoming more profuse, of a purulent character, and often streaked with blood. When these symptoms attain their highest degree of intensity, and caverns are formed, hectic is followed by great exhaustion towards noon and at night. The appetite is lost. A colliquative diarrhœa may supervene from irritation, indicating the growth of tubercles and ulceration of the intestines. The sputa may be pure pus, as from an abscess; but they may become little more than a rusty sanguineous mucus. The pulse rapidly increases to 110 or 150. Emaciation is excessive; the hair falls of; catamenia cease; pleurisy or pneumothorax may supervene; ulceration of the larynx may occur; and, amidst this general wreck of existence, the mind is often firm, collected, and even hopeful to the very last.

As soon as the cavity which forms is in free communication with the bronchi, the cough may be greatly relieved, and the duration of this stage may last many months. As the fatal end approaches, the appetite fails; sleep can only be obtained by narcotics; the integuments on which the patient lies are apt to inflame, and even to die from constant pressure; œdema of the feet and ankles sets in, or ascites; and with the approach of death, suffocative dyspnœa may render the death-struggle extremely painful. In other cases a wandering delirium enshrouds the mind, or coma supervenes. A prolonged and weary illness is thus tranquilly ended by the gentle approach of death.

As there is no *single sign* by which the existence of tubercles in the lungs is clearly indicated, the general symptoms must invariably be judged of in combination and comparison with the information obtained by *physical signs.* Most physicians believe that the slightest subclavicular dull percussion sound, the less forcible, deep, harsh, or tubular inspiration, with lessened vesicular murmur, prolonged expiration, increased vocal resonance, and flattening of the apex of the lungs, when combined with the well-known general symptoms already mentioned, leave little doubt that the actual presence of destructive lesions, and perhaps, but not necessarily, tubercle is indicated. The value of thermometric observation, as indicative of a pretubercular stage of phthisis, has been shown by Professor Sidney Ringer. A persistent elevation of temperature is the invariable precursor of the growth of tubercle in any organ; existing for several weeks before diminished weight or other physical signs indicating the growth of tubercle in the lungs could be appreciated. After a certain time, however, growth of tubercle begins to be apparent by physical signs in the apices of the lungs. The temperature may be taken as a measure of the amount of the deposit, and any fluctuations in it indicate corresponding fluctuations in the intensity of the disease. It is a more accurate measure of the amount of lesion than either the physical signs or general symptoms.

The physical signs of pulmonary phthisis, in the order of their relative frequency, may be stated as follow:—*Dulness of percussion* (constant): *deficiency of respiratory murmur; bronchial voice; rough inspiration; expiration prolonged; mobility of chest wall lessened; flattening of chest wall; tubular quality of respiration; dry crepitation; wavy inspiratorg sound.* The signs denoting a few small scattered tubercles are,—(*a.*) *Inspiration rough and dry, and its intensity increased;* (*b.*) *Expiration prolonged—compared with its normal intensity and duration, as eight to two;* (*c.*) *Bronchophony in rare cases* (POLLOCK). Of indurations, and perhaps tubercle in groups or masses, the physical signs are,—*Pulmonary crumpling sounds; dry crackling rhonchus; sonorous sibilant rhonchus, indicative of bronchial irritation; inspiration rough and dry, intensity increased; the intensity and duration of expiration increased, and its quality blowing; slight bronchophony; diminished local fremitus; slight dulness, localised and distinct; heart's sounds transmitted; and, in certain cases, a subclavian murmur.* The relative frequency of the physical signs are as follows, commencing with the most frequent:—*Mobility of chest walls lessened; flattening of chest walls; dulness on percussion; deficiency of respiratory murmur, rough inspiration; expiration prolonged; tubular quality of respiration; bronchial voice; dry crepitation; wavy inspiratory sound* (POLLOCK). If the chest of a patient labouring under incipient phthisis be carefully examined, we may at first observe nothing remarkable, unless the indurations shall be large in amount or confined to one side of the chest. Abnormally rapid respiratory movements indicate a large amount of tubercle. There is decreased expansion of the chest in the *infra-* and *supra-*clavicular regions on the affected side, or flattening of the chest walls at the place affected. If both sides are equally affected, this flattening may escape attention. If the disease be further advanced, the patient is emaciated, with a singular immobility or incapacity of dilatation of that portion of the chest immediately below the clavicle, so that he breathes chiefly by his shoulders and diaphragm. He is unable to "fill his chest," especially during forced inspiration. The respirations are greatly increased in frequency, and in the later stages of the disease the respiratory movements are still more rapid. The clavicles appear peculiarly prominent in consequence of the flattening, depression, or falling in of the *supra-* and *infra-*clavicular regions. A fluctuating impulsive movement may be obvious in the upper intercostal spaces, generally on the left of the sternum, due to the action of the pulmonary artery or base of the heart. At a still more advanced stage, if an abscess has burst into the cavity of the pleura, and caused *pneumothorax*, the affected side is not only motionless, but distended, and, as it were, bulging out. *Palpation* during the *early stage* may disclose deficient expansion in the *infra-* or *supra-*clavicular regions; and if there is increased vocal fremitus at the apex of the lung, it betokens consolidation there. In the *advanced stages* of the disease, marked vocal fremitus betokens a large cavity, superficial, and in free communication with the air-passages. It may be rhonchial, or gurgling, or like fluctuation. If the lung has shrunk, the heart may be felt beating out of its proper place. *Mensuration* with callipers, in the early stage, may detect a slight diminution in the *antero-posterior diameter* of the *infra-clavicular* region, and a decrease in the local expansion movement. As the disease advances, the size of the side affected diminishes both in its *transverse* and *antero-posterior* diameter. *Percussion* during the earlier stages, so long as air penetrates the pulmonary tissue equally on both sides of the chest, does not convey any definite information. Dulness does not always follow even when considerable consolidation exists at the apex, unless the masses are very superficial, when the sense of resistance to the fingers is increased. Percussion should be practised during deep inspiration, and then during deep expiration. If tubercles exist, percussion, under a full inspiration, shows the resonance *increased* on the affected side, but very *slightly* as compared with that on the healthy side; whereas, percussion under a full expiration shows the dulness to be greater on the affected side than over the healthy lung (FULLER, pp. 42, 44, 365). It is differences rather than actual quality of sound on which an opinion must be based. In the more advanced stages, percussion elicits evidence of dulness more intense and more widely spread. In the later stages, where *vomicæ* exist, percussion will be absolutely dull if one or more small vomicæ are filled by purulent material, or surrounded by consolidated lung. If, however, the same cavities are empty and superficial, without thickening of the pleural membrane, the dulness may be slight, or the sound on percussion resonant, though shallow or amphoric. "Large empty vomicæ, with thin tense walls, yield an amphoric or cracked-pot resonance, and, except in rare instances of enormous superficial vomicæ, with thin tense walls, it is almost impossible to judge of the size of a cavity by the results of percussion" (FULLER, *op. cit.*, p. 369). A superficial empty cavity, with resilient walls, having free communication with the upper air-passages, yields a cracked-pot sound, especially when the mouth is open, and all obstacles to the egress of air removed. *Auscultation* yields extremely variable results. Slight harshness of respiration is the first indication noticeable, with prolongation of the *expiratory* murmur, and "jerking irregularity of the respiratory sounds." When these phenomena are persistent, and confined to one side of the chest, they indicate phthisis, especially if the phenomena of "dry clicking" be added. These phenomena are earliest marked in the *supra-scapular, supra-clavicular,* and *infra-clavicular* regions. If the physical signs of bronchitis (namely, coarseness of respiration, with small bubbling râles and sonorous sibilant rhonchi) are persistent in these regions, and inaudible below the second intercostal space, and still more so when confined to one lung, their existence warrants the suspicion of phthisis. If the disease is advanced, and new growth involves bronchial tubes of considerable size, there is almost entire absence of vocal resonance and of respiratory murmur over the part affected, with the sounds of breathing exaggerated in the adjoining parts. If consolidation is extensive, the sounds of the heart and large vessels are transmitted to a greater extent than in health. The diagnostic value of murmur in the pulmonary artery is variously estimated. It is often present in many phthisical patients at the second left sterno-costal articulation (FULLER, p. 366. See also SIEVEKING, in *Lancet*, Feb. 11, 1860). As the disease advances into the second stage, auscultation suggests thin irregular-sized bubbling râles, from the passage of the inspired air through the softened and liquefied tubercles. It may also prove that coarse and hollow-sounding respiration exists over a more extensive surface than heretofore; or that the respiratory sounds are of a blowing character (bronchial breathing). In still more advanced conditions, with more or less empty cavities, the respiration is heard to be blowing or amphoric and metallic; and if the fluid contents of the cavity rise above the level of the permeable bronchi which lead into the cavity, large irregular bubbling râles or distinct gurgling may be heard. Well-marked pectoriloquy (from a mere whisper) is the form of vocal resonance most pathognomonic of pulmonary tuberculosis (FULLER, l. c., pp. 363 to 370).

(*c.*) *Acute Miliary Tuberculosis, or Primary Tubercular Phthisis,* from its rapid course, has also been named acute pulmonary consumption. It is expressed by febrile symptoms running an extremely rapid course, denoting the severity of the constitutional disturbance, proving fatal in from twenty days to ten or twelve weeks; and due to (1.) acute miliary tuberculosis, or (2.) acute pneumonic phthisis, or to both combined. It has usually been considered a comparatively rare disease; but it would appear to be unusually prevalent among the soldiers in Paris, among whom also the chronic form of phthisis is very common, probably more so than even in our own army (PARKES, *Army Med. Dep. San. Report,* 1860, p, 357). The disease occurs in two forms:—(1.) The result of extensive infiltration of pneumonic material in the lungs, with irregular softenings in the centres of some, or with small excavations surrounded by patches of fresh hepatisation. (2.) That in which there is general studding of both lungs with semi-transparent grey granulations, combined with pneumonia in its firs stage, bright arterial injection, or hepatisation (WALSHE). The two form may co-exist in the same lung.

The **Symptoms** are those of an intense febrile affection. During *thirt* days of a fatal case recorded by Dr. Parkes, the pulse ranged from 11 the lowest, to 178, the highest, and having a mean of 140, reckoned b the number of observations. The respirations *per minute* ranged from 3 to 60, having a mean of very nearly 50. The temperature usually fluct ates over 102° and under 104° Fahr.—once or twice only exceeding 104 and thrice only getting below 102° Fahr. The functions of the lungs a deeply impaired. The invasion occurs while the patient is in a state of health, or the fever may be remotely preceded by various depressing influences, such as immediate exposure to cold and wet; after which rigors ensue, followed by acrid heat of skin. The rigors recur on several successive days, followed by perspirations, and sometimes crops of sudamina. Epistaxis, followed by coryza, may occur on the second day of seizure (WALSHE). Prostration sets in early, so that in a few days the patient may be unable to stand. There is thirst, total anorexia, epigastric tenderness, dry lips and tongue, dental sordes—all signifying great intestinal disturbance. Diarrhœa is rare, and constipation may be extreme, even with abdominal pain and ulcerated intestines. Restlessness, *insomnia, cephalalgia, vertigo, tinnitus aurium,* diurnal wandering and nocturnal delirium, bespeak cerebral complications and the probable growth of tubercles in the arachnoid membrane. The physical signs vary with the amount of tubercular growth or *pneumonic infiltration* in the lung, and are similar to those which have been described.

Prognosis.—Pulmonary phthisis sometimes terminates life within a few weeks, or extends over six or eight months; or it may occasionally last several years, with marked intermissions in its progress. There are certain circumstances which promote the chances of a remission, of which important advantages may be taken in the treatment—namely, (1.) Originally good constitution; (2.) non-inheritance of scrofula; (3.) direct inheritance of longevity; (4.) limited extent of local lesion; (5.) integrity of other organs, unimpaired digestion, absence of fever, vigorous nervous system, quiet pulse; (6.) influence of age—the period when the young body is making its growth is the period of greater danger. The tolerance of lesions and the periods of remission are less during the growing age, and are increased just when the body has completed its process of increase. Improvement in cases of pulmonary phthisis has been effected, by medical treatment in hospitals, in about 14 per 100 more frequently in persons following "open air" and "medium" occupations than in those whose trades are "confined;" but that *death* or *deterioration* was as frequent as improvement in those who followed confined trades. The chances of improvement are 5 per cent. greater in persons who come from the country to a salubriously situated hospital in town than in townspeople (WALSHE). More than half the cases of phthisis undergo temporary stages of improvement, more or less permanent; and the time the disease has existed, rather than the stage the disease has reached, is an important element in calculating the probable benefit a patient may derive from treatment in hospital. A natural tendency to a slow course is a more important element of success in the treatment of the disease than the fact of that treatment having been undertaken at an early period (*Med.-Chir. Review,* Jan., 1849). The cases of phthisis more usually rapid are such as arise from hereditary constitutional causes, or from the influence of exanthemata, especially measles, or of typhoid or other fevers. These run their course with implication of several organs at an early stage of the disease. The mortality numerically from consumption is much higher than from any other disease in this country; and amongst the class of society resorting to assurance offices the mortality seems to be about one-half of the whole.

Treatment.—Looking to the varied pathological conditions and textural lesions in the lungs comprehended under the name of phthisis, every case requires special study. In one case anæmia is prominent, and calls for *iron*, perhaps for *arsenic;* in another, continued but small hæmoptysis may call for *ergot;* in another, a racking and frequent cough may call for special applications and remedies; in another, dyspepsia may call for *alkalies* or *acid bitters*, or *pepsine;* in another, nervous unrest may call for *bromides;* in another, laryngeal troubles may call for special local medication; in another, we have to aim at controlling excessive sweating, or checking an exhausting diarrhœa (Dr. JAMES SAWYER, *Brit. Med. Journal*, 1880, p. 845).

If, unfortunately, a pulmonary catarrh does not abate, or a pneumonia does not resolve itself within the usual period, but catarrhal symptoms prevail in the apices, and pyrexia is persistent, with the severe disturbance of the general health which has been described, extension of the pneumonic process must be prevented by absolute rest in bed, talking being forbidden, and coughing as much as possible repressed. Whenever considerable pyrexia exists in the evening, all duty occupations should be given up, strict rest, equal temperature, the chest to be covered with *linseed poultices*, frequently repeated, or *belladonna linament*, and *local bleeding by leeches*, if pleuritic pains exist. No counter-irritation should be applied to the seat of lesion while pyrexia lasts. Moderate action of the skin is to be maintained. The body should be sponged with *rectified spirits of wine and hot water* in equal parts (COGHILL). *White meats only* are to be eaten: and as much *milk* as may be desired. A pint of "*warm milk from the cow*" should be taken as often as possible.

Should the pyrexia still continue considerable, *digitalis*, *opium*, and *quinine* are the remedies most successful in reducing temperature. Heim's pill was much employed by Niemeyer, as it still is in Germany, for this purpose. It consists of *Pulv. herb. Digitalis*, gr. x.; *Pulv. rad. Ipecac.*; *Pulv. Opii puri.* a a., gr. v.; *Ext. Gentiani*, q. s.; *misce et divide in pil. xx.* One pill three times a day. It is of advantage that quinine to the extent of gr. xx. be added to this. Each pill will then contain half a grain of digitalis, one quarter grain of opium, one quarter grain of ipecacuanha, and one grain of quinine; all of which may be held in form by glycerine or syrup. Dr. Coghill adopts the following modification:—namely, one quarter of a grain of *opium;* half a grain of *digitalis powder;* and from one to three grains of *quinine*, to be given every three to six hours, watching the effect on the pulse. When there is great dryness of skin and tongue, half a drachm of *salicine*, and two drachms of *Liquor ammonia acetatis*, may be given instead of the pill. Occasional intercurrent attacks of pneumonia mark extension of lesions. They are *asthenic* in character, and must be treated accordingly; but in all the sthenic pneumonias or acute bronchitic attacks, no remedy equals th *tincture of aconite.* It acts as we read of blood-letting doing (see Vol. I p. 322). It should be given at the rate of *one minim* every quarte of an hour till the temperature is reduced to the normal—the thermomete being in use every two hours. If the temperature drops too suddenl *quinine* must be given in place of *aconite.*

The general principles of treatment described under "Scrofula," Vol. I are to be adhered to. The use of so-called "cough-medicines," *antimony*, *ipecacuanha*, and *squill* especially, are to be avoided; also the use of *mercury, purgatives,* and *neutral salts.* It is unphysiological to administer narcotics or sedatives when cough is accompanied by expectoration. The latter must be expectorated: if retained, it is injurious. The morbid surface which produces the expectoration must be directly acted on. In the suppurative expectoration of bronchitis or pneumonia, and that from a pneumonic or tubercular excavation of the lung, the processes are identical with the suppurative processes in surgical wounds or ulcers. Dr. Coghill finds that the best mode is to bathe the suppurative surface in an antiseptic atmosphere by the inhalation of such materials as *creasote, carbolic acid,* and *thymol.* Warm moist inhalations do not affect the respiratory tract beyond the larynx; and are only of value in affections of that region. When used warm they increase the sensitiveness to cold. By using the breath only as the antiseptic medium (inhaling through the mouth and respiring through the nose), we get the whole of the residual air in the lungs saturated with the antiseptic material. This is the principle of Dr. Coghill's "respirator-*inhaler*," which should be used especially after "fits of expectoration," so as to act immediately on the surface which has furnished the matter expectorated. It cannot be used too often; and should quite supersede "cough mixtures." It is, of course, only of use in the second and third stages of phthisis and the suppurative stage of bronchitis; and especially in the retained fetid expectoration of *bronchiectasis.* Dr. Coghill's standard formula (to be modified at discretion) for an antiseptic solution consists of one drachm of *glycerine,* one drachm and a half of *creasote,* two drachms of *carbolic acid,* and one ounce of a *saturated solution of thymol:* of which ten to fifteen minims are to be inhaled twice or thrice a day by the respirator-inhaler. A few drops of *chloroform,* or of *ether,* may be added as a sedative to each dose. In the first stage of phthisis and other conditions where there is cough and no expectoration, and in which the cough is injurious by shaking and shattering the tender and infiltrated lung tissue, the following is an excellent formula :—namely, *Croton-chloral hydrate* and *codeia,* āā gr. xij.; *Aqua Laurocerasi glycerine* and *Syrup of tolu.* āā ad. ℥ij. A dessert-spoonful, or two teaspoonfuls, may be taken every two hours when cough is troublesome.

To foster and cherish an appetite for food is another great aim of reatment. One of the best tonics, if pyrexia does not exist, is the syrup of *iron, quinia,* and *strychnia*—of which the formula by the late Professor Easton, of Glasgow, has been given at p. 1099, Vol. I. Let it be given in very small doses at first; followed up by the use of cod-liver il "of the most agreeable, clearest, sweetest, and most scentless kind." The *brown oil* ought never to be prescribed, except, perhaps, to an Esquimaux. The oil should be given in *teaspoonful* doses at first. Dr. B. W. Foster, of Birmingham, drew the attention of the Profession to he advantages of *ether* in promoting the digestion and absorption of atty food, and especially of cod-liver oil. A stomach once intolerant f all fat will good-naturedly accept full doses of cod-liver oil after the se of ether. Sometimes it has been given separately, before or after he oil; but it is best given combined with the oil—a mixture now enerally known as "*Etherised cod-liver oil.*" To every two drachms of the *oil* ten, fifteen, or twenty minims of *ether* are to be added, according to the wants of the case. The *Æther purus* of the British Pharmacopœia must be used, so that the oil may not be rendered muddy, as it will be if the ether contains alcohol or water. The ordinary dose of oil ought not to exceed two drachms, to which more than twenty drops of ether may be added, if necessary. The ether is indicated wherever there is inability to digest fat, of which it makes an emulsion. It seems to determine, when introduced into the stomach, a considerable flow of pancreatic juice (CLAUDE BERNARD; See *Brit. Med. Journal,* Nov. 21 and 28, 1868; also, *Medical Press and Circular*, 1869). *Extract of malt* (TROUSSEAU) is a remedy of recent repute. It is a genuine extract, consisting of the soluble constituents of the malt and of the bitter extractive matter of the hop. Two or three table-spoonfuls of it are to be taken daily, diluted in spring water or in warm-milk, or in any other liquid that may be desired. There should be a careful choice of meats and drinks. Meats rich in *osmazone* and roasted are to be preferred; wines and malt liquors are better than mere alcoholic fluids when alcoholics are required. Broth made of coarsely brown rye meal, beef, mutton, or other flesh meat, boiled to make a soup, is also of service as a diet. I have found the following soup also very serviceable :—Take of *linseed* half an ounce; *fine bran,* one ounce; *water*, one quart. Boil these for two hours, and strain then add beef, mutton, or any other meat that may be fancied, to the amount of one pound, and boil to a soup, with vegetables, to which celery seed or other flavouring may be added. The whole quantity ough to be reduced one-third.

Arsenic is an efficient improver of the general nutritive and assimilativ functions. *One* or *two* drops of Fowler's solution, or $\frac{1}{50}$ of a grain o arsenious acid, should be given once, twice, or thrice a day, *during the mea* continuing it for months, with occasional intermissions (CLYMER). Decide benefit may follow the use of *iodide of potassium,* which may be combine with the *ammonio-citrate of iron,* or *tincture of the muriate,* in a bitt infusion with *calumba* or *cascarilla. Chloride of calcium* has been used f some years by Professor James Sawyer, of Birmingham, who writes ve highly of its advantages in combination with cod-liver oil or its etheris emulsion. He gives 10 grains, dissolved in a drachm of water and wi a drachm of glycerine, in a wineglass of milk, twice a day, immediat after meals (*Brit. Med. Journ.*, June 5, 1880). Dr. Coghill, from extensive experience at the large hospital at Ventnor, regards t *hypophosphites* and *lactophosphates* alternately as the best general tonics phthisis. They are in no way *specific.* They should not be given if e temperature in the evening is above 99°·4 Fahr., and, on account of th r *pyretic* character, should only be given after breakfast and lunch. e *hypophosphite of iron* is so powerfully *pyretic* as only to be given in extr e anæmic phthisis, where the whole daily thermometric range is low. s formula consists of *Hypophosph. Sodæ, Hypophosph. Calcis,* ā ā, ʒi.; *Glyce i vel Syr. Limonis,* ʒij.; *Decoct. Cascarilla ad.*, ℥vj.; of which a table-spoo l in water may be taken as required, to which three to five minims of *q. strychniæ* is a good addition to increase appetite. The *pharmacopœal sy of the lactophosphates* is very good; also the French "Dusart's." I an aperient is wanted it should be given early in the morning. Its nece ty very often in phthisis depends on a sluggish liver, hence the following is a good formula:—*Salicylatis Sodæ*, ʒij.; *Phosph. Sodæ*, ʒv.; *Sulph. Potass. ad*, ℥ij.; *Pulv. Zinzib*, ʒi.; of which a teaspoonful in hot water is to be taken early in the morning. If there is *an elongated uvula*, the end should be snipped off; if there is irritation about the pharynx and upper part of the larynx, it may be relieved by the inhalation of some of the atomised fluids.

Counter-irritation to the chest-wall is a stereotype method of cure in local pulmonary congestions. When properly used in the earlier stages, before there is much loss of strength, it is undoubtedly beneficial; but later it is weakening and annoying. Croton-oil liniment is the chief favourite; but a prompt and not too severe application of the following ointment is recommended by Dr. Fuller:—

℞. *Hydrarg. Chlor. Mit.*, gr. viij.; *Iodinii*, ʒss.; *Alcohol*, fʒiss.; *Unguent. Simp.*, ℥j. M. Rub in a portion over the affected lung, morning and evening, until a pustular eruption comes out.

A solution of the nitrate of silver (thirty grains to the ounce of distilled water) may be painted on the skin beneath the clavicles every evening until the skin is darkened, and repeated after the cuticle peels off. Gentle and continuous irritation of the skin may be kept up by wearing constantly on the chest a piece of flannel wet with a weak solution of iodine in glycerine and water, covering the cloth with oilskin, to prevent too rapid evaporation and soiling of the clothes. The *night-sweats*, so often annoying and profuse, diminish under general systemic improvement and restored cutaneous functions. The best *anhydrotic*, and almost the only one in use at the hospital at Ventnor, in the Isle of Wight, is a pill at bedtime, containing $\frac{1}{80}$ of a grain of *atropia*, which may be combined with *digitalis* and *quinine*. Bathing with *vinegar and water*, *spirits of wine and warm water*, *alum and water*, are well-known remedies, as well as *gallic and tannic acids*, and *infusion of common garden sage*. *To relieve the diarrhœa* Dr. Coghill finds the *lead and opium* pill of the British Pharmacopœia the best astringent, with a *milk and arrow-root* diet. A flannel bandage around the abdomen should be worn. *Bismuth*, with *opium*, is also of use. To control the exhausting attacks which occur in the more advanced stages, Dr. Frank, at Cannes, suggested the use of *coto bark;* and Dr. J. B. Burney Yeo (after an experience of over three years) has found it to arrest the flux and relieve intestinal pain and irritation in a short time. A *fluid extract of coto* is prepared by Ferris & Co., of Bristol, the dose of which is from five to eight minims (*The Practitioner*, 1879, p. 257). The treatment of *Hæmoptysis* has been already indicated at p. 742, *ante*.

As regards the prevention of phthisis, although Sir Thomas Watson states that he does not believe it to be contagious, he adds:—"Nevertheless I should, for obvious reasons, dissuade the occupation of the same bed, or even of the same sleeping apartment, by two persons one of whom was known to labour under pulmonary consumption" (*Lectures on the Practice of Physic*). "It behoves the physician to warn the patient's friends of the dangers incident to long-continued attendance on him, especially if the disease be in an advanced stage. It would be the height of imprudence for a healthy person, especially if young and of a scrofulous diathesis, to sleep in the same bed, or even in the same apartment, with a consumptive patient; for although the malady might not be communicated directly from one to the other, unless possibly under the condition of some tubercular matter being accidentally introduced into his air-passages or into some other part of his system, the surroundings and the air would be calculated to predispose him to the disease" (Fuller, *On Diseases of the Lungs and Air-passages*, p. 431; see also p. 750, *ante*).

It is of importance to know the physical nature of the climates of the world suitable for the tuberculous patient. (See Appendix to third edition of Dr. Walshe's work *On Diseases of the Lungs;* the late Sir James Clark's classic work *On Climate;* and Dr. Theodore Williams's Lettsomian Lectures in *Brit. Med. Journ.*, 1876.) Dr. Williams's statistics show the benefit to be derived from life at sea. In cases where destructive lung disease is suspected, but in which actual lesion does not exist, a sea voyage to the Cape, or to Australia, or to New Zealand, round Cape Horn, should be resorted to at once. The prevention of certain forms of pulmonary phthisis may be looked for in the unrestricted enjoyment of pure out-of-door air. Continuous indoor labour is to be avoided, and measures taken to prevent bronchial catarrhs by sufficient warm clothing and the constant use of flannel next the skin in this country. The effects of hydro-therapeutic treatment and of muscular exercise at the mountain sanitoria of Switzerland, along with the prescribed useof wine, fruit, and nutritious diet, are of much importance to be considered. The purity of the air of mountains consists practically in the absence of all organic particles. The transparency of the air, which is dependent on its greater dryness, and the more powerful action of light, depending upon the same cause, aid materially the operation of its purity. The effect of residence in mountains on the dimensions of the chest is also worthy of careful consideration, and has been studied by M. Armieux, at Barèges, at a height of more than 3,000 feet. He ascertained that in the case of ninety-six soldiers sent up to Barèges there was, after four months' residence, a distinct increase in the measurement of the chest—a fact which has a very important bearing on cases of threatened phthisis in the young and that a mountain climate is to be considered as favourable to th development of the thoracic cavity, and consequently as improving th respiration. (See also Dr. W. Marcet in *Proceedings of R. S.*, Vol XXVIII., p. 498.) Mountain air may thus be useful in certain case and in certain periods of phthisis. It acts by its purity, by its drynes and transparency, as well as by the diminution of atmospheric pressure These principles have only, or nearly only, been applied in Switzerland but mountain stations for similar cases might very well be selected i some parts of France. After all, neither barometer nor thermomete neither hygrometer nor anemometer, can determine what is the suitabl climate for such and such a phthisical patient. All depends on th nature of his constitution, and on the way in which the variou meteorological conditions affect him, and on the local lesions in his lun (Drs. J. Macpherson and Gouraud in *London Med. Record*, July 1. 1876).

SECTION V.—DISEASES OF THE PLEURA.

PLEURISY.

LATIN EQ., *Pleuritis;* FRENCH EQ., *Pleurésie;* GERMAN EQ., *Pleuritis*—Syn., *Rippenfellent-zündung;* ITALIAN EQ., *Pleuritide.*

Definition.—*Inflammation of the serous membrane that lines the cavity and covers the viscera of the thorax. It is characterised at its outset by febrile chills, followed by an acute sharp pain in some part of the chest, frequently called* "a stitch in the side." *It is usually confined to one spot about the lateral regions of the thorax. The acts of respiration are performed rapidly. They are shallow, irregular, and incomplete. A dry short cough supervenes, and the pulse is hard and quick. The natural serous secretion of the pleural sac is arrested in the first instance, but soon becomes increased in quantity and altered in quality, having a tendency to assume the* corpuscular *character. The effusion may rapidly increase, and may ultimately assume a sero-purulent character, the parietes of the corresponding side of the chest being dilated accordingly.*

Pathology.—(*a.*) *Causation.*—Pleurisy may acknowledge an *idiopathic origin,* as when it arises from exposure to cold, especially by currents of air when the person is heated. But many disbelieve in cold being able to establish pleurisy in a healthy person (HYDE SALTER, FULLER). When exposure to cold is followed by pleurisy, it is most likely due to some *morbid condition of the blood,* as in rheumatism (*rheumatic pleurisy*), or to *constitutional causes. Anæmia, pyæmia,* the specific toxæmia of *eruptive fevers, small-pox, scarlatina,* the *materies morbi* of *rheumatism, gout, scrofula, carcinoma, Bright's disease, typhus, enteric fever, pyæmia, puerperal fever, alcoholism, syphilis, or even retained excreta, with pyrexia as the result of over-feeding,* are all apt to set up inflammation in the pleura. Or pleurisy may acknowledge some *adjacent irritation,* as *pneumonia,* constituting *pleuro-pneumonia,* when the inflammation spreads by continuity from neighbouring parts. There are also numerous cases in which some growth in the lungs, such as *tubercle* or *cancer,* advances to the pleura; or in which cavities in the lungs or abscesses of the liver may rupture into the pleura; or it may be due to wounds and injuries, such as a fractured rib. Dr. Anstie also suggests extreme muscular over-exertion in continuous public speaking as a cause.

HYDROTHORAX, or "*water on the chest,*" is generally traceable to increase of lateral pressure within the veins, especially obstructed action of the ight side of the heart, and decrease in the amount of albumen in the erum of the blood (NIEMEYER). Like other dropsies, it is a symptom of, ather than a substantive disease; and ought to be returned under the rimary ailment of which it is a secondary affection. It is one of the esions in *general dropsy.*

(*b.*) *Morbid Anatomy.*—The inflammatory phenomena begin in the ubpleural tissue by hyperæmia of the vessels, and afterwards the red lood penetrates the vessels of the pleura itself, and there is sometimes lood extravasation. It becomes thickened and swollen from interstitial mph infiltration. The secretion from the serous surface is suspended at first, and the membrane is dry, with its free surface rough, from granulations consisting of newly-formed fusiform cells, and filaments of connective tissue, with elongated delicate capillary vessels, coiled into loops within the new growths (FOERSTER, p. 590, *ante*). There are at least two distinctive forms of pleurisy—namely, (*a.*) Where the pleura thickens, and the material of inflammation is of the adhesive kind, gluing the opposed surfaces together; (*b.*) where the pleura also thickens, but the materials of exudation, after pushing off the epithelium, continue to produce corpuscular forms, and fluid accumulates within the cavity of the pleural sac. The quantity of fluid effused varies much, from a few ounces to many pints, separating the usually opposed surfaces, distending the cavity, and compressing the lung. Its amount may cause displacement of surrounding organs—heart and mediastinum, depression of the diaphragm, expansion of chest walls, and bulging of intercostal spaces. If the effusion be considerable, the lung collapses, contains no air, and no longer crepitates. It is then said to be "*carnified;*" its vessels are devoid of blood. The bronchi, even to the large trunks, are contracted, and the whole lung is compressed towards its roots into the grooves formed between the sides of the bodies of the vertebræ and the heads of the ribs to their angles. If pressure is soon removed and the lung be inflated, it will still expand more or less perfectly. Usually it is covered over and hidden from view, by a layer of lymph continuous with that which lines the thoracic parietes. Should the pleuritic effusion be less in quantity, some fluid appears spread all over the lung. The greater quantity is collected at the lower portions of the chest; or it exists in cavities or loculements, formed by masses of lymph in septa. The lymph may be loose, rendering the serum turbid or flocculent; or it is more solid, and adheres with tenacity, becoming organised at both surfaces. The formation and vascularisation of these membranes are rapid—often effected in the course of forty-eight or even twenty-four hours. If the patient dies shortly after an attack, these adhesions are soft, easily lacerable, and extensible; if he survives a longer period, they may be of greater tenacity, indurated, and with difficulty broken through. Their most common seat is over the anterior lobes o the lungs, or the portion of pleura from the mamma to the axilla Extensive *adhesions* are characteristic of dry pleurisy. *A scanty but fibrinou* effusion generally accompanies acute or chronic pneumonia, cirrhosis, o other forms of pulmonary phthisis. *Abundant sero-fibrinous exudatio* consists of—(*a.*) A yellowish green serum; (*b.*) a quantity of coagulate fibrinous masses which float in the serum in the form of flakes, or whic traverse the serum in a loose network, or is precipitated on the pleur in the form of a membrane. A few pus-corpuscles may be found in th form of pleurisy, both in the fibrinous deposit and in the serum, and th transition to suppuration and *empyema* is very gradual. The serum turbid in proportion to the quantity of the pus, and the deposit is mo yellow. When pus accumulates in quantity, the condition is term *empyema*, which is said to be *true* when the pus is secreted by the pleu and *false* when it results from the bursting of an abscess of the lung in that cavity. The quality of the pus in *true empyema* varies from a laudab pus to a sero-purulent fluid. In quantity, it varies from a few ounces many quarts. It sooner or later tends to point, and may open into t lung; or make its way through the chest walls, and form a sinus between the ribs, which may become carious; or it may accumulate into an abscess between the rib and the skin. But pus never makes its way externally until the lung has first become compressed (WILKS). Suppuration generally occurs in the pleurisy of *small-pox, scarlet fever, measles, pyœmia, puerperal fever,* and from perforation.

HYDROTHORAX occurs when a watery fluid or serum is effused into the cavity of the pleura of either or both sides. It rarely occurs in the absence of pre-existing disease of the pleuræ, lungs, heart, or great vessels, nor without the influence of some specific or constitutional disease, as a "passive dropsy of the pleura." The quantity of fluid varies from a few ounces to many pints—eight or nine pints. It is distinguished from that of *pleuritis* by the absence of fibrinous coagula.

PNEUMOTHORAX implies a collection of air or gas in the cavity of the pleura on either or both sides, generally co-existing with fluid, when the condition is termed *hydro-pneumothorax.* It may occur—(1.) When no communication exists between the pleura and the external air, and is due either to the spontaneous evolution of gas from decomposing fluid in the pleura, from gangrene of the pleural membrane, or from the secretion, generation, or exhalation of air from the pleura—a phenomenon of doubtful occurrence *per se.* (2.) When a communication takes place between the pleura and the alimentary canal, as in cases of softening and perforation of the œsophagus or stomach. (3.) When a communication occurs between the pleura and the atmosphere, through an opening in the chest wall, the result of penetrating wounds of the thorax, or of parietal abscess. (4.) When a communication occurs between the pleura and the bronchi, either the *result of violence,* rupturing the lung-substance, and tearing the pulmonary pleura; or the *result of disease,* causing perforation of the pulmonary pleura from without inwards, as in cases of *empyema;* or from within outwards, by ulceration. Such ulceration may be due to *destructive lung disease, hydatid cysts, cancerous growths, pneumonic gangrene, pyœmic* or *bronchial gland abscess;* or, it may be the consequence of *emphysema, pulmonary apoplexy,* or minute bronchial abscesses perforating the lung during the course of typhoid fever (GAIRDNER). To detect the existence of any minute opening in the pleura after death, the side of the chest affected should be filled with water, and if the lung is then gently inflated, bubbles of air will escape if a perforation exists, however minute. It commonly occurs in the area comprised between the third and sixth ribs (WALSHE, FULLER, CHAMBERS), "motion being greater there than in any other part of the chest," and therefore the pleura is less likely to be protected by adhesions there. Ulceration of the pulmonary pleura is the efficient cause of pneumothorax in 90 per cent. of the cases in which air is found in the pleura (WALSHE, FULLER).

General Symptoms and Progress of Pleurisy.—It may be acute or chronic. In the acute form its local symptoms are strongly marked, the patient suffering severe *continued pain* in the affected side, of "a dragging, shooting character," greatly exasperated by coughing or forced inspirations, movement, pressure, and percussion, so that the lungs can only be imperfectly filled with air. The costal pleura seems to be first affected as a rule; and the seat of pain is generally limited to one point;

usually about the centre of the mamma, or just below that part towards the lateral attachments of the diaphragm. While the pain is constant, it sometimes remits in severity, and with the occurrence of effusion often totally disappears. When effusion is scanty, localised and circumscribed to a small patch, the pain is generally piercing when a "breath is fetched," and greater during ordinary than forced respiration. It is described as "a stitch in the side." Coughing and sneezing are especially painful. Pressure from without or from within greatly increases the pain. Respiration is imperfectly carried out, in a hurried, shallow, and irregular manner. The body is bent towards the affected side. The tongue is commonly white. If the disease be limited to an effusion of lymph, the pulse is seldom more than 90 to 110, or 120, "hard and concentrated in impulse, but deficient in resistance." Either form of *pleurisy* is generally accompanied by a short troublesome cough, with or without expectoration. The respirations are increased in frequency (30 to 35)—a more constant symptom than even the local pain—and, unless dyspnœa exists, their frequency is unnoticed by the patient. While one *respiration* is performed, only *there* beats of the heart, in place of *five* or *six*, occur. The patient is restless, and at first lies on the affected side. If the effusion is great, the symptoms are more acute: rigors usher in more intense fever, with full and frequent pulse, headache, coated tongue and throat, pains in the back and limbs. Chills are recurrent (thus differing from pneumonia), so that the disease may be mistaken for ague. At the commencement, pain in the side is sharp; but abates as the fluid effusion rapidly increases, and often ceases altogether before effusion is complete. Dyspnœa is constant, and may become severe. The disease advances for six or eight days, when improvement ought to take place from rapid abatement of the fever, leaving the fluid to be reabsorbed, which absorption ought to begin immediately in favourable cases, and progress rapidly (NIEMEYER); but even after many weeks a considerable remainder of fluid may still exist. Or the fever, being acute at first, moderates towards the end of the first week, or a little later, but does not wholly subside; and although exudation does not continue, yet absorption remains in abeyance. By and by a reaccession of all the symptoms takes place, and effusion again occurs to a greater extent than before. In this way the disease may fluctuate for months, and, as a rule, it terminates fatally. Another form develops in a latent and slow way (*latent pleurisy*), without inflammatory fever and without pain—shortness of breath being the only source of distress to the patient, who ultimately seeks advice on account of failing strength, having become pale and thin. The abdomen may be tumid, from pressure downwards of the diaphragm. The amount of fluid in the pleura is then very great, and may be highly albuminous. Prostration and debility are extreme; and, finally, such cases generally end in pulmonary phthisis (NIEMEYER).

The physical signs of pleurisy are delicately varied according to anatomical conditions. At the earliest stage, when serous secretion is arrested, the expansion of the walls of the chest is diminished on the affected side, mainly on account of pain. The percussion sound is not perceptibly altered, and the sounds of respiration are weak, because imperfectly performed. The characteristic *friction-sound* may perhaps be detected, if listened for in the *infra-mammary* or *infra-axillary* regions. At first it is slight and grating, but becomes louder as lymph is formed. When the secretion has returned, increased in quantity, the signs continue as described; but the clearness of the percussion-sound becomes diminished, and the *friction-sound* is of a rubbing or grating character. The patient himself sometimes experiences a distinct sensation of friction. The period of inflammatory effusion being established, the infra-mammary and infra-axillary regions become more or less bulging; the projection of the intercostal spaces of the affected side during both respiratory acts becomes most obvious; the thoracic vibration from the voice is abolished where the fluid intervenes, and also friction-murmurs there. The area of dulness, and of the peculiar sounds, may be changed by altering the position of the patient. The natural respiratory murmurs become greatly intensified above the level of the fluid. When effusion exists on the right side, the sounds of the heart are more clearly audible than in the natural state in the right axillary region, because the lung is more solidified by the pressure of the effused fluid. But pleurisy may exist without any of the general symptoms. There may be neither local pain, cough, dyspnœa, nor febrile action, and yet effusion may have occurred to such an extent as to have reached the clavicle, while the patient remains utterly unaware that his chest was the seat of disease (WALSHE). The physical signs alone reveal the disorder—*latent pleurisy*—a form of disease which had no existence in nosology previous to the time of Laennec. Whenever, therefore, the least suspicion exists of disease in the chest, especially in elderly persons, or those liable to constitutional affections, percussion and auscultation must never be neglected; and the "*cyrtometer*" ought to be used to determine enlargement (p. 539, *ante*). In children the chest is dilated considerably at an early period, and there is less displacement of parts than in adults. If suppuration (*empyema*) is about to occur, the pulse is extremely small and frequent, from 120 to 150. Any acute pain which may have existed subsides, but the anxiety and restlessness of the patient are increased, and a state of collapse shows his imminent danger. The symptoms of *local empyema* may follow effusion coming on insidiously, but generally the patient is ill; and the history of an acute attack is always to be found—a partial absorption having taken place, leaving a certain amount of matter locked up at the bottom of the chest.

In hydrothorax the effusion may be so slow that the lung adapts itself to its presence, and the symptoms will be less marked, although the effusion may be large. When it occurs suddenly, the functions of the lung are almost at once suspended, the countenance is livid, and the breathing greatly disturbed. When the effusion is abundant, there may be difficulty of respiration (which is carried on rather by the shoulders and diaphragm than by the intercostal muscles), some expectoration, lividity of the face or lips, œdema of the legs, and either a full labouring pulse, or one that is small, frequent, and intermitting; the urine is extremely scanty. As long as effusion is moderate the patient can lie flat in his bed without experiencing any inconvenience. In the event, however, of the effusion being so considerable that the function of the lung is suspended, the patient is unable to lie down from the sense of suffocation produced by the fluid gravitating towards the root of the lung, and compressing the larger bronchi. He therefore sits propped up by pillows, with his head bent forwards. When hydrothorax is symptomatic, or consecutive to affection of the heart or of other disease, it is generally preceded by swelling of the legs or eyelids, by the urine being plentiful and albuminous, or scanty, high-coloured, and loaded with the usual salts, and by most of the symptoms of general dropsy. Such effusion seldom takes place into the chest till a few days before death, rendering its agony doubly painful and suffocating.

The signs of pneumothorax are, marked deficiency of respiratory murmur, without dull percussion, and with metallic phenomena, such as "a clear click, as of water dropping into a well" (*tintement métallique*), audible with almost every inspiration, and succussion sound in a more or less well-marked degree. But these phenomena only present themselves when pneumothorax is very extensive. Pain may be moderate in degree, or not characteristic. Dyspnœa may be merged in a more chronic affection of the chest; fever may hardly be observed amid the hectic of phthisis or of empyema; while the more special sensation of rupture, and the suddenly-felt rush of fluid or of cold air into the chest, are quite as often absent as not (GAIRDNER, l. c., p. 387). Signs of fluid as well as air in the chest (*hydro-pneumothorax*), may be discovered within a few hours after the presence of air has excited sufficient irritation to establish pleuritis and serous effusion. The general symptoms in typical cases are comprised in physical distress and mental anxiety, expressed in the countenance. The complexion is pale and dusky, and the lips more or less livid. The voice is weak; the skin moist or covered with a cold clammy perspiration; the pulse is quick and feeble; and the respiration is extremely hurried (FULLER, l. c., p. 204). The *physical signs* in typical cases are:—*Vocal fremitus,* diminished or altogether annihilated. The intercostal spaces are more than usually elastic or resilient; and the heart may be more or less displaced. The side may be enlarged to an extreme degree, with increased width and bulging of the intercostal spaces. *Percussion* elicits a clear tympanitic resonance, which sometimes changes its character, and becomes amphoric and of a metallic quality over the trachea and larger bronchi. The area of clear resonance on percussion may extend considerably beyond its normal limits, as the mediastinum, the heart, and the diaphragm are more or less displaced. *Auscultation* furnishes different results, according to the amount of the effused air. If the quantity of air be small, the respiration-sounds are weak and distant, and the vocal resonance is weak; if it be great, so that the lung is thoroughly compressed, the respiratory sounds and the vocal resonance are almost or altogether absent, except in the interscapular region, at the root of the larger bronchi, where diffused blowing respiration and diffused but loud vocal resonance may still be audible (FULLER, l. c., p. 204). Certain signs, resulting from the co-existence of air and fluid in the same cavity (which are not met with either in *pleurisy* or *pneumothorax* when they exist independently of each other) occur with the combined condition named *hydro-pneumothorax*, and are characteristic. These are,—(1.) *Fluctuation*, which is felt by the patient as well as by the observer, when the patient's body is abruptly jerked or shaken. (2.) A ringing, splashing sound—the *succussion sound* of Hippocrates—heard under the same circumstances. (3.) A remarkable *metallic tinkling*, which sometimes accompanies succussion of the patient, but which may accompany cough or inspiration, or a sudden change in the patient's posture (FULLER, l. c., p. 205).

Diagnosis.—Should the pleurisy be chronic, it is impossible to distinguish between it and hydrothorax, except by the previous history. The diagnosis, also, between *hydrothorax* and *œdema of the lung* is not always easy. Let a patient have an acute pleurisy, and if, after the urgent symptoms have passed, there still remains localised dulness, with absence of breath sound, and perhaps distant tubular breathing in one part of the chest, some fluid may be considered to be left, and this is pus. If the case has been one of pleuro-pneumonia, and there result after the acute attack severe physical signs, then also an *empyema* is present. Even if it have been thought, from the nature of the attack, that the inflammation was confined to the lung alone, yet after a time, when all other symptoms have ceased, if there remains a localised dulness, with absence of lung breath-sounds, it implies that a pleurisy had acccompanied or succeeded the pneumonia, and as a result a *local empyema* is present (WILKS, *Brit. Med. Journ.*, p. 929, June 21, 1879). It is often doubtful whether the pain is due to pleurisy, to rheumatism, to neuralgia, or to shingles. The absence of pain distinguishes hydrothorax from acute pleurisy.

Prognosis.—Some cases of *hydrothorax* recover, but the prognosis is in all cases extremely grave and doubtful. *Simple idiopathic pleurisy* on one side of the chest, occurring in a person whose lungs are not chronically diseased, almost always terminates favourably, if the effusion has not been copious, if observed in time and treated judiciously; but when it occurs as a complication in other diseases, the result may be doubtful, especially if air finds its way into the cavity (*pneumothorax*). When *pneumothorax* affects the whole, or nearly the whole, of one side in tubercular disease, it is usually fatal after periods varying from minutes to weeks. *Limited* pneumothorax is less fatal, in which the lung gives way by a mere *pin-hole perforation.* Pleurisy, in relation to pneumothorax, is not to be regarded as a fatal complication, but as a healing power, the formation of adhesions often anticipating perforation of the pulmonary pleura, limiting the escape of air to a part only of the cavity, and the pin-hole opening is sealed up (GAIRDNER, l. c., p. 396). Prognosis is most unfavourable in perforation from disease in the tissue of the lung, contrasted with the more favourable prognosis in traumatic cases, in which the chest walls are wounded, but in which the lung is not wounded. Those cases, *cæteris paribus,* are most apt to run an untoward course where there are great accumulation of air and great displacement of the thoracic organs (FULLER). In cases where the lung has been compressed for a long period, it will not again expand after the fluid has been absorbed; and the space which the lung occupied becomes filled up by the falling in of the surrounding parts, the ribs being retracted and approximated. The shoulder is lowered, the spine bends, usually to the diseased side, and all the diameters (especially the antero-posterior) of the chest are diminished.

Treatment.—In acute pleurisy venesection can be dispensed with. It neither cuts short the malady nor prevents effusion. It may only be adopted in cases of severe *dyspnœa*, arising from collateral hyperæmia, if the compressed part of the lung is attended with signs of œdema. At the commencement of less severe cases the use of cold compresses, and of local blood-letting by leeches or cupping, may be repeated in the course of a day or two, until relief becomes permanent; but the local blood-letting is only to be had recourse to if the cold compresses do not relieve the pain and the dyspnœa within two or three hours. *Large hot poultices* of *linseed* with *mustard* should be always employed when local pain on inspiration is present, of a "catching" or "stabbing" nature. If the pain and dyspnœa are not relieved within two or three hours, the method originally recommended by Dr. Frederick T. Roberts, in his *Handbook of Medicine* (page 508), of *mechanically fixing the side affected, so as to prevent its movements* as much as possible, ought to be at once adopted. "Strips of properly adherent plaster, spread on a firm material, are cut about four inches wide, and sufficiently long to extend round the side from mid-spine to mid-sternum, or a little beyond. These are laid over a variable extent of the chest, according to the severity of the case, it being sometimes requisite to include the whole side—*beginning below.* The patient is directed to EX-*pire* deeply, and at this time a strip of plaster is fixed at mid-spine, and drawn tightly round the side *in the direction of the ribs;* then another strip is laid over this, *but across the course of the ribs;* the third follows *the direction of the first, overlapping about half its width;* the fourth that of the second, and so on in alternate directions. A strip may also be passed over the shoulder, and kept down by another fixed round the side across its ends." *Tartar emetic* subdues the inflammatory tendency. Its use must be abandoned as soon as the acute symptoms have passed away. *Digitalis,* rather than *tartar-emetic,* may be given in recent cases in the form of *infusion.* In the more latent cases *one grain* of the *powder of the leaves* may be combined with a grain of *quinine. Blisters* are not to be used until the acute stage is past. When pain has ceased for some days, and absorption of the fluid proceeds slowly, and the disease promises to become chronic, a succession of blisters may be of use, applied over the lateral region of the chest, *but not over the seat of pain.* Diuretics ought at the same time to be freely given. The *compound tincture of iodine,* in doses of *twenty minims freely diluted,* is a valuable medicine at this juncture; and so also is the *liquor iodi of the British Pharmacopœia of* 1867. After free evacuation of the bowels has been effected, *calomel,* to the extent of producing the *slightest* mercurialisation, is the most beneficial line of treatment, *to be adopted in certain cases only*—namely, *in otherwise healthy persons in whom the pleurisy is not the result of a constitutional disease.* The more rapidly slight mercurialisation can be produced the better. During the first six hours, small doses of *calomel with opium* (*a grain and a half of the former,* combined with a *sixth of a grain of the latter,* or more, if the pain continues acute) should be given every half hour, while mercurial ointment is rubbed into the skin of the affected side near the axilla every fourth hour (WALSHE); or *half grain* or *grain doses* of *opium,* every *three* or *four* hours, in combination with *one or two grains of calomel, and half a grain of digitalis,* may be given, *and the whole side may be covered with a piece of linen spread with mercurial ointment:* over this is placed a poultice covered with oiled silk (FULLER, l. c., p. 184). The patient must be carefully watched, so that neither *ptyalism* nor *narcotism* is produced. The moment mercurial action has been established, the further administration of the mineral must cease. *Opium* and *digitalis* are to be continued after the use of *calomel* has been suspended; and with them may be given two grains of *squills* or of *nitrate of potash*, as a diuretic; or after twelve or more hours very small quantities of *tartar emetic* in solution may be given at night, combined with small doses of *opium* and *ipecacuanha*, to allay general irritation.

In the chronic stage of pleurisy there is a necessity for generous diet and tonic remedies, such as *quinine*, in aid of any diuretics and absorbents which may be employed. If the patient be kept too low after the stage of active inflammation has subsided, or be unduly depressed, it will not only be impossible to induce absorption, but there will be great danger of the fluid becoming sero-purulent. When a succession of blisters has failed in relieving the patient, *ioduretted lotions*, or *ioduretted ointments*, combined with the internal administration of *cinchona* with *tincture of iodine*, *iodide of potassium*, and small doses of *bichloride of mercury*, *nitre*, *acetate of potash*, *squills*, *digitalis*, *and cantharides*, have each in their turn effected the desired object (FULLER, l. c., p. 186). The fluid has been gradually reabsorbed, and recovery has ensued by a combination of *iodine* remedies, taken internally, and applied externally—*e.g.*, *syrup of the iodide of iron* (ʒii.), with *simple syrup* (℥ii.), and a teaspoonful taken every two hours, in conjunction with the external application of a weak solution of *iodine* (*iodine*, ʒss.; *potass. iodidi*, ʒii.; aq. distil., ℥ii.) upon the affected side of the chest (NIEMEYER). Other formulæ most useful in such cases are—(1.) A diuretic in the form of a pill, composed of *digitalis*, *squills*, and the mass of *pill. hydrargyri*, of each a grain and a half. *Nitrate of potash* (twenty grains), combined with *tincture of the perchloride of iron* (fifteen minims), three times a day, may at the same time be given (FULLER, CHAMBERS). Professor W. T. Gairdner recommends as a diuretic the *cream of tartar* electuary, in which the *cream of tartar* is mixed in equal proportions with *treacle*, *honey*, *marmalade*, or *syrup of ginger*, and in some cases flavoured with a few drops of *peppermint oil*. The dose is a teaspoonful, repeated as often as the stomach will bear it, or as the urgency of the case demands. *Jaborandi*, in the form of infusion, has recently been advocated in the treatment of chronic pleurisy with serous effusion. (2.) A lotion, to be applied over the chest by *spongeopiline*, or by *lint* covered with *oiled silk*, composed of—*Bichloride of mercury*, gr. iv.; *tincture of iodine* (*compound*), ʒiv. to ʒvi.; *glycerine*, ℥iii.; and distilled water, ℥ivss. (3.) One or other of the following ointments may also be rubbed upon the skin, over the side of the chest—*Bichloride of mercury*, gr. iv. to vi.; *iodine ointment*, ʒiv. to ʒvi.; *lard*, or *vasaline*, ʒiv. to ℥vi.; or, *bichloride of mercury*, gr. iv. to v.; *iodine of potassium*, ʒii.; distilled water sufficient for solution; and *lard*, or *vasaline*, ℥i.; Ft. Ung. Feeding the patient upon the driest possible diet, and withholding water as much as possible, has been successful in some cases (SCHROTH).

As a means of relieving the chest in chronic pleurisy and empyema, the operation of puncturing the chest and letting out the fluid—an operation to which the name of *paracentesis* has been given—is a useful one; and the practical question is as to the time when it should be performed. The condition of the patient's health and respiration, and the absorption or non-absorption of the effused fluid, are the aids to a decision for or against its performance. As long as the breathing is not seriously embarrassed, and the general health does not decline, so long are we justified in making full trial of our remedies. But as soon as extreme shortness and distress of breathing, or lividity and anxiety of the countenance, denotes serious interference with the functions of life, delay is no longer justifiable, and it becomes our duty at once to give our patient the chance which the operation affords. In the hands of the American physician, Dr. Bowditch, and Professor W. T. Gairdner, of Glasgow, and Dr. Budd, of London, this operation has been the means of saving many lives. Its effect is to relieve the mind as well as the lungs of the patient from great oppression. Although before the operation he may be quite weak, he is often able after it to get up and walk. Digestion becomes at once improved, and strength is rapidly regained. The cough, however, is apt to augment during the first few days; the pulse retains its quickness; friction-sounds occasionally become developed, and several months may elapse before the vesicular murmur becomes properly re-established in the lung. Generally, the operation of *paracentesis thoracis* in simple pleurisy is not to be performed unless the life of the patient is in jeopardy—that is, in cases "in which the effusion continues and increases, and the side, instead of shrinking, enlarges; the functions of the lung on that side are entirely abolished; nay, the use of the remaining lung is greatly interfered with by the pushing over of the mediastinum; and the patient is in imminent danger of suffocation." In such cases the oppressed lung must be relieved by "*letting the fluid out*" (Sir THOMAS WATSON, *Lectures*, Fourth Edition, Vol. II., p. 128). "Life is plainly in jeopardy when the vital functions of the lungs or of the heart are greatly hindered; when symptoms present themselves of approaching death by *apnœa* or by *syncope*. If we discover no cause for those symptoms except the increasing pressure of liquid pent up in the pleura, we are warranted in ascribing them to such pressure, and bound to act upon that persuasion." Also, if death by *asthenia* appears inevitable, the patient losing ground from day to day, and when all other means of getting rid of the pent up fluid have failed, the patient should not be denied the *chance* which the operation affords. Also, "whenever the effused liquid consists of *pus*, it should be let out" (l. c., p. 130). The risks of allowing a localised empyema to remain are great (WILKS, l. c.) With the arrangements of Dr. Bowditch's syringe we are enabled to remove fluid *before* the *false membrane* thickens over the compressed lung, and so makes it difficult to expand again. The trocar and small canula take the place of an exploring needle. Therefore, if the lungs are not bound down by adhesion, or prevented from expanding by the thickness of the *false membrane* which covers it, Bowditch's syringe and stopcocks enable the fluid, even when purulent, to be withdrawn without air being admitted. Mr. Lister's antiseptic method and precautions as to opening an abscess ought to be adopted. But when air has been admitted, and when the fluid within the cavity of the chest has become changed thereby—is purulent and perhaps fetid—the evacuation, *under antiseptic precautions*, of the contents by "drainage" seems the most efficient remedy.

In Pneumothorax.—Puncturing the chest walls may give temporary relief, by allowing the air to escape; but the operation ought only to be done in cases where the dyspnœa is urgent, and the displacement of the viscera such as demands that relief which the operation is calculated to give. Otherwise, the treatment of pneumothorax is best conducted by small doses of *morphia—i.e., stimulant doses*—frequently repeated, combined or not with *ether* or *alcohol*, with a view to overcome collapse, relieve dyspnœa, and subdue pain. When reaction follows collapse, as indicated by heat of skin, strength and hardness of the pulse, soreness and pain of the affected side, local blood-letting by leeches, with saline aperients, may be used if the strength of the patient warrants such a line of treatment. *Turpentine* and *poppy fomentations*, followed by *blisters*, are also indicated if life is sufficiently prolonged after the primary inflammatory symptoms are subdued. The inhalation of small quantities of *chloroform* is useful. In other respects the treatment is similar to that already stated.

SECTION VI.—DISEASES OF THE RESPIRATORY SYSTEM NOT STRICTLY LOCAL.

HAY ASTHMA.

LATIN EQ., *Asthma ex Fœnisicio;* FRENCH EQ., *Fièvre de Foin;* GERMAN EQ., *Heu-Asthma;* ITALIAN EQ., *Asma dei Mietitori.*

Definition.—*A peculiar catarrhal affection occurring during the summer months, especially during the inflorescence of the grasses, or during the drying and conversion of the newly-mown grass into hay in May, June, and July.*

Pathology.—(*a.*) *Causation.*—There is usually a predisposition in a small number of susceptible persons (*idiosyncrasy*), and the exciting cause may be the inhalation of some palpably minute emanation from the inflorescence of the grasses. The powder of *ipecacuanha* will also produce the affection; so will fine *dust*, the composition of which is unknown, or at least is not known to contain anything so specific as *ipecacuanha*. The *dust* of grain or *flour* has been known to produce the same effect; and at one time mere effluvia or odours were believed so to affect the nervous system as to cause the occurrence of asthmatic fits. The odours of *mint*, of the *rose*, of *various flowers*, and other strong perfumes, have produced difficulty of breathing in some people, with dry cough, as in hay asthma. The mere mechanical influence of minute particles floating in the air may be sufficient in some to cause this disease; on the other hand, the nature of the particles may be *specific*, as in the case of *ipecacuanha*, or inflorescent particles of certain grasses, or of flowers cut down with the grass in the progress of haymaking.

Symptoms are those of a violent catarrh, attended with much sneezing and redness of the mucous membrane of the nose and eyelids.

Treatment.—Regarding it as the result of irritation (specific or mechanical) from fine particles of matter (indefinite dust or specific powder) floating in the air, the use of a respirator of *fine cotton*, or of *crape* or *cambric* in several folds, should prevent the affection, and ought to be tried by those who suffer every year about the months of May and June. In other respects the local catarrh must be treated as described under "Cold," "Coryza," and "Bronchitis" (pp. 703, 716, *ante*).

(SPASMODIC) ASTHMA, OR BRONCHIAL ASTHMA.

LATIN EQ., *Asthma;* FRENCH EQ., *Asthme;* GERMAN EQ., *Asthma;* ITALIAN EQ., *Asma.*

Definition.—*A disease which culminates in paroxysmal attacks of difficult breathing, of varying duration. The dyspnœa seems to be immediately dependent on more or less extensive contraction of the smaller bronchi, due to spasm of their circular fibres. The breathing is accompanied by a wheezing sound, a sense of constriction in the thorax, great anxietas, and a difficult cough. The attack usually terminates by the expectoration of a quantity of mucus from the lungs, which varies considerably in appearance and in amount. In some instances the mucus is thick and heavy, in others it is light and frothy, whilst in the severer forms there may be only a few dark pellets coughed up before relief is obtained* (PRIDHAM). *In the hours immediately succeeding the fit, a remarkable diminution of the urea and chloride of sodium may occur, which would imply a considerable arrest either of formation or elimination, probably the former* (RINGER, PARKES); *or to the starvation that is generally enforced at that time* (SALTER).

Pathology.—(*a.*) *Historical.*—Few diseases have been the subject of greater doubts and differences of opinion as to its nature than asthma. Not unfrequently it has been confounded with dyspnœa; and the terms *dyspnœa, asthma,* and *orthopnœa,* were formerly employed to designate different degrees of difficulty of breathing. Their signification must now be more precisely defined. *Dyspnœa* is a term which denotes difficulty of breathing generally, and may be due to various causes already noticed. The significance of *asthma* is defined above, and its pathology is about to be considered; while the term *orthopnœa* signifies that great difficulty of breathing in which the patient is incapable of respiring except in the erect posture.

(*b.*) *Causation.*—There is no period of life at which asthma may not make its appearance—from the earliest infancy to old age. A large number of patients commence to be asthmatic within the first ten years of life, the numbers affected increasing from twenty to fifty years of age (SALTER). Many of the best marked cases date from early infancy—so early that it were difficult to say the disease was not congenital. It often depends upon hereditary transmission and conformation—nine out of ten cases. The melancholic temperaments, the sanguineo-melancholic, the nervous, and the irritable, are most liable to the affection; and the male sex is much more disposed to it than the female (WOOD). About 80 per cent. are men (PRIDHAM).

There are good reasons for regarding asthma as a general or constitutional disease; and it is believed by not a few to be connected with the gouty or rheumatic diathesis. The disposition of the attacks to recur at distant but gradually diminishing intervals; the division of each attack into nightly paroxysms, with marked remissions during the day; the duration of the earliest fits for several days or a week, are circumstances which point to the constitutional nature of the affection. Many asthmatics inherit gout; and in some instances both diseases can be traced in the family. Where gout and asthma have prevailed in previous generations there may be alternate attacks of asthma and of gout. When women are the subjects of asthma, gout prevails in their families in a larger proportion than in men (Pridham).

The dyspnœa of the asthmatic paroxysm is due to spasmodic contraction of the bronchial tubes; and the phenomena of the paroxysm are in a great measure due to reflex action (Hyde Salter). But the irritation may be also *centric,* as during hysteria (*emotional asthma*); or *direct,* as in the case of hay asthma. The exciting causes of the paroxysms are mainly due to fatigue and physical exhaustion—sudden or violent mental emotion—certain conditions of the digestive organs—gastric irritation—the irritation of a loaded rectum—irritation of an eruption on the skin and its sudden subsidence—the irritation of certain substances and articles of food, such as cheese, nuts, almonds, raisins, and sweet things generally, salted meats, condiments, preserved and highly-seasoned foods, fermented liquors, especially malt liquors, and sweet wines.

When the powers of digestion are insufficient to assimilate the food taken, and wherever the disease has occurred in any member of a family for one, two, or even three generations back; such exciting causes as imprudence in eating or drinking, an attack of bronchitis or influenza, atmospheric influences, certain odours, mental excitement, and the like, may at any time or period of life bring about spasmodic asthma; otherwise such exciting causes have no influence upon its development.

When once expressed, it seldom fails of recurring, but the intervals between the paroxysms are of uncertain duration. The fits may return periodically every ten days or a fortnight; and in more severe cases they will recur every night or early morning, at exactly the same hour. It is apt to recur in the spring and autumn, and after exposure to cold and wet. It is not only paroxysmal, but often periodic—by days, weeks, months, or even years. Diurnal asthma is very common, especially when associated with chronic bronchitis, heart disease, and impaired digestion. Asthma occurring once a year is usually a winter affection complicated with bronchitis. It has been observed to recur in females just after the menstrual discharge, or immediately before it.

Symptoms and Forms of Asthma.—(1.) In which the lungs seem to be mainly concerned, the source of irritation being applied to them, as some material respired which provokes the bronchial tubes to spasms by direct contact with their mucous surface (*e.g.*, asthma from fog, smoke, fumes of various kinds, *ipecacuanha powder*, or that of hay; from animal emanations; from certain atmospheres; and, lastly, from blood-poisoning, as after faulty digestion of beer, wine, or sweets). (2.) In which there is a reflex source:—(*a.*) When asthma follows an error in diet, or supervenes on a full meal; (*b.*) when the source of irritation is transmitted from a loaded rectum or from uterine irritation; (*c.*) when it arises from the sudden application of cold; (*d.*) periodic asthma. (3.) Complicating bronchitis, bronchial irritation, heart disease, or pulmonary emphysema.

In a patient labouring under a severe paroxysm, the upper part of the chest seems motionless, while the inferior portions are acting within a very confined range. The muscles passing from the head to the shoulders, clavicles, and ribs are rigid. The abdominal muscles act powerfully to increase the capacity of the chest, so that its walls are kept fixed in a condition of extreme inspiration. The chest is enlarged in every way, the diaphragm descends, the abdomen seems fuller, and its girth is increased. The whole of the lungs, but particularly the posterior parts, are labouring with a loud and deep sibilous, sonorous wheeze, accompanied with a mucous rattle. No respiratory murmur exists. Dry tube sounds alone are heard—rhonchus and sibilus of every variety, note, and pitch. There is complete stagnation of air in the chest. The sounds are so small, so general, that they indicate universal spasmodic constriction and their diffusion over the smaller tubes. But those spasms constantly change their place, disappearing in one and making their appearance in another, as indicated by the sounds continually changing their character and their site. Percussion indicates distension with air; and should an air-cell have burst, a rubbing sound will denote the effusion of air into the cellular substance of the lung. As the fit subsides, respiration becomes puerile, and by degrees the breathing returns to its usual state. In fatal cases the respiration becomes tracheal, and slight hæmorrhage may take place. The duration of the fit varies from a few minutes to two or three hours, or the whole night; or for three or four days, or many weeks.

The stomach in asthmatics is apt to be greatly distended after eating, for asthmatic patients are generally large feeders. The tongue becomes coated, and there is a tendency to fissures in it; the eyes are red and prominent. Emaciation generally progresses, and there is inability to rest horizontally in bed, or to walk up a hill. The secretions from the bowels are more or less abnormal, and the urine shows a variable deposit. In severe cases, when remedial measures are not taken to subdue the constitutional affection, the nights may be passed in great distress. Unable to lie down in bed, the paroxysms may be so severe that the patient almost anticipates death before morning—were it not that he becomes accustomed to the severe nature of the symptoms—till a copious heavy expectoration is with difficulty thrown off from the lungs. As the day advances he becomes somewhat relieved, although still in great discomfort. In some difficulty of breathing is constant, and always worst after a meal.

Premonitory phenomena may indicate the approach of a paroxysm; but they are liable to great variation. Some suffer from fearful headaches, or the heart labours with so much palpitation, and such irregularity of action, that rupture of a blood-vessel seems imminent. Eruptions on the skin sometimes lessen, and even disappear. There may be some warning during the night of the approach of an attack by huskiness o the throat; and during the middle of the night, or towards early morning, the patient is awakened by an oppression which renders it impossible for him to lie down again. In a short time the paroxysm gains strength breathing is effected by jerks, each aspiration being accompanied by a spasmodic effort, as if it would burst open the chest. The contractions o the muscles in the neck and below the ribs in front of the chest are great and often most painful. These attacks are not of equal violence at a times. The majority of asthmatics also know that an attack is comin on by certain feelings in themselves, or by certain conditions of the system, of which they are aware for several days before. Extreme drowsiness and sleepiness may thus indicate the approach of a paroxysm. Itchin under the chin is another common symptom of an approaching paroxysm

This itching is incessant, of an indefinite creeping character, and scratching does not relieve it. It may extend over the sternum and between the shoulders. Such precursory phenomena must be looked upon as an integral part of the paroxysm. Extreme wakefulness, unusual mental activity, and buoyancy of spirits constitute one set; loss of appetite, flatulence, costiveness, and certain peculiar uneasy sensations in the epigastrium constitute another set of premonitory phenomena (HYDE SALTER).

The time at which a paroxysm commences is almost invariably in the early morning, from three to six o'clock. In some the usual time is the evening, just after getting into bed, and before going to sleep. Profuse *diuresis* may attend the first stage. The urine is a pale limpid water, like the urine of hysteria (*nervous urine*), and generally lasts for the first three or four hours, when it ceases. *Neuralgic* pain is another early symptom, in the form of deep-seated, aching, constant, and wearying pains in the limbs, joints, or testicles. The characteristic wheezing may commence while the patient is yet asleep; and as the difficulty of breathing increases, he gradually or partially awakes, and sits up in bed "in a miserable half-consciousness of his condition." A temporary abatement occurs, and sleep may again overpower him, to be again awoke, and again to sit up. The *dyspnœa* increases, so that he can lie back no more. He throws himself forwards, plants his elbows on his knees, and, with fixed head and elevated shoulders, labours for breath like a dying man. The appearance of an asthmatic person is very characteristic. The countenance may bear the signs of distress. His back is rounded, his shoulders are elevated, and his gait is stooping. During a fit his chest, back, shoulders, and head are fixed; and, when he looks from object to object, he turns his eyes like a person with a stiff neck. His shoulders are raised almost to his ears, and his head thrown back and buried between them. The better to raise his shoulders, and to spare muscular effort, he fixes his elbows on the arms of a chair; or he plants his hands on his knees; or he leans forward on a table; or sits across a chair, and leans over the back of it; or he stands grasping the back of a chair, and throwing his weight upon it. In this attitude a patient has been known to stand for two days and nights unable to move (SALTER). Sometimes the patient may lean against a piece of furniture sufficiently high to rest his elbows upon in a standing position. At every breath the head is thrown back, the shoulders still more raised, and the mouth a little opened, with a gasping movement. The expression is anxious and distressing. The eyes are wide opened, strained, turgid, and suffused. The face is pallid; and if dyspnœa is extreme and prolonged, it becomes slightly cyanotic. The labour of breathing is so great that beads of perspiration stand on the forehead, or even run down in drops upon the face. If the bronchial spasm is protracted and intense, the body-temperature falls. The extremities especially get cold, blue, and shrunken. This union of coldness and sweat, combined with the duskiness and pallor of the skin, gives to the asthmatic so much the appearance of a dying man that even the initiated may fear that death is impending. The pulse during the paroxysm is always small and feeble in proportion to the intensity of the dyspnœa, and immediately the paroxysm yields the pulse begins to resume its normal volume.

Diagnosis.—It may not easily be confounded with any disease of the chest or larynx, if auscultation and percussion are attended to, with a due regard to the history of the case. It is only when asthma complicates other diseases that its diagnosis may be obscure and its treatment uncertain. It has been confounded with—(1.) Spasmodic affections of the larynx; (2.) severe cases of sudden and *acute bronchitis;* (3.) *angina pectoris;* (4.) *hydrothorax;* and it may be associated with the development of lesions of the heart and great vessels, and ultimately leads to them.

The character of the *dyspnœa in asthma* is peculiar. It is unlike the dyspnœa of heart disease, or of that of bronchitis, or of that of emphysema. The distinctive features of each are,—(1.) *Heart dyspnœa* is intolerant of the slightest exertion, or of the recumbent position; and sitting up or stillness may cure for the time the most violent paroxysm; the breathing, too, of heart dyspnœa has a panting and gasping character, and not the wheezing labouring character of asthma. (2.) *Bronchitic dyspnœa* is short, crepitous, and accompanied with cough. (3.) The *dyspnœa of spasmodic asthma* is of such a nature as to shut off the air-supply, and it gives the most positive evidence of narrowing of the air-passages. The wheezing, or shrill sibilant whistle, ever changing its place, is positive evidence of bronchial contraction. There is an absence of cough, and the sounds are dry. (4.) The *dyspnœa of emphysema* is abiding, varies but little, and has no wheeze. It may complicate each of these conditions.

Prognosis is favourable so far as a fatal issue. Death during a paroxysm is a rare event. It never occurs unless the asthma is complicated with some fatal disease. The patient may have the control of the disease in his own hands to a great extent.

Treatment comprises, (1.) What should be done during the fit; and, (2.) during the intervals. When a patient is labouring under a fit of either form of asthma, to tranquillise his suffering and shorten the attack is the aim of treatment. Any exciting cause must be removed. An undigested meal, or constipation, must be got rid of—by an emetic in the one case, or by an enema in the other. The patient should have a strictly tonic regimen; and *camphor mixture,* to the extent of about an ounce and a half, combined with a drachm of the *spirit of nitrous ether* and some *morphia,* may be given every hour, or every two hours, for a short time. Some milder narcotic may be substituted for morphia, as *tincture of hyoscyamus* to the extent of about fifteen drops for each dose. Or *asafœtida, castor* *musk,* or *hydrocyanic acid,* to the extent of ♏iij. every six hours, may b substituted. If the fit should occur after a hearty meal, and after a emetic has been given, the *tincture of rhubarb,* or the *sulphate of magnesi* should be continued in repeated small doses. If the attack be long, *arrow* *root* or *sago,* with small quantities of wine or brandy, should be given t support the patient under his exhausting sufferings. *Ipecacuanha, tart* *emetic,* and *tobacco* are the drugs which most rapidly relax spasm as dire depressants. There is, however, great danger in the use of the latter, fro unmanageable and dangerous collapse. Tobacco ought never to be indulg in by the asthmatic, except as an agent in the cure of his disease. Th only can he look to it for relief, and it should be smoked from a pi *Ipecacuanha* ought to be given in a dose of twenty grains at the onset the paroxysm. The feelings of the sufferer should be consulted as to t temperature to which he should be exposed during the paroxysm. Wh there is any organic lesion the fresh air is grateful and reviving. On the contrary, when the paroxysm is purely a spasm of the bronchial tubes, warmth, by relaxing spasm at their ultimate divisions, is generally more useful than cold. Patients learn by experience what will give them most relief. *Strong coffee, spirits and water, ice* rapidly swallowed, may suit individual cases.

The treatment during the interval is all-important. Few cases will be found of true spasmodic asthma which are not entirely under the control of well-regulated dietetic management. By many physicians a rule of life as to diet is the only certain treatment of asthma. Extremely strict dietetic treatment and sedatives during the intervals of the paroxysms must be prescribed. A plan of treatment somewhat as follows may be laid down, after that suggested by Mr. Pridham of Bideford and Dr. Hyde Salter of London :—The secretions from the bowels are first to be corrected by *Pilulæ Aloes cum Myrrhâ*, gr. iii.; *Pilulæ Hydrargyri*, gr. i.; *Extracti Taraxaci*, gr. ii.; *Extracti Strammonii*, gr. ss., made into two pills, to be taken at bedtime, followed by a saline aperient in the morning. Or giving, every alternate night, *Pil. Hyd.*, gr. iv.; *Pulv. Ipecac.*, gr. i., in the form of a pill. And on the following morning, *Mist. Sennæ comp.*, ℥i.; *Bicarbonatis Magnesiæ*, gr. x.; *Bicarbonatis Sodæ*, gr. viii.; and during the day small doses of compound rhubarb powder. After thus attending to the general secretions for about ten days, a strict diet is to be commenced, which must be regularly weighed out and adhered to, the hours of meals being fixed. (1.) *Breakfast* at *eight* A.M., to consist of half a pint of green tea or coffee, with a little cream, and two ounces of dry stale bread. Dr. Salter allows an egg *or* (*not and*) a mutton chop, or some cold chicken or game. Tea is better than coffee, and milk and water better than either. (2.) *Dinner* at *one* P.M., to consist of two ounces of fresh mutton, without fat or skin, and two ounces of dry stale bread or well-boiled rice. Beef and lamb should be rarely eaten, pork or veal never. There should be no cheese and no dessert (SALTER). Three hours *after* dinner (not sooner) half a pint of weak brandy and water, or whisky and water, or dry sherry and water, may be taken, or toast-water *ad libitum*. Water is the best fluid to drink. (3.) *Supper* at *seven* P.M., to consist of two ounces of meat as before, with two ounces of dry stale bread. The patient is not to be allowed to drink any fluid whatever within one hour *before* his dinner or supper, and not until three hours after either of these meals. At other times he is not limited as to drinks, otherwise than that all malt liquors are to be prohibited. *Soda* or *seltzer*-water may be indulged in when thirsty. With this dietetic treatment, sedatives, such as three grains of the *Extract of conium*, are to be taken four times a day—namely, at the hours of seven, twelve, five, and ten,—the dose to be gradually increased to five grains four times a day. To each of these pills a *fourth* of a grain of the *Extract of Indian hemp* may be added, which may be gradually increased to one grain in each dose. Under this treatment in a few days distressing symptoms subside; and after the regimen has been strictly persevered in for at least a month, two ounces more of meat may be permitted, if digestion is sufficient. The stools must be repeatedly seen by the physician; and the stomach must not have more to do than it can accomplish. The powers of digestion are known to be recovering when there is a craving for food as the hour of nourishment arrives. If flesh is gained, strength improves; and while the tongue cleans, the appetite improves, the distension of the stomach lessens, and the powers of digestion recover. The patient ought to be able to sleep six or seven hours at a time, and to lie in bed all night. If these results follow, the ultimate cure of the disease may be looked for; but it may at the same time be taken for granted that the asthmatic can never with impunity eat and drink as other people. It is only by the exercise of self-denial that the patient has it in his own power to live a life of comparative ease and comfort. Many patients not possessed of such resolution, self-denial, and strength of mind, will say such dieting does not suit their constitution, and that they cannot or will not persevere; but no trial of the remedy can be considered sufficient which does not embrace a period of at least six months, the physician taking care to ascertain the weight of the patient, his age and height, before commencing any treatment. It is difficult to persuade many people to live so abstemiously. Many cannot control their appetite; or they believe that in so limiting themselves in regard to diet they will injure their constitution. It is necessary to subdue by sedatives other than opiates the abnormal ravenousness of appetite. Asthmatics are generally dyspeptics; and the most simple rule regarding diet is:—Let no food be taken after such a time in the day as will allow digestion being completed or the stomach empty before going to bed. The time when the last solid food should be taken will depend upon what the bedtime is. If ten, or half-past, then three or four should be the dinner hour, after which no more solid food should be eaten.

Open air exercise must be freely taken, but not within three hours after eating animal food, and the exercise must be always short of fatigue. The greatest punctuality is necessary as regards the taking of food, of exercise, and of medicine; and the bowels should act immediately after breakfast, either naturally or by enema. Success depends on the regularity with which all the functions of the body can be performed; and care should be taken to rest body and mind, at least, for one hour after food. Smoking *stramonium*, the *inhalation of chloroform* or of *ether* or both, although they appear to soothe and mitigate the paroxysm in some cases, yet they do not appear to *shorten* the attack in any. The inhalation of *nitrite of amyl* has been recently recommended. The fumes of *stramonium* are to be collected in an inverted glass bowl with a narrow mouth. The bowl being charged to its full, is placed under the mouth of the patient, who is directed to inhale, to the fullest extent in his power, the smoke which has been collected in the bowl. Or the *stramonium* may be smoked like tobacco, "then puff the smoke into a tumbler, and inhale it cold into the lungs" (Bullar). Or it may be smoked as an Oriental smokes the hookah, in which the smoke is purified by being passed through water. Cigars and cigarettes of *stramonium* are now sold by the chemists. The fumes of brown paper saturated with a solution of *nitrate of potash* sometimes also relieve the spasms. *Indian hemp*, in doses of from two to four grains of the extract, or thirty minims of the tincture will often relieve the spasm for the time being, but may fail ever after (See Watson, *Lectures on the Practice of Physic*, Vol. II.)

Only one remedy—*jaborandi*, and its alkaloid *pilocarpin*—has been found not only to remove the urgent symptoms of asthma, but to exercise, at the same time, a favourable influence on the disease itself. It produces a powerful revolution in the distribution of the blood, by attracting a large volume to the skin and salivary glands; and by diminishing its volume through copious perspiration and salivation, congested internal organs are relieved in a proportional degree. Dr. Berkhart has found the alkaloid preferable to *jaborandi*, because—(1.) It acts more rapidly; (2.) it does not produce strangury; (3.) the dose can be more accurately regulated. In cases where the cardiac muscles are in a state of fatty degeneration, the influence of pilocarpin may induce the most alarming symptoms; which soon spontaneously subside, the heart regaining its previous force. But if it lingered to do so, a subcutaneous injection of $\frac{1}{120}$ or $\frac{1}{60}$ grain of atropine, or one drop of *liquor atropiæ* (B. P.), immediately restores the balance. *Pilocarpin, or muriate of pilocarpin,* is more suitable in the treatment of the younger asthmatics, but is by no means contra-indicated in patients of more advanced age. The dose should not exceed *one-third of a grain.* Dr. Berkhart has never used more than *ten drops* of *a* 2 per cent. *solution,* injected under the skin of either arm. During the action of the drug, the patient should preserve the recumbent posture—which the almost immediate relief obtained will enable him to do—and he should be carefully watched until the effect has passed off. It is desirable to have a solution of *atropine* or *liquor atropiæ* always at hand, in case of need. It is well, also, not to use pilocarpin soon after the patient's meals; if the dyspnœal seizure occur under these circumstances, there are, it is needless to say, other remedies more suitable (*Brit. Med. Journal*, June 19, 1880). Similar results may be obtained in less urgent cases by the internal administration of the powder of the leaves, in doses of 30 to 90 grains, infused in boiling water—the water and grounds being swallowed together.

CHAPTER XIX.

DISEASES OF THE DIGESTIVE SYSTEM AND PARTS CONNECTED WITH IT.

Section I.—Diseases of the Mouth.

STOMATITIS.

Latin Eq., *Stomatitis;* French Eq., *Stomatite;* German Eq., *Stomatitis*—Syn., *Entzündung der Mundschleimhaut;* Italian Eq., *Stomatitide.*

Definition.—*Inflammation of the mouth.*

Pathology.—*The mouth,* especially in children, is liable to various forms of inflammation, which have received the generic name of "stomatitis." The inflammation may be either, (*a.*) *simple,* (*b.*) *ulcerative,* or (*c.*) *vesicular.*

(*a.*) *Simple erythematous inflammation* occurs in patches, and may be due either to hot or acrid substances taken into the mouth, to cold, or to the irritation of the teeth, especially when they have tartar upon them. It may be also due to gastric derangement, in which case small doses of *magnesia*, or of *rhubarb with soda*, will generally restore the stomach to a healthy condition, and the stomatitis will speedily subside. (*b.*) *Ulcerative stomatitis,* in its milder form, is also known by the name of *noma;* but there are different grades of the ulcerative lesions, from *noma* up to *cancrum oris.* The milder form, *noma,* generally commences at the edges of the gums opposite the incisors of the lower jaw, where the gums become white and spongy, and separate from the teeth, as if mercury had produced its specific effects. Ulceration begins and extends along the gums until the jaws are implicated; and as the disease advances the cheeks and lips begin to swell, becoming tense and indurated. The teeth may fall out; and the gums, assuming a gangrenous condition, the breath becomes intolerably fetid. There are generally enlargement and tenderness of the submaxillary glands. (*c.*) *Thrush*—Syn., *Aphtha, vesicular stomatitis,* usually commences as a simple stomatitis; but very soon small, round, transparent, greyish or white vesicles appear, the base of each being surrounded by an elevated marginal ring, which is pale and firm. Fluid soon escapes from the ruptured vesicle; an ulcer forms, which spreads, bounded by a red circle and an elevated border. These ulcers are sometimes covered by a pultaceous layer, mainly formed of the secretion of the mouth, which is greatly altered and increased. The disease chiefly attacks the new-born infant; but a similar condition may be seen in the adult towards the termination of long wasting diseases, and especially in phthisis. The disease also frequently occurs as a sequela of measles. The whole surface of the mouth exhibits unusual redness, with here and there cord-like exudation in irregular patches, preceded by vesicles, especially behind the lips and about the tip of the tongue. These patches are thrown off from time to time, and the mucous membrane below is then seen to be of a bright red colour; but they speedily form again. The adjacent glands are apt to become tumid and tender. The skin is commonly hot and dry; thirst is considerable; swallowing seems to give pain; and diarrhœa may ensue to a degree which soon proves fatal in an infant. In some forms of the affection microscopical parasitic plants occur—usually the *oidium albicans,* or so-called *thrush fungus.*

Treatment.—The mouth should be frequently washed with emollient fluids, such as *linseed infusion, diluted glycerine* and *biborate of soda,* or *honey* mixed with *biborate of soda.* The following lotion may be painted over the lips and mucous membrane of the mouth and tongue, namely:—*Sodæ Biboratis,* fl. dr.; *Glycerini,* fl. oz. ii.; *Aquæ Rosæ,* fl. oz. iv.; *misce* (TANNER). *Creasote, vinegar, carbolic acid, glycerine,* and *alcohol* are also recommended as local applications. The late Dr. Symonds, of Bristol, recommended combinations of *turpentine,* with *glycerine* and *alcohol;* equal parts of *turpentine* and *glycerine,* applied with a soft brush two or three times a day, being, in his opinion, a curative application, whether the lining of the mouth be red and puffed, or pale and œdematous, or studded with superficial ulcers, or opaque yellow accumulation of secretion or epithelium. He also recommended that *follicular ulcers* on the inside of the lips and cheeks and tip of the tongue should be treated by *sulphate of copper* applied once or twice a-day (*Brit. Med. Journal*, March 13, 1868). In severe cases, where the breath becomes fetid, with the submaxillary glands large and swollen, the lips and gums tumid, the face flushed and swollen, and the fever intense, *chlorate of potash* must be freely given internally, to the extent of *five* grains every four or six hours. Besides these remedies, the solution of the *pernitrate* of iron, internally, has a beneficial effect upon the sores, prescribed in the following formula:—*Liq. Ferri. Pernitratis*, gtt. xl.; *Syrup. Aurant*, fl. oz. ss.; *Aquæ*, fl. oz. vss.; *misce*. A fourth part may be given to a child three or four years of age four times a-day (DUNGLISON). In cases where parasitic vegetable productions abound (*parasitic thrush*), the application of a solution of *sulphite of soda* (fl. dr. i. to fl. oz. i. of water) removes the deposit in twenty-four hours (JENNER). The secretions of the mouth being acid, the salt is decomposed, and sulphurous acid is set free, which destroys the parasite. A change of air is often absolutely necessary to restore the patient to health; and *arsenic* and *iodine*, combined with good diet, are useful restorative agents in repairing the faulty nutrition of the child.

CANCRUM ORIS—*Syn.*, GANGRENOUS STOMATITIS.

LATIN EQ., *Gangræna Oris*—Idem valet., *Stomatitis Gangrænosa;* FRENCH EQ., *Gangréne de la Bouche;* GERMAN EQ., *Noma*—Syn., *Wasserkrebs;* ITALIAN EQ., *Cancro della Bocca.*

Definition.—*The more severe form of inflammation of the mouth.*

Pathology.—It occurs in children of debilitated habits, between *two* and *five* years of age especially. A copious flow of saliva, a tumid appearance of the cheek, with fetor of breath, ought to suggest an examination of the mouth, when small vesicles of a greyish-red or even black appearance, surrounded by a red base, may be seen on the inside of the lips or on the tumid cheek. As the disease advances, the swelling, surrounding hardness, heat, and pain increase. An ash-coloured eschar may then appear in the centre of the cheek, within the cavity of the mouth, surrounded by a glossy tumefaction of the parts; and on the outside of the cheek a hard indolent swelling. So rapid may be the spread of this destructive disease that in a few days the lips, cheeks, tonsils, palate, tongue, and even half the face may become gangrenous—the teeth falling from their sockets, while horribly fetid saliva and fluid flow from the parts.

Treatment.—The disease is analogous to hospital gangrene, and an early recognition of its nature will suggest an efficient application of *strong nitric acid* to the slough. The mouth must be frequently syringed with a solution of carbolic acid, in the proportion of half a drachm dissolved in a gallon of boiling water, and allowed to become warm or tepid.

Tonics and antiseptics must be freely given. Beef-tea, wine, brandy, quinine, and chlorate of potash are also indicated.

SECTION II.—DISEASES OF THE TONGUE.

GLOSSITIS.

LATIN EQ., *Glossitis;* FRENCH EQ., *Glossite;* GERMAN EQ., *Zungenentzündung;* ITALIAN EQ., *Glossitide.*

Definition.—*Inflammation of the tongue.*

Pathology and Causes.—The tongue is liable to various forms of inflammation of its covering and of its substance generally, resulting in various forms of ulcers. The inflammation may occur spontaneously, or from causes which give rise to idiopathic inflammation of other organs; or it may result from acrid substances taken into the mouth from the stings of insects, such as the bee and wasp; or from the specific action of mercury, of scarlet fever, of small-pox.

Symptoms.—There is generally great tumefaction from infiltration of serum, while fever, mental depression, and general weakness prevail, with pain and heat of the tongue, the colour of which is of a deeper red than usual. The swelling may be so great as to cause the tongue to project beyond the teeth, or even to be thrust so far back as to cause dyspnœa. Dr. Graves relates a case of inflammation affecting one-half the tongue, the median line forming the boundary between the swollen and the healthy parts.

Treatment.—Active cathartics are generally of great service, and they are to be given as *enemata.* Blood must be taken directly from the tongue in such cases. Incisions along the superior surface of the inflamed organ, followed by the action of the vapour of hot water, may reduce the swelling and relieve congestion. The relief afforded by these measures is frequently almost instantaneous. Two or three applications of six leeches at a time to the inflamed parts may produce a speedy decrease of the tumor (GRAVES). If suffocation is imminent *tracheotomy* or *laryngotomy* must be performed. *Erythematous inflammation* of the tongue will generally yield to a combination of *bismuth* in *glycerine,* such as *bismuthi subnitratis,* gr. xx.; *glycerini,* ℥i.; *aquæ,* ℥vii. Of these make a lotion with which to wash the mouth and tongue.

Morbid sensibility of the tongue may be soothed by a weak solution of *bromide of potassium,* ʒss. ʒi. to ℥vi. of water, used as a mouth-wash (SYMONDS).

ULCER OF THE TONGUE.

LATIN EQ., *Ulcus;* FRENCH EQ., *Ulcére;* GERMAN EQ., *Geschwür;* ITALIAN EQ., *Ulcera.*

Definition.—*Inflammation, simple or specific, terminating in ulcers.*

Pathology.—Chronic ulceration of the tongue is the most frequen affection of this organ. It may occur as—(*a.*) *Simple ulceration of th tongue,* in most instances caused by derangement of the digestive systen In young subjects the aphthous patches already described may be notice In adult patients such ulcerations will generally be observed about the ti or edges. The remainder of the tongue will be furred, and there will be the general symptoms of dyspepsia. This simple ulceration may pass off under the use of suitable constitutional and dietetic remedies; but it may be also very obstinate, and resist every effort at local or general treatment. (*b.*) *Syphilitic ulceration* may be primary, secondary, or tertiary. When primary disease of the tongue occurs, it must be treated in the same manner as a primary chancre in other situations. Secondary ulceration of the tongue is one of the earliest sequelæ of syphilis. There are two forms in which it appears:—(1.) As a simple denudation of epithelium, giving rise to a glossy psoriasis-like patch, which creeps from place to place, and which heals and breaks out again repeatedly. Accompanying it there are frequently similar patches on the mucous membrane lining the cheeks, and on the inner surface of the lips. (2.) Condylomatous patches on the tongue are not at all uncommon in secondary syphilis. They are mostly on the back part of the organ, associated with similar patches on the tonsils and fauces; the epithelial denudation, on the other hand, being usually seen on the anterior part of the tongue. In both cases the ulcers are symmetrical; and this symptom is a most valuable diagnostic sign of secondary syphilitic ulceration of the mouth and tongue. Tertiary ulceration of the tongue may occur in the form of deep fissures, in a longitudinal direction, with enormous hypertrophy of the papillæ along the edges of the fissure; or as nodes—the gummata of modern syphilographists. These nodes occur as an inelastic induration on the upper surface. Sometimes they are situated so far back as to require the laryngeal mirror to bring them into view. They frequently soften, burst, and leave a large sloughy ulcer, with ragged, thickened, and hardened edges. Ulcers of the tongue resulting from the action of *mercury* are usually associated with similar ulcerations of the gums and mercurial fetor of the breath.

The **Treatment** of syphilitic ulcerations of the tongue varies according to the severity and form of the affection. In the secondary varieties the local application of solutions of *chloride of zinc*, with washes of *chlorate* or *permanganate of potash*, are frequently sufficient; but sometimes the application of *solid nitrate of silver* is desirable. In the tertiary forms of syphilitic ulceration, *solid nitrate of silver* must be applied daily, and large doses of *iodide of potassium* frequently administered. In mercurial ulcers, constitutional treatment of a tonic character, disinfectant gargles, and generous diet are indicated. It may here be mentioned that in all cases of diseases of the tongue, all irritants, as spices and pepper, as well as smoking, sucking of lozenges, or any other measure calculated to increase the flow of saliva, must be strictly prohibited. (*c.*) For cancer of the tongue, the reader is referred to the subject of general cancer.

SECTION III.—DISEASES OF THE FAUCES AND PALATE.

QUINSY—*Syn.*, CYNANCHE TONSILLARIS.

ATIN EQ., *Cynanche Tonsillaris;* FRENCH EQ., *Esquinancie;* GERMAN EQ., *Angina Tonsillaris*—Syn., *Parenchymatöse und Phlegmonöse Entzündung des Rachens;* ITALIAN EQ., *Angina Tonsillare.*

Definition.—*Acute inflammation of the tonsils, which may or may not lead to suppuration.*

Pathology.—(*a.*) *Causation.*—Exposure to cold is almost always the cause of the attack; but in some cases it appears to be due to constitutional disturbance. Whether rheumatism and gout have any predisposing influence, it is undoubtedly true that some people are much more liable to quinsy than others; and a patient who has had one attack rarely escapes a second, unless radical treatment has in the meantime been adopted. In adults enlarged tonsils commonly predispose to the affection. Moreover, in persons subject to quinsy there is almost invariably disease of the follicles of the tonsils, so that the mucus, instead of being freely secreted, becomes hard and cheesy, and, blocking up the follicles, strongly predisposes to inflammation of the gland whenever the slightest cold is taken. Quinsy is most common during the autumn months. It is rare in children, and before puberty. It is also rare in the aged, and appears to be a disorder mainly confined to youth and middle life. According to the Mortality Returns of England and Wales, nearly 200 persons die annually from the effects of quinsy; but this may be due to an error of diagnosis—cases of death from diphtheria and scarlatina being probably returned under the head of quinsy. It is exceedingly improbable that a fatal result has ever occurred from simple uncomplicated tonsillitis.

Symptoms.—Inflammation of the tonsils is usually preceded by some shivering and fever, which are succeeded in a few hours by a sense of pain and dryness in the throat. The symptoms now increase in severity, and with great rapidity. The patient experiences great pain in deglutition, and on attempting to swallow, drink is sometimes ejected through the nostrils. There is a continual dull aching when the throat is at rest; the voice is altered, being thick and nasal, and the patient can hardly breathe, except through his nose; he has earache, and frequently is somewhat deaf. There is a constant flow of saliva from the half-open mouth, and a frequent desire to clear the throat of the viscid mucus which adheres to it. The tongue is covered with a thick clammy fur, and the breath is very offensive. These symptoms, combined with feverishness and loss of appetite, increase in severity, until either resolution takes place, or pus having been formed escapes. The degree of prostration which attends tonsillitis is usually out of all proportion to the severity of the local lesion. The attack generally subsides in a week or ten days, and rarely lasts a fortnight. It is often very difficult to examine the throat, because the patient is unable to open his mouth widely. Should a view be obtained, one or both tonsils may be red and swollen, and the passage through the fauces more or less completely blocked up. Although all the soft structures are swollen and œdematous, only one tonsil is generally affected at a time; but it is not uncommon for the inflammation as it subsides in one tonsil to attack the opposite side. The sub-maxillary glands are usually sympathetically enlarged.

Treatment must depend entirely on the stage at which the disease comes under notice. In the early period resolution may almost invariably be brought about by the administration of *guaiacum*, in the form of lozenges. Dr. Mackenzie prescribes a lozenge containing three grains of the extract of guaiacum mixed with black currant paste, every two or three hours. But I have found the drug most beneficially prescribed in the following formula:—

℞. *Magnes. Sulph.*, ʒvi.; solve in aquæ ℥viii.; adde *Pulv. Guaiaci*, ʒiss.; *Pulv. Tragacanth. Co.*, ℈ii.; misce bene. One-sixth part of this mixture may be given every four hours till the bowels are freely moved.

Minim doses of *aconite tincture* every hour will maintain a regular and steady action in subduing the inflammation. In addition, ice sucked constantly, and iced drinks, are most grateful to the patient; while, if ice cannot be obtained, cold thick gruel frequently affords relief. In other cases, however, greater relief is experienced by holding hot water in the mouth, and by the inhalation of hot steam, combined with sedatives—such as *benzoin* or *conium.* A mixture of mucilage or gruel, containing *nitrate of potash* or *borate of soda,* and a small quantity of *syrup of poppy, Batley's solution of opium,* or *prussic acid,* should be frequently but slowly swallowed in small quantities—in tea or table-spoonfuls; *the amount of narcotic ingredient being carefully regulated,* so that a definite quantity is consumed in a given time. *Externally,* a mustard poultice should be first applied, and afterwards linseed poultices. The latter may be constantly repeated until the attack subsides; spongiopiline may answer better than the linseed poultices, and is more cleanly. If suppuration has commenced before the practitioner sees the case, great relief will be afforded by puncturing the abscess with a guarded knife: the incision should always be made towards the median line. After the abscess has been relieved, the recovery of the patient is usually very rapid; but the prostration is sometimes considerable, so that nourishment and stimulants are required in abundance. It has been already remarked that a patient who has once suffered from quinsy is likely to suffer again; and the question naturally arises as to what preventive treatment should be adopted? In those cases in which there is any chronic enlargement of the tonsils, excision should be performed; but in some cases the glands, though extensively diseased, do not remain enlarged when the acute inflammation has passed away. Under these circumstances the tonsil may be amputated during the attack of quinsy. This plan of treatment is commonly adopted in Germany on its own merits, without any question as to the recurrence of the inflammation.

SLOUGHING SORE THROAT—*Syn.*, PUTRID SORE THROAT; CYNANCHE MALIGNA.

LATIN EQ., *Angina Putris*—Idem valet, *Cynanche Maligna;* FRENCH EQ., *Angine Gangréneuse*—Syn., *Angine Maligne;* GERMAN EQ., *Angina Maligna oder Gangrænosa;* ITALIAN EQ., *Angina Maligna.*

Definition.—*Idiopathic ulceration of an acute form, attacking the tonsils and apidly running into sloughing of the fauces.*

Pathology and Symptoms.—The disease is a very grave, often a most ntractable, affection, and as rapid as it is grave (POLLOCK). The first symptoms are those of quinsy; but on examination at a comparatively early stage, the whole throat will be seen to be generally œdematous, of a dusky-red colour, and covered with patches of grey or yellowish membrane. As these sloughs spread there is a discharge of a most offensive character through the nose, the patient is unable to swallow, and all fluids are ejected through the nostrils. With the progress of the disease the sloughs separate, and with them the uvula, or large portions of the soft palate, may also come away. If the patient recovers, swallowing is for a long time difficult, and the voice permanently nasal. This form of sore throat is probably the result of profound general blood-poisoning. The symptoms are not always so serious as just described, but the recorded cases of recovery are few in number. It occasionally happens that after an incision into a suppurating tonsil, the wound takes on a sloughy appearance, and severe quinsy may itself terminate in gangrene; but both these conditions, though serious enough in themselves, are different from the idiopathic cynanche under consideration.

Diagnosis.—The affection must be distinguished from malignant scarlet fever and from diphtheria.

Treatment.—In all cases of sloughing sore throat a stimulating and tonic treatment must be pursued. Tincture of the *perchloride of iron*, *quinine*, and *chlorate of potash*, are the most suitable internal remedies. Disinfecting gargles of *carbolic acid* or *permanganate of potash* are not only agreeable to the patient, but very useful in checking the offensive discharges.

ENLARGED TONSILS.

LATIN EQ., *Tonsillæ Intumescentes;* FRENCH EQ., *Hypertrophie des Amygdales;* GERMAN EQ., *Hypertrophie der Tonsillen;* ITALIAN EQ., *Tonsille Ipertrofiche.*

Definition.—*Hypertrophy of the tonsil, generally with induration.*

Pathology and Symptoms.—Hypertrophy of the tonsils is most common in early life; but the causes of this condition have not been satisfactorily ascertained. Rich and poor are alike subject to it, and probably a strumous diathesis is the strongest predisponent. In some cases it is probably due to a very slow interstitial inflammation, the result of syphilis; while in other cases it occurs as a sequel of exanthematous fevers. The development of the disease is usually unattended with pain; and attention may be first drawn to the enlargement by the thickness of articulation and breathing, snoring in sleep, and sometimes deafness. On examination, the glands are seen to be more or less hypertrophied, the mucous membrane covering them being much thickened and unevenly pitted, from enlargement of the follicles, which are sometimes blocked up with cheesy matter.

Treatment.—Although enlarged tonsils may be the result of constitutional derangement, they in themselves also interfere with the proper development and health of the body. Thus, they constantly obstruct the free passage of air into the chest, and thus interfere with the due development of the chest and the proper oxygenation of the blood; they give rise to an unhealthy secretion in the mouth; they sometimes interfere with sleep; and, in the case of adults, they subject the patient to repeated attacks of quinsy, with its associated conditions of difficult deglutition and pain.

The importance of removing the diseased and redundant portions of the affected gland will be at once evident. Tonics, sea-bathing, and other constitutional means, do not appear to exercise any beneficial effect in either diminishing or arresting the growth of tonsils predisposed to hypertrophy; nor are the applications of caustics of the slightest value, unless they are so strong as to destroy a large portion of the gland. It is better, therefore, to excise the enlarged tonsil in all cases when the respiration is affected, or where there is disease of the follicles predisposing to frequent attacks of inflammation and suppuration. The best method of removal is with a guillotine or wire écraseur. Dr. Mackenzie has lately invented a double guillotine, by which, in one operation, both tonsils are removed. This is a great desideratum in the case of children; as, after one tonsil has been removed, the practitioner has great difficulty in inducing his little patient to allow a second similar operation. Hæmorrhage seldom happens after removal of the tonsils. Should it occur, ice will generally check it; and if this does not answer, a few half teaspoonfuls of a fluid containing tannic and gallic acids in suspension, slowly sipped, will be certain to stop all bleeding.

ELONGATED UVULA.

LATIN EQ., *Uva Descendens;* FRENCH EQ., *Elongation de la Luette;* GERMAN EQ., *Verlängerung des Zäpfchens*; ITALIAN EQ., *Ugola Allungata.*

Definition.—*Relaxation or paralysis of the uvula, which becomes so elongated that its tip may touch or rest upon the surface of the tongue.*

Pathology.—(*a.*) *Causation.*—It is a most common result of inflammation of the throat; and is met with chiefly among clergymen, officers of the army and navy, singers, and those who are obliged to make great vocal efforts, or to continue the use of their voice when suffering from catarrh. The condition generally depends on simple relaxation of the mucous and submucous tissues, but the *azygos uvulæ* muscle may occasionally form some part of the increased growth.

Elongation of the uvula is always inconvenient, and sometimes even serious.

Symptoms.—The patient may experience nothing more than a slight tickling cough, accompanied by the sensation of a foreign body, only inconveniently felt at intervals. In other cases, the cough is constant and troublesome, and followed by frequent expectoration of little pellets of mucus. The constant tickling may produce a sensation of nausea, and occasionally vomiting takes place on waking in the morning. On lying down at night the symptoms are often increased, and the patient may suddenly awake with spasm of the glottis, brought on by reflex irritation set up by the elongated uvula. These constant attacks bring the patient into an extremely nervous state. The want of sleep and constant cough give rise to so much emaciation and weakness, that at first sight he appears to be the subject of phthisis or other serious disease.

On examination of the throat the cause of these symptoms will be at once evident; but it is important to observe the length of the uvula in relation to the level of the back of the tongue; for that which in one patient would be but a slight and harmless relaxation, would in another give rise to serious and distressing symptoms. When a patient opens his mouth, he always at the same time takes an inspiration, so that the uvula, being drawn up, and to a certain extent invaginated, may appear much shorter than it really is. In examining the throat, therefore, it is a good plan to tell the patient to EXpire, so that the real length of the uvula may be ascertained. The larynx of patients suffering from this condition is generally more or less congested.

The **Treatment** of elongated uvula must depend on the degree of relaxation present, and the previous duration of the affection. Slight and recent cases often get well by the persevering use of astringents. Gargles, containing *tannin*, *rhatany*, or *perchloride of iron*, lozenges, and local applications, may all be used with advantage; and, indeed, it is found convenient to brace up the mucous membrane by employing these different methods at the same time. When the uvula has been considerably relaxed for some months the elongated portion should be snipped off. While hardly any slight affection of the throat produces such serious symptoms as elongation of the uvula, there is no slight operation that gives such complete and permanent relief as its removal. Should any hæmorrhage follow the operation, treatment similar to that recommended after excision of the tonsil must be adopted.

SECTION IV.—DISEASES OF THE PHARYNX.

PHARYNGITIS.

LATIN EQ., *Pharyngitis;* FRENCH EQ., *Pharyngite;* GERMAN EQ., *Rachencatarrh;* ITALIAN EQ., *Faringitide.*

Definition.—*Inflammation of the textures of the pharynx.*

Pathology.—Inflammation of the pharynx may be either *acute*, *sub-acute*, or *chronic*. *Acute pharyngitis, or catarrh of the pharynx,* is generally met with in association with a similar condition of the fauces, and is but very rarely seen to exist alone. The exciting cause is almost invariably exposure to cold, but anything which tends to depress the vital power will predispose to an attack. The form of inflammation is most frequently of an œdematous character, and it generally terminates by resolution within a few days; but it occasionally extends to the larynx, in which case it is of much more serious import.

The **Symptoms** are—slight pyrexia, with a sensation of dryness and soreness in the throat, followed by great pain and difficulty of swallowing and by more or less dyspnœa if the swelling extends towards the windpipe When the larynx itself is affected the symptoms are those of œdematous laryngitis.

Prognosis.—The opinion given should always be guarded; but th disease is seldom serious when confined to the pharynx.

Treatment should consist in hot steam inhalations of a sedative character, poultices externally, and scarification if œdema takes place. When the uvula is much swollen and infiltrated with serum, it should be at once removed.

Sub-acute Pharyngitis may occur either as an early stage of the acute form, or as a less serious inflammatory affection. In this condition it is the mucous membrane alone, not the areolar tissue, which is inflamed. The *symptoms* are less severe than in acute pharyngitis; and *treatment* consists in the application of astringent solutions, and the use of astringent lozenges. The frequent sucking of small pieces of ice is of great service in reducing the hyperæmic condition.

Chronic or Granular Pharyngitis (*Clergyman's sore throat*).

Pathology.—(*a.*) *Causation.*—Patients who suffer from granular pharyngitis are generally the subjects of some constitutional weakness, the strumous diathesis being frequently present in a marked degree. There is also, in most cases, a history of over-exertion of the voice, the disease being most common amongst clergymen, public speakers, concert singers, and hawkers. These conditions—constitutional delicacy and excessive use of the voice—must therefore be regarded as the chief predisposing causes; but the exciting cause is generally exposure to cold, or direct irritation of the pharynx from inhaling a vitiated atmosphere.

(*b.*) *Morbid Anatomy.*—The disease appears in two forms—the *hypertrophic* and the *exudative.* In the former, the morbid process is chiefly confined to the mucous membrane, the pharynx being studded with granulations or masses of large swollen epithelial cells. In *exudative* pharyngitis the follicles of the pharynx are hypertrophied, and inclose small plugs of concretion, the result of fatty and calcareous degeneration of their epithelial contents. The pathological relation between the two forms still remains to be discovered.

The **Symptoms** of granular pharyngitis come on gradually, being at first confined to a sense of dryness in the throat and a slight tickling cough. These symptoms are followed at a variable period by hoarseness and weakness of voice, by a sense of burning in the pharynx, and by a constant harsh cough, with expectoration of scanty, viscid mucus. At a more advanced stage of the disease the senses of taste, smell, and hearing, may be impaired, owing to the extension of the morbid process to the orifices of the Eustachian tubes, and the lining of the palate and nasal passages: or the larynx and upper part of the œsophagus may become involved, giving rise to hoarseness and dysphonia, and to considerable pain in swallowing. The appearances seen on inspection of the fauces vary according to the nature of the disease. In the *hypertrophic* form the pharynx is dry, congested, and covered with small granulations, or, at a later period, with broad flattened elevations; the superficial veins are injected and tortuous. In the *exudative* form, the pharynx is also dry and congested; the follicles are enlarged, and discharge viscid mucus of a milky colour, or plugs of cheese-like matter. In other cases the exudation is of a chalky nature. The tissues generally are relaxed and the uvula elongated.

Granular pharyngitis cannot be easily mistaken, when the objective appearances are at all well developed. It is, as a rule, amenable to persevering treatment; but the weakness of voice is often very persistent.

Treatment.—The patient's general health must be improved by tonic measures, but the most important part of the treatment consists in the persevering use of local applications. In the *exudative* form the spots should be scraped, the secretion emptied, and solid *nitrate of silver* applied to each diseased follicle. Lozenges of *chlorate of potash* and of *bitartrate of potash* are useful in stimulating the follicular secretion to a healthy condition. In *hypertrophic pharyngitis* the raised papules must be destroyed one by one. The best application for this purpose is a paste consisting of equal parts of *caustic soda* and *unslaked lime;* this is mixed with water, as required, into a creamy paste, and applied to each papule with a finely-pointed glass or aluminium rod (Dr. MORELL-MACKENZIE). The caustic effect of this application is instantaneous. The patient therefore should be directed to wash his mouth with water immediately after the application, so as to prevent any other part of the throat being burnt by contact with the caustic. A combination of *ergotine* with *tincture of iodine* is also efficacious (*ergotine*, grains xx.; *tinct. iod.*, *f* ʒi.; *glycerine*, to make *f* ℥i.) To be applied to the pharynx twice a day with a camel-hair brush.

In all cases of pharyngeal disease, every form of spice, pepper, mustard, and piquant food should be strictly prohibited, and all fluids should be taken at a moderate temperature.

ABSCESS OF THE PHARYNX: RETROPHARYNGEAL ABSCESS.

LATIN EQ., *Abscessus Post-pharyngeus;* FRENCH EQ., *Abcès Rétropharyngieu;* GERMAN EQ., *Retropharyngeal Abscess;* ITALIAN EQ., *Ascesso Retrofaringeo.*

Definition.—*Inflammation of the pharynx, resulting in a limited collection of pus in the form of an abscess.*

Pathology.—Small collections of pus occasionally form in the wall of the pharynx as the result of catarrh, but these are speedily evacuated, and are unattended with danger. A more serious form of abscess, the retropharyngeal abscess, occurs in children, generally as the result of an idiopathic inflammation of the glandular structures of the pharynx of a scrofulous nature, but occasionally as a sequel of scarlet fever or of vertebral caries.

The **Symptoms** vary with the situation of the abscess. When seated at the upper part of the pharynx, there is difficulty of swallowing and breathing, and nasal modification of the voice. When seated lower down respiration is more seriously interfered with, and dangerous attacks of suffocation are not unfrequently present. In very young children the disease is often attended with general convulsions. The inflammatory swelling may extend to the sides of the neck, causing spasm of the sterno-mastoid muscle and wry-neck. These symptoms usually subside at once on the evacuation of the abscess, but the discharge of pus i sometimes so copious as to cause instant suffocation.

Diagnosis.—Retropharyngeal abscess has to be distinguished from œdem of the glottis, laryngeal croup, and foreign bodies in the larynx. As rule the physical examination of the throat furnishes the necessary criteria but it is further distinguished from laryngeal croup by the absence o dysphagia in the latter affection.

The **Prognosis** in idiopathic abscess is favourable if the pus is evacuated at an early period of the disease; when, however, there is caries of the vertebræ, the abscess is liable to fill again, or to burrow and form obstinate sinuses.

Treatment.—It is very rarely possible to prevent the formation of pus, and the expedient usually demanded is a speedy opening of the abscess at its most depending part. Cod-liver oil and other anti-strumous remedies are also imperatively demanded.

SECTION V.—TREATMENT OF DISEASES OF LARYNX AND PHARYNX BY THE USE OF ATOMISED FLUIDS.

The treatment of diseases of the *pharynx* and of the *larynx* by the use of *atomised* or *spray-producing fluids*, in which medicated vapour and gases are brought to act upon the parts, has received a great impetus since better appliances have been devised for using such remedial agents. The aim of such instruments is to atomise or pulverise, or very minutely to divide the fluid, by causing it to be thrown as a fine shower or spray, so as to be inhaled as such by the patient. Dr. Andrew Clark's hand-balls, fitted with Bergoon's tubes and Maunder's atomiser, constitute an efficient and useful arrangement for applying fluids in this way up the nostrils, to the back of the throat, or over the entrance to the larynx (DA COSTA). Dr. Richardson's well-known instrument for the production of local anæsthesia is also an excellent atomiser of fluid, and may be used for a similar purpose.

Where it is desirable to make the topical application directly upon the diseased part, these are the most simple instruments that can be used without loss of time by the physician; but where *inhalation* simply of vapour or heated air variously medicated is all that is required, nothing can be better than that used by Dr. Fergus, of Glasgow; or an improved apparatus for spray inhalations devised by Dr. James Adams of Glasgow. Siegle's apparatus, and Dr. Morell-Mackenzie's eclectic inhaler, and Martindale's portable inhaler, are all of them useful and valuable instruments for inhalation of suitable remedies.

On this most useful mode of treatment Dr. Da Costa makes the ollowing observations:—"(1.) Inhalations by means of atomised fluids re an unquestionable addition to our therapeutic means; but they are othing but an addition, and not a substitute for all other treatment. 2.) In most acute diseases of the larynx, and still more so in acute dis- rders of the lungs, their value, save in so far as those of water may tend o relieve the sense of distress, &c., and aid expectoration, is very doubt- ul; though in some acute affections, such as in œdema of the glottis and n croup, medicated inhalations have strong claims to consideration. (3.) n certain chronic morbid states of the larynx, particularly those of a tarrhal kind, and in chronic bronchitis, they have proved of great lue. (4.) In the earlier stages of phthisis, too, they may be of decided lvantage; and at any stage they may be a valuable aid in treating the mptoms of this malady. (5.) Their influence on such affections as oping-cough and asthma is not satisfactorily proved. (6.) They furnish a decided and unexpected augmentation of our resources in the treatment of pulmonary hæmorrhage. (7.) They require care in their employment; and in acute affections we should consider whether, as they have to be used frequently to be of service, the patient's strength justifies the disturbance or the annoyance their frequent use may cause. (8.) In any case, to be of service, they ought to be carried on as a treatment with a distinct object, and not intermittingly or spasmodically resorted to."

Table of Doses for Inhalation (DA COSTA):—(1.) *Alum*, 10 *to* 20 *grains*, to 1 fluid ounce of distilled water.—Suitable in chronic catarrhal states of the pharynx and air-tubes, particularly in bronchial affections with excessive secretion, when, as in most inflammatory conditions of the respiratory mucous membrane, it may be advantageously united with opium. In a somewhat stronger solution, 30 grains to the ounce, it is useful in pulmonary hæmorrhage. As an astringent, it is generally more of a sedative, and more suited to conditions of irritation than tannin (FIEBER). (2.) *Tannin*, 1 *to* 20 *grains*.—Useful for the same affections as alum. Employed in cases of laryngeal ulceration and excrescences, in œdema of the glottis (TROUSSEAU), and in croup. Here, as well as in pulmonary hæmorrhages, it is used in large doses. In ordinary cases of laryngeal or bronchial disease, it is well to commence with a comparatively weak solution—5 grains to 1 fluid ounce of water. If the remedy occasion much heat and dryness, its use should be discontinued. (3.) *Iron* (*perchloride of*), $\frac{1}{8}$ *to* 2 *grains*.—In earlier stages of phthisis; in chronic pharyngitis or laryngitis it may be used in a stronger form—3 grains to 1 fluid ounce of water. As a weak inhalation, it is useful in hysterical aphonia. Stronger solutions—2 to 10 grains to the ounce—are useful in pulmonary hæmorrhage. In ordinary cases in which we wish a non-astringent salt of iron, the lactate, citrate, or phosphate may be also used, though they are not, on the whole, as available as the chloride. (4.) *Nitrate of Silver*, 1 *to* 10 *grains*.—Is useful in ulcerations of pharynx and larynx, and in follicular pharyngitis. A face-shield is always to be used. 10 grains to the ounce only to be used in cases of ulceration. (5.) *Sulphate of Zinc*, 1 *to* 6 *grains*.—In bronchial catarrh with excessive secretion; in aphonia connected with chronic laryngeal catarrh. The *Chloride of Zinc* (2 to 5 grains) is also useful in the same conditions. (6.) *Chloride of Sodium*, 5 *to* 20 *grains*.—Promotes expectoration and diminishes sputa; employed in phthisis. (7.) *Chlorinated Soda* (*Liquor Sodæ Chlorinatæ*), $\frac{1}{2}$ *to* 1 *drachm*.—In bronchitis with offensive and copious expectoration; in phthisis. (8.) *Chloride of Ammonium*, 10 *to* 20 *grains*.—In laryngeal and bronchial catarrh, acute as well as chronic, to promote expectoration; also in capillary bronchitis. The dose best borne is not above 10 grains to the ounce. (9.) *Opium* (watery extract of), $\frac{1}{4}$ *to* $\frac{1}{2}$ *a grain*.—In irritative coughs, and as a adjunct to allay irritation. Also for its constitutional effects. Dose o *tincture of opium*, 3 to 10 drops. *Acetate of morphia*, one-twelfth to one eighth of a grain, has been administered; but large doses require muc caution. (10.) *Conium* (fluid extract of), 3 *to* 8 *minims*.—Irritative cough asthma; feeling of irritation in larynx. (11.) *Hyoscyamus* (fluid extrac of), 3 *to* 10 *minims*.—Spasmodic cough; hooping-cough. One-half grai of the extract, gradually increased; or the tincture may be employed. (12 *Cannabis Indica* (tincture of), 5 *to* 10 *minims*.—In spasmodic cough an phthisis. (13.) *Iodine, 2 to 15 minims* of the tincture.—In chronic bronchitis, phthisis, functional aphonia, and hay asthma. (14.) *Arsenic* (*Liq. Arsenicalis*), 1 *to* 20 *minims.*—Nervous asthma (TROUSSEAU). (15.) *Tar Water*, 1 *to* 2 *drachms* of officinal solution.—In offensive secretions from bronchial tubes; in tuberculosis; as an antiseptic in gangrene of lungs. (16.) *Turpentine*, 1 *to* 2 *minims.*—In chronic bronchitis with offensive secretions; in bronchorrhœa; in gangrene of lungs. (17.) *Lime Water*, used of officinal strength, or stronger.—In diphtheria; in membranous croup. (18.) *Water, Distilled.*—Cold, in pulmonary hæmorrhage; warm water in asthma; in croup; and in bronchitis.

For various other modes of applying topical applications to the larynx, the student is referred to Dr. Morell-Mackenzie's work *On the Laryngoscope.*

SECTION VI.—DISEASES OF THE ŒSOPHAGUS.

ŒSOPHAGITIS.

LATIN EQ., *Œsophagitis;* FRENCH EQ., *Œsophagite;* GERMAN EQ., *Entzündung der Speiseröhre;* ITALIAN EQ., *Esofagitide.*

Definition.—*Inflammation of the textures of the œsophagus.*

Pathology.—(*a.*) *Causation.*—Inflammation of the œsophagus is a rare disease. Morbid poisons seem to have little influence over this portion of the alimentary canal, and atmospheric vicissitudes are seldom followed by inflammatory affections in it. The most frequent causes of inflammation of the œsophagus are, accidentally drinking boiling water; swallowing corrosive liquids, as the mineral acids; and wounds, most commonly inflicted in the act of committing suicide. Children a few days old are sometimes affected with slight inflammatory affections of the œsophagus.

(*b.*) *Morbid Anatomy.*—Inflammation is characterised by a deep redness of the part, generally terminating by resolution, but occasionally followed by separation of the cuticle. Lymph may be thrown out. In new-born children points of lymph may be found on the mucous membrane, apparently an extension of thrush affecting the mouth and pharynx. After puberty inflammation is still more rare, but there are some few instances. Cruveilhier found among the preparations of Dupuytren a remarkable example of inflammation of the œsophagus, terminating in the formation of a false membrane, which coated this canal throughout its whole length. Abercrombie also gives the case of a gentleman, aged twenty-six, who caught cold, and died in about three weeks. The whole of the pharynx was covered by a loose adventitious membrane, which extended over the epiglottis, and portions of it were found lying in small irregular masses within the larynx at the upper part. A similar membrane was traced through the whole extent of the inner surface of the œsophagus quite to the cardiac orifice.

Ulceration may occur in the œsophagus as a result of irritant poisons. These ulcers in general form on the anterior surface, and by continued extension they may penetrate the posterior surface of the larynx or pleura, so that the patient may die from the escape of food into the lungs or pleural cavity. Occasionally the ulceration takes place from without inwards. The cicatrices are very apt to induce stricture. In cases of poisoning with the mineral acids, the whole œsophageal canal may become constricted and narrowed, and the mucous membrane puckered up and contracted, so as greatly to diminish the calibre of the canal generally. More commonly the stricture is partial, one circular muscular fibre perhaps having been abnormally contracted, and in this state bound down by adhesive inflammation, diminishing the diameter of the canal at that part to at least one-half. Dr. Baillie mentions a case in which, from this cause, the diameter of the œsophagus was so reduced as hardly to allow a garden pea to pass; yet in all other respects the œsophagus was healthy.

Symptoms.—The symptoms of *œsophagitis* are almost entirely local, and consist principally of pain, of dysphagia, of the expectoration of a thick viscid mucus, and perhaps of vomiting. Emaciation follows the loss of nutrition, and the patient ultimately dies from inanition. Stricture may be induced by the careless introduction of a probang.

Diagnosis.—The diseases with which lesions in the œsophagus may be confounded are similar states of the stomach; and the diagnosis is often difficult and perplexing. Stricture may be confounded with the spasmodic affections caused by an irritated state of the lung or trachea. Dr. T. Clifford Allbutt,[*] of Leeds, has been the first in this country to direct attention to auscultation of the œsophagus as a means of diagnosis, suggested by some papers of Hamburger (*Œsteireichische Medicin Jahrbücher,* for 1867-8-9).

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[*] This method of auscultation depends upon the audibility of the swallow both in the neck and thorax. It is necessary to educate the ear upon a healthy subject, who is requested to take a mouthful of water, and to swallow it at a signal. The operator then places the stethoscope (Sibson's stethoscope is the best for the purpose, but any stethoscope will do) first upon the trachea, anywhere between the *hyoid bone* and the *supraclavicular fossa.* The signal being given, the patient now swallows, and as he does so, a very distinct resonant gurgle is heard. This sound, which is very loud at the hyoid bone (where the water is, as it were, slung through a tube into the observer's ear), becomes duller as the instrument is removed to deeper parts of the neck. Below the *cricoid cartilage* the sound is more heavy or solid in character, and the morsel is, as it were, shoved downwards with a whiz. To examine the lower part of the œsophagus, the stethoscope must be carried down the *left side* of the spines of the first eight dorsal vertebræ. Here the sound is still more distant, though very distinct, and is like a smooth body slipping through with a sort of cluck. By repeated observations upon the healthy subject, the operator must make himself thoroughly familiar with *the tone*, with the probable size of the morsel, with the *energy of the œsophageal contraction*, with *the rapidity of it*, and also with *the direction of the morsel.* The rapidity of the passage of the morsel is ascertained by putting the stethoscope over the cardiac orifice while a finger is placed upon the larynx. The moment of commencing deglutition is known precisely by the rise of the larynx; the moment of its completion is recognised by the ear. The *rate* of the swallow is generally slower in weakly persons at all times, and in healthy persons after a long meal. The *direction* of the swallow may be reversed, as in regurgitation. In this case the gulped fluid eddies, as it were, in a funnel, with a prolonged resonant gurgle; or the direction of the swallow may be diverted, as where the œsophagus is perforated, and the matters escape into the pleural cavity or larynx. Hamburger had more than one case of the kind; and he prepared Dr. Allbutt to recognise this condition, which was quite easy when one was thus forewarned. The small quantity of diverted fluid passed through the chink in the œsophagus with a kind of sizzing murmur. *Pyopneumothorax* was present as a consequence, and there was thereby a metallic quality added to the tone. Or, while the swallow still runs in the tube, it may be heard *to cross the vertebral column*, and to appear on the right side only, for a greater or less distance. Hamburger relates a case of this kind, in which it was found, after death, that the *œsophagus had been pushed over by an aneurism of the descending aorta.* The swallow was in no way interfered with, so that *it seems desirable to auscultate the œsophagus as a routine in cases of suspected aneurism of the descending aorta.* The *energy* of the swallow must be

Prognosis.—Simple *œsophagitis* is probably often recovered from, as is seen after wounds of the throat partially dividing the œsophagus; but the chronic forms of inflammation may lay the foundation of disease leading to the ultimate death of the patient. Ulceration extending into the thoracic or pericardial cavity has been in all cases fatal.

Treatment of *œsophagitis* is by small local bleedings, by warm cataplasms to the neck, and by moderately acting on the bowels. In the treatment of the more chronic forms some sedative is essential. There is always some danger, in the use of the probang, of rupturing the canal, or of causing an ulcer. When the case is hopeless, from the small quantity of aliment which reaches the stomach, life may yet be prolonged by enemata of soups, milk, egg, wine, or other nutritious fluid matters. The question of an artificial opening into the œsophagus or the stomach may have to be considered.

SECTION VII.—RELATION OF THE ABDOMINAL VISCERA TO THE WALLS OF THE ABDOMEN.

The regions into which the abdomen is usually divided by lines are like those of the thorax, already described, quite arbitrary; and Figs. 19, 20, 22, already given (p. 530, *ante*), are referred to here; likewise the description given at p. 529, as to the fixed skeleton points which determine the direction of these lines, and the regions mapped out by them.

The vertical lines which have reference to the abdomen are five in number, and run as follow :—(1.) From the insertion of Poupart's ligament, in the external tubercle of the pubes, to the acromial extremity of the clavicle (right and left side); (2.) from the posterior boundary of the axilla (the inferior edge of *latissimus dorsi*) to that point of the crest of the ileum on which it falls vertically, Fig. 20 (right and left side); (3.) along the spinous processes of the vertebræ, from the sacrum to the nape of the neck (Fig. 23). The *transverse lines* indicate horizontal planes :—(1.) On the

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considered apart from its *rapidity*. In cases of dilated œsophagus, the fluid may pass the tube quickly enough; but the absence of energy of contraction is known by the want of grasp. The fluid passes down the tube with a squirting or running sound, not as though slung in a piece. It must be remembered, however, that in obstruction at the cardiac orifice, the accumulation of mucus may, and often does, modify or prevent the usual sounds for some five or six inches above the seat of the disease. *The tone of the swallow* is changed *in quality* by the presence of rough ulcers and the like on the inner surface of the tube, so that it becomes deadened; though the more common and more easily known result is the *prolongation of the morsel*, which, more than all other changes, catches the ear. Almost all changes in the inner surface of the tube may be regarded as diminutions of its calibre, and as checks to its peristalsis; so that, where disease exists, be it ulcer, be it contraction, be it tumor, we hear there *a slackening of the rate* at which the morsel is slung downwards, and *a prolongation of the morsel itself*. It tails off and slackens as it passes by. If the stricture be tighter, the morsel, when it reaches the spot, eddies through with a creak, or even with a squeak. If the stricture be tighter still, we hear the resonant regurgitation. Spasmodic dysphagia of a nervous nature causes no stethoscopic disturbances (*Brit. Med. Journ.*, Oct. 2, 1875).

point of the xyphoid cartilage; (2.) on the last short ribs; (3.) on the anterior superior spinous process of the ileum on each side; (4.) on the upper margin of the *os pubis.* These map out the abdominal walls into *thirteen* regions, of which five are anterior, four are lateral (two on each side), and four are posterior. The *anterior regions* are *epigastric* (No. 4 on the figures), *umbilical* (No. 5), *hypogastric* (No. 6), right and left *inguinal* (No. 11). The *lateral regions* comprehend the right and left *hypochondriac* (No. 9), the right and left iliac (No. 10). The *posterior regions* embrace the *inferior dorsal* on the right and left (No. 15), the right and left *lumbar* (No. 16). The best idea of the contents of these regions is obtained by defining first *the limits of the liver.* It fills the right *hypochondriac* region (No. 9, Figs. 20 and 22), filling up the concavity of the diaphragm; and it is almost completely concealed by the arch of the ribs. A part of the left lobe projects into the *epigastric* region (No. 4, Figs. 20 and 22) and *left hypochondriac.* It also projects upwards into the *infra-axillary* region (No. 8, Figs. 20 and 22), where it is separated from the thoracic wall by the thin lower margin of the right lung. Its upper margin is on a line nearly with the level of the nipple, about the fifth intercostal space—less frequently beneath the fifth rib. In the perpendicular axillary line its margin is about the *seventh* intercostal space—seldom under the seventh rib; close to the vertebral column its margin is in the tenth intercostal space—less frequently in the ninth (FRERICHS). At the median line the upper boundary of the liver cannot usually be distinguished from the lower margin of the heart. It is best made out by drawing a straight line from the point of contact of the right margin of the cardiac dulness with the upper boundary of the liver to the apex of the cardiac dulness on the left (CONRADI, FRERICHS, Vol. I., p. 30). Percussion of the liver after a meal is to be avoided; and any obstinate constipation or accumulations of gas which may be present must be removed before percussion. In the *epigastric* and *left hypochondriac regions* lies the stomach. The *umbilical region* is crossed by the transverse colon, passing from right to left a little above the umbilicus. The convolutions of the jejunum and ileum occupy the *umbilical* and *hypogastric regions.* The large intestines surrounding the convolutions of the lesser intestines occupy the *iliac* and *lumbar regions* on each side. The kidneys are equally shared between the *infra-scapular* (No. 14, Figs. 21 and 23) and the *inferior dorsal regions* (No. 15, Figs. 21 and 23). The spleen in its greater bulk is in the same region on the left side.

SECTION VIII.—METHODS OF EXPLORING THE ABDOMEN.

These are principally three—namely, *inspection, manual examination* (*palpation*), and *percussion.* (See also page 26, Vol. I.) *Inspection* furnishes information relative to *size, form,* and *movement;* and such information ought always to be acquired when the chest, as well as the abdomen, are both exposed simultaneously in a good light, the patient being protected from cold by a previous regulation of temperature in a room suited for the purpose of such an examination. The eyes of the patient ought to be directed away from the examiner. *Palpation* furnishes information relative to position, size, consistence, elasticity, spontaneous movement or mobility, and the presence of vibrations which may reach the surface. Care should be taken that the hand, when applied, should not be cold. *Percussion* furnishes information relative to the comparative solidity of regions, and thereby indicates the condition of the organ immediately below the seat of percussion.

The chief objects to be held in view in exploration of the abdomen are the following:—To ascertain—(1.) its form and size; (2.) its degree of tension or solidity; (3.) its temperature; (4.) sensibility or tenderness over any part; (5.) the presence or absence of tumor in or amongst the viscera; (6.) the presence or absence of fluids in the peritoneal sac; (7.) the nature and extent of the intestinal contents. The following points should also be made a note of:—(1.) The apparent age as related to the real age of the patient. (2.) Complexion and physiognomy. (3.) The existence of glandular swellings, ulcers, or varicose veins. (4.) Results of examination by rectum and vagina. (5.) The existence of fluid, free or encysted. (6.) Microscopic examination of such fluid removed by tapping or otherwise. A great addition to our knowledge has been made in the diagnosis of malignant tumors of the ovary, and of malignant peritonitis, by Dr. James Foulis, of Edinburgh, and afterwards by Mr. Knowsley Thornton, of London. They both describe characteristic groups of large pear-shaped, round, or oval cells containing a granular material, with one or several large clear nuclei, with nucleoli and a number of transparent globules or vacuoles (*Brit. Med. Journ.*, July 20, 1878).

Section IX.—Relative Weight, Measurement, Bulk, and Specific Gravity of the Solid Viscera of the Abdomen.

Weight.—The *liver*, the *kidneys*, the *spleen*, and the *pancreas* are the organs which require special notice here; and in order to obtain positive data for the solution of many questions regarding their pathology, it is necessary to know the relative weight of these viscera.

The Weight of the Liver ranges in the adult from *fifty* to *sixty* ounces (avoird.)—*i.e.*, from 800 to 960 drachms.

According to the experience of Frerichs, the relative weight of the liver in healthy individuals may vary from $\frac{1}{17}$ to $\frac{1}{50}$ of the body; and in adults it fluctuates between $\frac{1}{24}$ and $\frac{1}{40}$. During the period of greatest growth of the body, the liver does not become enlarged in a manner proportionate to the increase of the entire body; and its diminution in old age is for the most part in advance of that of the body. Its substance in this respect, therefore, presents a marked contrast to the muscular tissue of the heart; for whilst the heart increases progressively up to an advanced age (Bizot, Boyd), the mass of the liver diminishes Frerichs, Boyd); and in old age, as a general rule, there is senile trophy of the organ.

The weight of the spleen in the adult ranges from *four* to *ten* ounces avoird.)—*i.e.*, from 64 to 160 drachms. The weight in *malarious ypertrophy* has risen as high as eighteen, twenty, and forty pounds. t has, in some conditions of *atrophy*, weighed not more than *half an* *ounce.* The weight in relation to the body is as 1 to 350 or 400 up to forty years of age, and as age advances as 1 to 700.

The weight of the kidneys ranges from four and a half to five and a half ounces each; the left being about one-fourth of an ounce heavier than the right; and the weight of both together, in proportion to the body, is as 1 to 240.

Measurements.—(1.) The *liver* measures from ten to twelve inches from side to side, six to seven inches from front to back, and about three and a half inches thick at posterior and thickest part of right lobe. (2.) *The spleen* measures about five inches in length, three and a half inches from front to posterior edge, and one and a half inch thick. (3.) *The kidneys* each measure about four inches in length, two inches in width, and one inch in thickness, the left being generally a little longer and thinner than the right.

Bulk (represented by cubic inches of water displaced):—(1.) *Liver,* average bulk from 88 cubic inches (KRAUSE) to 100 cubic inches (BEALE). (2.) *Spleen,* average bulk, 9¾ to 15 cubic inches (KRAUSE). (3.) *Kidneys,* average bulk, about 12 cubic inches each.

Specific Weight or Gravity.—(1.) The *liver* ranges from 1·050 to 1·060 (KRAUSE); and in fatty degeneration 1·030, or less. (2.) *Spleen,* about 1·060. (3.) *Kidneys,* about 1·050.

SECTION X.—DISEASES OF THE PERITONEUM.

PERITONITIS.

LATIN EQ., *Peritonitis;* FRENCH EQ., *Péritonite;* GERMAN EQ., *Bauchfellentzündung;* ITALIAN EQ., *Peritonitide.*

Definition.—*An inflammation of the serous membrane lining the cavity of the abdomen, and covering the viscera contained in that cavity.*

Pathology.—(*a.*) *Causation.*—Inflammation of the peritoneum may manifest itself as a *secondary* affection during the course of some specific disease, such as *malarial fevers, scarlet fever, Bright's disease, scrofula,* and the like; as the extension of some irritative process, or by *perforation;* it may also be produced by *hepatitis, splenitis, enteritis;* by *cancerous, typhoid,* and *tubercular* deposits in the subperitoneal tissue. *Intus-susception* or *strangulation* of the intestine, hernia, inflammation or abscess of mesenteric glands, ulceration (typhoid or tubercular) of the bowels, the bursting of an abscess, or of an aneurismal tumor into the abdominal cavity, are also occasional causes. Mechanical violence, as the kick of a horse, a penetrating wound, the operation for hernia, lithotomy, ovariotomy, laparotomy, or of paracentesis, are occasional causes regarded as *traumatic.* Errors of diet and drink, especially frequent intoxication, are also causes. The disease termed *gin colic* is a chronic inflammation of the peritoneum. Sudden and great changes of temperature are also causes, especially in women at the period of menstruation. Children may die of peritonitis after fevers; but the disease is most common between the ages of twenty and forty. Women appear to die more frequently from it than men; and this greater liability to peritonitis in the female arises, perhaps, from the great sympathy between the uterus and the peritoneum—a sympathy which is strongly marked, not only at the time of parturition, but also at the periods of menstruation. At the former period, puerperal peritonitis often becomes contagious among the parturient females. Its existence as a primary affection, like pleurisy, is generally ignored. But there is a class of cases which may be described as forms of *idiopathic peritonitis* in which effusion suddenly occurs into the peritoneum analogous to cases of latent pleurisy (Dr. GEORGE JOHNSON and Mr. R. H. MEADE, *Brit. Med. Journ.*, Sept. 16 and 23, 1876). These cases mostly arise from cold. They are generally subacute, and often latent; in this respect bearing a great resemblance to the latent forms of pleurisy not uncommon in children and young persons. An *infective form* of *peritonitis* is also met with in the puerperal state, and as a sequence of purulent pleuritis. It occasionally occurs as a consequence of the passage of infective material from the pleural cavity along the lymphatics of the diaphragm to the peritoneum. "Pus sometimes makes a passage through the diaphragm. This either happens in a *diffuse* and *acute* manner, and sets up an acute and invariably rapidly fatal peritonitis, altogether analogous to the purulent, usually double-sided pleurisies, which also result from the extension of the purulent inflammation through the lymphatics of the diaphragm in purulent puerperal peritonitis" (ZIEMSSEN'S *Cyclop.*, Vol. IV., p. 620; BURNEY YEO).

(*b.*) *Morbid Anatomy.*—The peritoneum, like the pericardium and pleura (*q. v.*, pp. 589, 773), is liable to inflammation, either acute or chronic. Acute inflammation begins in the connective fibrous tissue, which becomes red and injected, with germination of its elements. At length the same phenomena pervade the serous membrane itself, when free exudation occurs, either as a fibrinous material or as a serous fluid, with or without pus-corpuscles. The colour of the peritoneum when inflamed is a bright arterial scarlet hue from the intensity of vascularisation. At first it is dotted with a number of small red points, which become confluent, forming streaks and patches, which in their turn coalesce; or a small central nucleus of inflammation may form and spread, till the whole extent of the peritoneum is one bright red colour. It is generally most marked where the coils of intestine touch and adhere. Some interstitial growth accompanies this diffuse inflammation, so that the peritoneum loses its transparency and is thickened. When redness does not exist, opacity may be the only evidence of the previous peritonitis. The consistence of the subperitoneal tissue and muscular coat of the intestines is greatly impaired. They are easily lacerable, so that the peritoneum may be detached in considerable portions; and the intestine readily tears. This inflammation may terminate by resolution, or it may advance to the effusion of serum, which may not exceed a few ounces. Occasionally it is so large as to fill the cavity of the abdomen, and constitutes inflammatory dropsy. The exudation may be of the fibrinous type, when coagulation in soft masses and flakes tends to occur; or the opposed surfaces may be glued and matted together by firmer lymph. In some cases the fluid predominates, and gelatinous-like masses are loose in the serum; or they are of such consistence and extent as to form a membrane, covering the entire surface of the abdominal walls as well as of the intestines. The period at which organisation of the lymph may begin, Mr. Hunter determined to be about twenty-four hours after irritation was set up. Pus may form to a few ounces, or to many pints, or it may even fill the whole of the abdominal cavity. The exudation may be purulent or mixed with blood, as in those forms which depend on blood-poisons, especially puerperal cases. The different acute inflammations have sometimes been described as succeeding each other; but all these different forms may co-exist in different parts of the peritoneum at the same time. Certain parts of the peritoneum are more liable to inflammation than others, as the convex surface of the liver or spleen, the right iliac fossa, the surface of the small intestine, and in females the broad ligaments, the Fallopian tubes, and the parts immediately adjoining them, as also the space covering the rectum and bladder. The parts the most rarely affected are those covering the stomach, bladder, omentum, and the mesentery. The liability of different parts of the peritoneum to inflammation seems to be in proportion to the liability of the organs it covers to become diseased; and these partial inflammations are the result of continuity from these diseased parts. The tendency to peritonitis is much greater in persons affected with *scrofula*, *morbus Brightii*, and other exhausting diseases, as well as in women at the menstrual periods, than in healthy persons. In the *idiopathic* and *latent* forms of peritonitis the inflammatory process seems to be principally or entirely confined to the parietal parts.

The forms of peritonitis to be distinguished are :—(*a.*) *Metro-peritonitis*, or *puerperal peritonitis*, generally infective, in which the products are of the most virulent septic character; (*b.*) *Chronic peritonitis ;* (*c.*) *Suppurative peritonitis ;* (*d.*) *Tubercular peritonitis ;* (*e.*) *Adhesive peritonitis ;* (*f.*) *Idiopathic* or *latent forms ;* to which may be added (*g.*) *Encysted inflammatory products* and local adhesions of the parts opposed to each other—forms which indicate a local source of irritation as the immediate cause of the inflammation. The *local* varieties are named after the part affected, as *parietal*, *hepatic*, or *splenic*.

Symptoms.—Peritonitis may be acute or chronic, partial or general, and latent or obvious. It may be ushered in by shivering and fever; but in many cases there are no preliminary symptoms, its onset being at once sudden, and well marked. Peritonitis from perforation of the intestine or stomach is characterised by the *suddenness* and *intensity* of pain, often referred to a particular region; but the whole abdomen soon becomes painful to pressure. The pain is constant, exquisite, and leads to such lowering of the heart's action that death may rapidly follow by *asthenia*, preceded by the most marked symptoms of collapse. If acute peritonitis should not terminate by resolution, but by effusion of serum or of lymph, the patient suffers a severe local pain, increased on pressure. He lies on his back, fearing to move, with a pale or flushed face, expressive of great suffering and anxiety. His pulse is from 90 to 120, and peculiar as an inflammatory pulse; for in proportion as it is frequent, so is it small, sharp, wiry, or thread-like. The tongue is coated, and the bowels are constipated. The presence of serum may be determined by fluctuation, or by percussion in some parts. If lymph exist, a rubbing sound may be heard under the stethoscope when the abdominal movements of respiration are not suppressed. The course of acute peritonitis varies from a few hours to ten or fourteen days; and when it terminates in *effusion of pus*, the pain in the abdomen is of the severest kind. The patient lies on his back, with his legs drawn up and bent, so as to relax the abdominal muscles. By fixing his pelvis he endeavours to keep the abdomen still; he is restless, and unable to bear the slightest pressure, not even the weight of a sheet. He is incessantly tossing his arms about. His pulse is excessively small and rapid, varying from 130 to 150, while he may be distressingly affected by retching and vomiting. These symptoms may continue without intermission for twenty-four, forty-eight, seventy-two, or more hours; when, with or without some previous shivering, pus is formed, and the pain, from being agonising, is bearable. But this subsidence of pain is not followed by any amendment; on the contrary, alarming collapse succeeds,—a cold clammy sweat breaks out over the body, while hiccup, and a pulse hourly increasing in frequency, proclaim the entire hopelessness of the patient's surviving beyond a few hours. When *acute peritonitis is local* (confined, for instance, to the surface of the liver or other organ) the pain is often limited to that part, while other symptoms vary according to the severity of the affection and the organ whose covering is affected. If about the rectal and vesical region the calls for micturition may be frequent. *Chronic peritonitis* often takes place without any great amount of suffering. The symptoms are those of abdominal soreness and uneasiness, with a full but sometimes rapid pulse. The intestines may be glued together, and sometimes pus has been found, without the patient suffering more than in ascites. When *chronic peritonitis* is partial, as of the liver or spleen, the patient may experience a dragging pain, increased by a change of position, arising from the parts being attached by adhesions. In the *latent cases*, enlargement of the abdomen may be the first symptom to attract attention. Tympanitic distension is often extreme. The abdominal respiratory movements may be quite suppressed.

Diagnosis.—The pain being increased on pressure, and the pulse rapid, with general uneasiness and evident danger of the patient, peritonitis is thus readily distinguished from colic. Its salient points of difference from *enteritis* will be noticed under that head. It is the *idiopathic* and *latent* forms of peritonitis which are so difficult to recognise. They almost always occur in adults, and effusion of fluid is rapid. They are not to be confounded with the tubercular forms of peritonitis which commonly occur in children, are very chronic in their character, and very intractable (R. H. MEADE). They are also to be distinguished from the intractable cases of ascites due to cirrhosis of the liver (Dr. GEO. JOHNSON).

Prognosis.—*General peritonitis* is usually fatal, the approach of death being indicated by the symptoms of collapse, with a rapid, feeble, and irregular pulse. *Partial* or *local peritonitis*, *traumatic*, often terminates without impairing the general health; and we may find extensive adhesions of the liver without any marked symptoms, as well as limited opacities of the membrane. Where the structure of the peritoneum is thickened or otherwise impaired, the patient may recover; but generally he relapses and dies of dropsy. Every attack of acute peritonitis is of grave import, and when pus is effused it is generally fatal; neither will the patient likely recover if the peritonitis is caused by subperitoneal tubercles, typhoid or cancerous lesions. Great dyspnœa with typhoid symptoms, or low nervous pneumonia, are unfavourable signs.

The **Treatment** of acute peritonitis must be active in proportion to the amount of pain, the rapidity of the pulse, and intensity of the inflammatory fever, which is marked by the peculiar depressing influence of the inflammation of the heart's action. In mild cases, when the pain is bearable, the pulse steady and under 100, *twenty leeches* over the abdomen, followed by *warm fomentations,* with the administration of *opium* in frequently repeated grain doses, ought to be the basis of treatment. *Leeches* applied to the abdomen is the only method of blood-letting likely to be useful, and a poultice may be required afterwards to encourage the bleeding in sthenic cases. But where the disease is due to blood-poisoning, constitutional states of ill-health, or to the puerperal state, blood-letting by leeches is injurious. In the severer forms the first indication is to relieve pain. The stomach may be unable to retain food; and vomiting may be present. Under such circumstances great benefit may follow the injection *per rectum* of a pint of strong meat soup slowly thrown up in three successive portions. After about three hours' relief of pain, a fresh new injection of soup is to be given, when the same relief to pain will follow, the pulse falling from (say) 124 to 104. Small quantities of broth and wine may then be borne by the stomach, administered every two hours (Dr. ANSTIE) *Action of the bowels* should be prevented for several days. *Opium* should be given in doses of one to two grains, repeated as often as its effects subside—generally every two, three, or four hours. *Morphia* may be given in repeated doses of a quarter to half a grain. *Morphia* combined with *chloroform,* as in the formula for *chloromorphine* or *chlorodyne,* will be found useful in allaying pain. It is not proven that *calomel* (inducing mercurialism) has any curative tendency in peritonitis. It has been prescribed traditionally; but the experience of Dr. Taylor regarding *mercury* in *pericarditis,* as well as of other physicians regarding the influence of mercury generally in the cure of these inflammations, tends to discard it from our methods of cure in such affections. Nevertheless, in the *idiopathic* and *latent forms,* Mr. Meade has found mercury the best remedy. In the purely latent forms he prescribes small doses of the *bichloride* with some *demulcent.* One-twentieth of a grain three times a-day is sufficient. It may be continued for two or three weeks or more. In the more acute cases he directs the abdomen to be covered with *dilute mercurial ointment* spread thickly upon lint; and he generally finds that the ascitic fluid begins to be absorbed as soon as the mercury slightly affects the system. Fomentations and water compresses are to be diligently employed on the same principle as described under *Enteric Fever,* Vol. I., p. 623. *Digitalis,* with other *diuretics,* may assist the cure. In the more chronic forms, where the abdomen has become distended, it is sometimes desirable to let off the fluid by tapping; after which remedies act more readily, and generally prevent the return of the effusion. Diuretics of *iodide of potassium, acetate of potass,* and *squills,* also help to effect the removal of fluid. The entire abdomen may be covered with cold compresses, and renewed every ten minutes; but warm cataplasms are better borne than cold compresses by many patients (NIEMEYER). In *chronic peritonitis* I have found *iodide of potassium,* combined with *alkalies,* most useful, together with inunction of the abdomen with the *iodine* or *biniodide of mercury ointment.* *Acute peritonitis* has been treated by *quinine* alone, in large and repeated doses, by M. Beau, at the Hôpital de la Charité. The remedy was given in the proportion of twenty to thirty grains in the twenty-four hours. When the abdomen can bear pressure, a flannel roller should be firmly applied round the body. *Chronic vomiting* is a distressing concomitant to be met by effervescing draughts, with ♏ j. or ♏ ij. of *dilute hydrocyanic acid*, or with ♏ iij. to ♏ v. of *tinct. opii.*; or one or two drops of *creasote* in aromatic water. *Diarrhœa* is common, hence it is necessary to combine any laxative remedy with an opiate; and the bowels may be so irritable as to oblige us to abandon all opening medicine, and to prescribe astringents, as *kino, catechu*, or *hæmatoxylon*, or the *mistura cretæ composita c. opio*, or the *acetate of lead*, and even *pure opium*, to the amount of two, three, or four grains a day. Under any treatment diarrhœa is dangerous and distressing. *The diet* in the acute forms of peritonitis should be of the mildest and least stimulant kind. During the course of this and several other diseases of the digestive system, immense distress and suffering often occur from the accumulation and retention of gaseous fluid in some portions of the intestinal canal (*tympanites*). *Tympanites* is best relieved by *turpentine* enemata; and by the introduction of a rectum tube. When this symptom reaches such extreme development as to endanger life, and after the introduction of a rectum tube fails to procure relief, experience justifies the propriety of making a puncture into the cavity of the bowel (generally the colon), through the abdominal parietes, to permit the gas to escape, and so relieve the tympanites. Life may thus be saved, and cannot be materially shortened by the operation, if it is performed by a trocar of the very smallest exploring size. The air gradually and rapidly escapes by the very smallest orifice, which is rather a separation of tissues than a wound, and soon closes up so completely as to leave no trace behind. The operation gives the greatest possible relief (see *American Journal of Med. Science*, N. S., XXIV., p. 543; *Obstet. Trans.*, for 1869, pp. 47, 48, by Dr. J. Braxton Hicks, in 1868; *Brit. Med. Journal*, for Oct. 21, 1871, p. 464, by Mr. J. Hancocke Wathen). The operation is not the same as that usually performed on cattle. In them it is generally the *rumen* or first stomach that is punctured (M'Bride in *Brit. Med. Journal*, Nov. 4, 1871).

ASCITES: DROPSY OF THE PERITONEUM.

Latin Eq., *Ascites;* French Eq., *Ascite;* German Eq., *Ascites*—Syn., *Bauchwassersucht;* Italian Eq., *Ascite.*

Definition.—*A collection of serum slowly effused into the cavity of the eritoneum* (see p. 112, Vol. I.).

Pathology.—(*a.*) *Causation.*—*Ascites* may result after undue exposure to ld and wet. . To this form the name of *active ascites* has been given; and though such cases have not the marked symptoms of inflammation, yet the brile reaction which generally accompanies them, and the fact that they eld to those remedies which subdue inflammation, point to inflammation the cause of effusion. There are cases in which the *ascites* obviously sults from *chronic peritonitis;* and, although the patient may suffer much pain, usually pain is wanting, or only occurs in occasional paroxysms. The urine is scanty, and may be free from albumen in both these forms of disease. The following causes of *ascites* are examples of the *passive* form, in all of which the cause tends ultimately to retard the flow of blood through the system of the *vena portæ*:—(1.) A *diseased heart*, or morbid state of the aorta, is often a primary cause of ascites. Dropsy may then first show itself by effusion into the abdomen, or into the areolar tissue of the lower extremities, causing *anasarca.* When effusion has taken place, the action of the heart may become more regular, its impulse more natural, the pulse slower and steadier, while the cardiac murmur also may disappear. But the apparent amendment is fallacious; the dropsical symptoms increase, more effusion occurs, first into one cavity and then into another, so that the patient seldom long survives. The urine in this form of dropsy is generally deep in colour, small in quantity, and of normal density. (2.) *Diseases of the liver* offer the best illustration of how morbid states obstructing the *portal* circulation are the main causes of *passive* or *mechanical* ascites. When ascites arises from a *diseased liver* it may be enlarged; but mere enlargement does not commonly give rise to ascites. The liver in most instances is smaller than usual; it is contracted and condensed, so that in shrinking its condensed textures compress the portal circulation. The condensation is of a peculiar kind. It generally results from compression of the proper hepatic substance, by a contracting tendency in the new connective-tissue which accompanies the portal vessels—namely, the *capsule of Glisson,* the active germination of which gives rise to the condition known as *cirrhosis,* or *hob-nail* liver. The patient may or may not be jaundiced. In the former case all the fluids effused are of a yellowish or greenish-yellow colour. The urine is loaded with bile, which is generally turned green by the addition of nitric acid. In a small number of cases the bile appears to be in a peculiar state of combination with the urine, so that the acid has no effect on it. The urine is always small in quantity, much loaded with the usual salts, and of a high density. The bowels are difficult to move, and the patient is liable to severe abdominal pains, simulating chronic peritonitis. The pulse continues for the most part natural, but the patient usually falls into a typhoid state, from which there is no recovery. The pressure may also be exerted on the portal system outside the liver, in some part of its fissure, as from hydatids, cancer, or abscess, or enlargement of glands in the fissure, aneurism, or perihepatitis. (3.) In ascites associated with *disease of the spleen,* it is uniformly enlarged, and can readily be felt occupying the left hypochondriac region; and thus the cause, though not its exact nature, may be determined. The early symptoms are similar to those which occur in dropsy from disease of the liver, and for the most part are secondary to hepatic obstruction. If the patient dies, it is generally by hæmorrhage from the stomach and bowels, often so profuse as to amount to many pints in a few hours, greatly exhausting the patient, and hastening the fatal issue. (4.) In dropsy from *disease of the kidney* the urine may or may not contain *albumen;* but in the great majority of cases it does. When albumen is absent (as the chronic forms of diseased kidney are all devoid of pain), we may not be able to determine either the seat or the nature of the disease with which ascites is associated; and the ascites may be attributed to an affection of the peritoneum, the liver, the spleen, the kidneys, the heart, or other viscus. The causes of ascites may be further summed up as comprehending undue exposure to sudden changes of temperature, loss of blood, obstruction of the portal circulation from morbid states of the liver, or spleen, or heart, and especially from dilatation of the chambers of that organ. The lesions of the kidney associated with ascites generally exercise their pernicious influence through diseases of the heart supervening during their course. Such cases prevail most between the ages of twenty and forty-five, while cases from disease of the heart and liver are most common from the ages of forty to sixty.

(*b.*) *Morbid Anatomy.*—The quantity of fluid contained in the peritoneum in cases of ascites varies from a few ounces to many gallons: three to four gallons are by no means unusual, and as much as eighteen gallons have been drawn off at one time by the operation of *paracentesis.* The quality of this fluid varies. In colour it is generally green or yellow; in consistence viscid, often containing so much coagulated matter as to be incapable of flowing through the canula. Cases of ascites may be examined after death, in which no affection of the peritoneum, or of any organ or tissue, can be discovered. More commonly, however, the peritoneum shows evidence of having been either acutely or chronically inflamed, some viscus diseased, such as the liver or the heart, or some tumor may be found pressing on the large vessels, and causing the effusion. It may also be the result of tuberculous or carcinomatous affections. The most frequent concomitant affection is disease of the heart and large blood-vessels, to which it is believed that at least one-fourth of all the cases of ascites is owing. The cavities of the heart may be enlarged, and its walls either hypertrophied or atrophied, or the valves diseased, or their action otherwise impeded, and the aorta may be pouchy—its elasticity and contractility being impaired. Morbid states of the liver and spleen, in every possible state and stage of disease, are the next most frequently associated affections. Sometimes *anasarca* accompanies *ascites.* It is an infiltration of serous fluid amongst the elements of the general connecting or areolar tissue of the body, passing up through and amongst the more loosely connected parts of the body generally, such as between the skin and the muscles. The fluid of *anasarca* is generally limpid and watery, composed merely of the serous part of the blood; while in other instances the fluid is viscid, contains lymph, and the organisable elements characteristic of inflammatory origin.

The **Symptoms** of ascites vary in some degree according to the cause. If the quantity of fluid effused be considerable, the abdomen is distended and shining, with a number of large superficial veins creeping over its surface. From the weight of the fluid, the gait of the patient is upright, like that of a pregnant woman; and if anasarca be present, he generally walks with his legs widely apart. In bed he is unable to lie down, on account of the fluid in the abdomen gravitating towards the chest and compressing the lungs, so that he is obliged to be raised towards the head and shoulders. If *anasarca* be also present and limited to the lower extremities, the upper portion of the body is in general emaciated, the sharp and pinched features and the withered arms, forming a striking contrast to the protuberant abdomen and swollen legs. On the contrary, if *anasarca* be general (as the true signification of the word implies), the trunk, the arms, the hands, the eyelids, and face generally, are tumid and swollen. The urine is often defective in quantity; the skin is dry, and the patient thirsty; his appetite impaired, and his spirits generally depressed. The progress of ascites is seldom accompanied by any severe constitutional symptoms. Bronchitis may take place, or the urine may become nearly suppressed, and similar effusions may occur in the cavities of the pleuræ. Digestion is impaired. Flatulence and constipation prevail. Dyspnœa may be urgent in proportion to the amount of fluid and of tympanites pressing up the diaphragm; and the heart's action may be similarly disturbed.

The presence of fluid in the abdomen may be determined by percussion; and the best mode is to place one hand on the abdomen, and to give a sharp but gentle tap on the opposite side with the fingers of the other, when, if fluid be present, fluctuation will be felt. If the quantity of fluid be small, the fluctuation is best felt by percussing the side of the abdomen from before backwards. The distension and peculiar form of the abdomen is characteristic. So long as the effusion is moderate, the shape of the belly will change with every position of the body. If the patient stands, the lower part of the belly is prominent—if he lies down it is broad. The existence of fluid in the areolar tissue of the trunk or extremities constituting *anasarca* is determined by the finger leaving a mark or "pit;" and the fluid being thus displaced, the part does not recover its original form and fulness for some seconds, and is said "*to pit on pressure.*" The fluid in ascites may appear suddenly, and the abdomen of the patient may be distended in a few hours, or the fluid may take weeks or months to accumulate.

Diagnosis.—Ascites is readily distinguished in the male from every other intumescence of the abdomen by the fluctuation on percussion. In the female it may be confounded with pregnancy, or with *ovarian dropsy,* which consists in the accumulation of fluid in one or more cysts, generally multilocular, within the substance of the ovary, or in a serous cyst connected with the uterine appendages. The grounds of diagnosis may be thus shortly stated:—(1.) The uniform symmetrical appearance, and general increased breadth across the flanks, possessed by the abdomen in *ascites* from the first, contrasted with the general *one-sided* growth of an ovarian tumor, which has grown up from the pelvis. (2.) Percussion gives useful diagnostic results when performed in the different positions in which a patient may be placed. In ovarian dropsy dull sounds are fixed and invariable in one place, whatever position is assumed by the patient. The dulness is chiefly in front of the abdomen, and may extend more to one side than another, the flanks being tympanitic. In ascites the dull sound follows the gravitating fluid in all positions. "When fluctuation is present, but it is doubtful whether it is ascitic or ovarian, an assistant's hand pressed edgeways into the median line over the tumor will break the wave of ascites, and thus clear up the doubt" (CHRISTOPHER HEATH). The development of hydatids in the abdominal cavity may also simulate ascites, and so may an over-distended bladder.

The **Prognosis** in young persons not labouring under any organic disease, is always favourable; but if it be consecutive to organic disease, a fatal termination is ultimately to be feared. No case is free from danger, the peritoneum may be so much impaired that absorption ceases to be effected through it. When it depends on moderate inflammation of the peritoneum it is often recovered from, especially if the inflammation depends on exposure to cold or to paludal poison. Ascites with albuminous urine, arising from mere functional disorder of the kidney, is generally recovered from; but if the structure of the kidney be impaired, the disease is always grave, and generally fatal. Ascites from disordered function of the heart may be recovered from; but if it depends on diseased structure, either of the heart or large vessels, temporary amendment may take place, but the patient fails to recover. Ascites depending on diseased structure of the liver or of the spleen is rarely recovered from, unless the primary disease be cured.

Treatment.—When ascites occurs without any obvious organic cause, and without albumen in the urine, the best remedy is the *bitartrate of potash,* administered in divided doses, as one drachm three times a day, or every six hours; or in one large dose, as half an ounce, combined, if the patient's bowels be confined, with ten to fifteen grains of *jalap.* When the smaller doses are used, it may be useful to add ten grains of the *citrate* or *tartrate of iron* to each dose. If these remedies should fail, one-sixth to half a grain of the *extract of elaterium* every night, or every other night, may be given. When ascites is combined with anasarca, *squills* afford most relief. Five to eight grains of the *pulvis scillæ,* three times a day, generally relieve the dropsy. If the stomach be irritable, *half a grain of opium* should be added to each dose. When the ascites arises from disease of the heart, the kidney being sound, and the urine free from albumen, the treatment must have reference to the nature of the cardiac lesion. If the valves of the heart are diseased, the patient may be greatly relieved by the administration of tonics, stimulants, and saline or drastic purgatives. An *ounce and a half of camphor mixture, with a drachm* of the *spirit of nitrous ether, fifteen minims* of the *tincture of hyoscyamus,* and *a drachm* of the *sulphate of magnesia* will form a draught which, taken *three times* a day, may greatly reduce the dropsy. The *tincture of squills* (♏x. to ♏xx.), with a *drachm* of the *acetate of potash,* has occasionally succeeded. Small doses of *elaterium,* as one-eighth to one-fourth of a grain three times a day, is a medicine that is also sometimes useful. Should the liver be inflamed or hypertrophied, without other alterations of structure, the dropsy may disappear with the cure of the hepatic disease. Bleeding by leeches, if the hepatic lesion be due to the congestion of inflammation, and the neutral salts, such as the *sulphates of magnesia* or *of soda,* or moderate doses of *calomel,* may give relief. If due to a cirrhotic liver *paracentesis abdominis* ought to be repeated as a *systematic* method of treatment (F. T. Roberts). Abdominal pains are relieved most effectually by fomentations. The bowels are often greatly constipated, and require drastic purgatives, as the *black draught, castor* or *croton oil,* or even *elaterium.* n ascites depending on enlarged spleen when it is simply hypertrophied, the *romide of potash,* and the *iodide of potassium,* in doses of five to eight grains hree times a day, have been found useful. My friend and colleague, Professor Maclean, has found that rubbing the *biniodide of mercury,* in the orm of an ointment, in the proportion of 15 grains of the *biniodide* to one unce of *lard* or *vasaline,* on the skin over the region of the enlarged spleen, has a marked beneficial effect in reducing the enlargement. The ointment, in a piece about as large as a nutmeg, is to be rubbed into the skin while the patient sits before a strong fire, or in the rays of an Indian sun; in the evening of the same day, half the amount is to be rubbed in lightly; and the ointment is not to be repeated at a less interval than 14 days. The invalided soldiers suffering from splenic enlargement invariably ask for a supply of the remedy when they go from hospital. The *biniodide of mercury,* similarly used, has been of great service in reducing the swelling of a goitre. The dropsy which occurs in young chlorotic women, in whom the urine *contains albumen* (the kidney being healthy in structure though disordered in function), is generally curable,—the most efficient remedy is the *bitartrate of potash* in *drachm* doses three times a day, combined with iron.

SECTION XI.—DISEASES OF THE STOMACH.

GASTRITIS.

LATIN EQ., *Inflammatio;* FRENCH EQ., *Gastrite;* GERMAN EQ., *Magenentzündung;* ITALIAN EQ., *Gastritide.*

Definition.—*Forms of inflammation tending to exudations and destruction of parts, or condensations of tissue, especially about the pyloric opening.*

Pathology.—(*a.*) *Causation.*—*Idiopathic gastritis* is an exceedingly rare disease. When gastritis occurs, it is generally the consequence of direct injury from irritant or corrosive poisons. The simplest and most frequent form of inflammation of the stomach is that which is brought about by excess in eating or drinking, especially of alcoholic drinks. It is characterised by active congestion and an excessive secretion of mucus—a condition described under the name of *gastric catarrh,* which may be either *acute* or *chronic.* In the *acute* form the mucous surface is reddened in spots by a fine injection, its tissue relaxed, and its surface covered by an abundant secretion of tough, clear, whitish bile, or blood-tinged mucus. The following are the conditions under which the lesion is apt to be developed (NIEMEYER):—(1.) In *fever patients,* when the secretions of the stomach are diminished. (2.) In debilitated and badly nourished persons. (3.) Excess in drinking spirits. (4.) One attack predisposes to others. The exciting causes are—(1.) Overloading the stomach with food, symptoms of acute catarrh generally appearing on the day following. (2.) The use of food difficult of digestion. (3.) The use of substances as food which have already begun to decompose before entering the stomach, such as spoiled meat, new or sour beer. (4.) Irritation from hot or cold substances, or from alcohol or spices. (5.) Use of substances which weaken the action of the gastric juice, and retard the movements of the stomach. Alcohol acts injuriously in this way, so does opium, and smoking tobacc before meals. (6.) The effects of cold.

(*b.*) *Morbid Anatomy.*—The morbid changes which are known to occu in the stomach are still very imperfectly connected with the expression o clinical phenomena. The morbid states of the stomach of the natur of inflammation, ascertained after death to have existed during life, and in some measure to have been expressed by certain symptoms, are—(1.) *Softening of tissue;* (2.) *glandular degeneration of the proper mucous substance;* (3.) *congestion.* There is a rare form of inflammation, described by Dr. Budd, in which lymph is effused into the submucous areolar coat. Hardening and contracting, it forms a dense gristly mass, which binds the mucous membrane to the parts beneath. Round the pyloric orifice such a morbid state often acts as a permanent stricture—*fibroid degeneration.* It almost invariably results from spirit-drinking, seldom occurs before the age of forty, and must be distinguished from cancer of the pylorus.

(1.) *Softening of the stomach* was first announced by Hunter, and confirmed by the experiments of Spallanzani, Wilson, Philip, and Carswell. Under certain conditions as to temperature and properties of the gastric juice the stomach, especially at death, and immediately after that event, may be dissolved or digested by the secretions from its own secreting glands. In certain diseases of a catarrhal kind, potent gastric juice is secreted by the empty stomach; or lactic acid, being freely generated from the saccharine principles of food, forms with the mucous membrane an efficient digesting mixture. Not unfrequently a softening of the stomach may be predicted with tolerable certainty by a peculiar train of symptoms, resulting from the presence of free gastric juice, or of a digesting acid, in the otherwise healthy stomach (Dr. JOHN GAIRDNER, of Edinburgh, BUDD). The softening does not take place till after death (ANDRAL, BUDD). It is apt to occur where there has been much cerebral disturbance, increasing debility and emaciation before death, as in *typhoid fever, cancer of the uterus, or peritonitis,* and in infants who die of *tubercular hydrocephalus;* in deaths from exhaustion, from inflammatory diseases of the brain, when vomiting is a constant symptom; in persons who die from phthisis and from ulcer of the stomach.

The **Symptoms** from which softening may be predicted are, that when, along with any of these diseases, there is much disorder of the stomach, such as pain and tenderness at the epigastrium, loss of appetite, thirst, frequent vomiting of acid fluids, and nausea, the lesion may be found after death. In the common form, the mucous membrane towards the splenic end is thin, and for the most part stained by hematine, very slippery, and appearing as a dark film gliding over the submucous tissue. The tubes appear under the microscope to be altered, chiefly by solution of their epithelium, while dark grains of melanic matter are deposited between them.

(2.) *Granular Degeneration of the Proper Mucous Substance* is by far the most common organic lesion—existing in 72 out of 100 cases (Dr. HANDFIELD JONES). It is expressed in a variety of forms, the most marked of which are—(1.) Peculiar morbid changes in the tubes, probably analogous to those that occur in the tubes of the kidney in Bright's disease, where the molecular contents of enlarged epithelial cells increase, ultimately leading to complete destruction, by filling the tubes with the debris; (2.) melanic and fatty deposits in the epithelium; (3.) interstitial deposit of nuclear and fibroid exudation at the expense of the gland-substance; so that hypertrophy of tissue may exist along with—(4.) atrophy of the tubular epithelium of the tubes themselves, and of the solitary glands—a form of cirrhosis generally associated with alcoholism (Dr. Wilson Fox). Thus, on the one hand, there may be an utter destruction of the tubular glands—actual loss of substance without replacement; on the other hand, there is no actual loss of substance, but a replacement of elements by granular matter within the tubes, without diminution or alteration of their form. These partial degenerations, even when they extend over a considerable portion of the stomach, do not materially interfere with the ultimate digestion of food. Any deficiency of stomachal digestion is compensated for by increased vigour of intestinal digestion (Dr. Handfield Jones). It is rare to find any evidence of the existence of such lesion during life, even when the degeneration is very extensive. I believe, however, from observations upon the intestinal mucous membrane in analogous glandular degeneration, that in cases characteristic of intestinal wasting, associated with anæmic states, the mucous membrane throughout the whole alimentary tract is similarly affected, and that an examination of the mucous membrane of the lips and buccal membrane of the mouth, indicates with great probability the change below, just as the condition of the tongue is the index from which we judge generally of the functional state of the alimentary canal. The general symptoms of such cases are anæmia and debility, without any obvious organic cause, associated with vomiting or nausea in the morning, no desire for food, and a sensation as if it never was effectually swallowed, but stuck at the diaphragmatic entrance of the stomach, causing the peculiar feeling of weight which attends indigestion and the abundant generation of gaseous fluids. Such symptoms indicat a participation of the whole alimentary tract in the lesions, and where th lesion is merely confined to the stomach, the absence of such symptoms is sufficiently accounted for by the compensation to stomachal digestio afforded by a healthy intestinal tract.

(3.) *Forms of Congestion* may be *passive* or *active*, associated with inflammatory lesions. Blood ceasing to flow freely through the lungs is throw back upon the right side of the heart; blood ceasing to flow freely throug the liver is thrown back upon the stomach, upon the one hand, or upo the spleen by the gastro-epiploic vessels, expressed by congestions of thes viscera; or, on the other hand, the portal system being obstructed, th blood reverts to the intestines, and expresses such an occurrence by ascit as well as by gastric venous congestion. Any mere mechanical impedime therefore, which prevents the blood returning from the stomach towar the heart will induce congestion of the stomach. The immediate effec of such congestion are expressed by *hœmatemesis*, or the vomiting of bloo the secretion of the gastric juice also is diminished, the stomach can dig less food, and requires longer intervals of rest between the meals. T persistence of such passive forms of congestion further leads to the depo tion of melanic matter, to disintegration of the tubular glands of t stomach, and the formation of ulcers in its mucous membrane; just varicose ulcers are established in the limbs from the passive congest which attends varicose veins in the lower limbs. Besides congest from mechanical impediments, there is a congestion of the stomach descri as *vicarious*—that is, as taking the place of congestion which ought to oc in other parts—as in women from stoppage of the catamenia. It been observed to occur in yellow fever, in malignant cholera, and in typhoid fevers. Congestion also arises during the development of gastritis.

Symptoms.—Abnormal states of the stomach are expressed by—(1.) *Vomiting, associated with lesions of other organs.* (2.) *Deficient secretion of gastric juice.* (3.) *Fermentive processes* (*alcoholic, butyric, or lactic*), *tending to the development of entophytes, such as sarcinæ.* (4.) *Indigestion, associated with and depending upon*—(*a.*) *Morbid states of those viscera which are conjoined with the stomach in the processes of digestion, such as the liver, pancreas, and small intestines;* (*b.*) *imperfect action of the kidneys, as in Bright's disease;* (*c.*) *defective or diminished morphological changes during the processes of nutrition in the tissues, generally expressed by altered secretions and excretions, as in many constitutional diseases;* (*d.*) *indigestion, associated with pyrosis and increased secretion of the juices of the stomach and salivary glands, and with cutaneous disorders, such as urticaria;* (*e.*) *indigestion, associated with drunken habits.* The diseases of the stomach with which one or more of these organic or functional states may be associated are,—*Gastric catarrh and gastritis, chronic ulcer, hæmatemesis, perforation, dilatation, stricture, gastric fistula, hernia, cancer, colloid tumors* (non-malignant), *sarcinæ, injuries, laceration, dyspepsia, gastrodynia, pyrosis.*

The symptoms of *gastric catarrh* usually take the form of a "*disordered stomach,*" expressed by headache, especially across the forehead, increased on stooping, associated with flashes before the eyes, and a sensation of tightness, as if the head would burst. Nausea and sickness exist, with sensations of heat and of cold, distaste for food, the sight or smell of which is apt to produce sickness, retching, and vomiting. The food already in the stomach undergoes an abnormal decomposition; lactic and butyric and acetic acids are produced, and fetid gases are set free as a result of these fermentive processes, with eructations into the mouth of sour and rancid matters from the stomach. The tongue is generally coated with a white creamy fur; the odour of the breath is offensive, the mouth feels slimy, and the taste is bad. *Gastritis* from poisoning causes general depression, so great in some cases as to simulate perforation. The pain generally spreads from the epigastrium over the abdomen, accompanied with vomiting of mucus or of bloody mucus, which may be followed by purging of similar evacuations, preceded by severe colicky pains, and followed by collapse, small pulse, cold skin, and clammy sweat. There are then grounds for suspecting that poison may be the cause of such symptoms (NIEMEYER).

Treatment.—Emetics may be required in cases where the stomach has been overloaded; and it is certain, from the gases and fluids causing prominence over the stomach, that it contains decomposing food. One scruple of *ipecacuanha*, with one grain of *tartrate of antimony*, is the safest and most efficient emetic. When injurious matters have passed into the bowels, causing flatulence and colicky pains, mild laxatives may be required, such as *rhubarb;* or, *compound infusion or mixture of senna*, or *fluid magnesia*, in small doses, may be given every hour or two hours, followed by five to ten grain doses of *bicarbonate of soda.* In *chronic* or *passive* congestion of the stomach, and in *chronic gastritis*, the advantage of *nitrate of silver* has been advocated by the late Drs. James Johnson and Symonds, and more recently by many other physicians. *Argent. Nitrat.*, gr. ½, *Ex. Hyoscyam.*, gr. ii. to gr. iv., made into a pill, may be continued every night, the quantity of the nitrate being gradually increased to two or three grains daily. It may not be continued, however, beyond six or eight weeks. Or *Argent. nit. Crystal*, gr. ss. to gr. i., *Aq. distilat.*, *f*℥ss.; may be taken at bed-time—the stomach being empty—and repeated every night, or every second, third, or fourth night, according to the severity of the gastric symptoms. I have found *Seller's Mixture of Bismuth* of great advantage; and *alkalies* combined with *morphia.* In cases of *acute gastritis,* mercurial purgatives by *calomel* are of service. Three to five grains may be given to an adult; followed by a dose of *castor-oil* or the *compound senna mixture* of the pharmacopœia. Where it is not desirable to act so searchingly on the small intestines, *blue pill* with *compound colocynth* or *rhubarb* pill in equal parts (*a a* gr. ii.), combined with one grain of *ipecacuanha* powder, is a mild and gentle laxative. *Iced water* to drink in small quantities, or small pieces of ice in the mouth, tends to allay thirst, and to appease pain; and the continuous use of hot water fomentations over the region of the stomach, as hot as the patient can bear them, is of great benefit (BRINTON).

CHRONIC ULCER OF THE STOMACH.

LATIN EQ., *Ulcus Longum;* FRENCH EQ., *Ulcère Chronique;* GERMAN EQ., *Chronisches Geshwür;* ITALIAN EQ., *Ulcera Cronica.*

Definition.—*Inflammation of the stomach, ending by loss of substance in the form of an ulcer.*

Pathology.—*Simple, chronic,* and *perforating* ulcers are described. The simple ulcer is generally solitary and deep, seldom larger than a shilling, circular or oval in shape, its edges clean, sharp, and well defined, as if punched out. It is generally situated along or near the lesser curvature, and usually nearer the pyloric than the cardiac orifice, and more frequently on the posterior than the anterior wall. It is more common in women than in men, nearly in the proportion of two to one. It is especially a disease of middle and advanced life. It is relatively more frequent amongst the poor than amongst the rich; and is especially found in maid-servants between the ages of eighteen and twenty-five. It tends to prove fatal by—(1.) *Perforation;* (2.) *hæmorrhage;* (3.) *exhaustion.* When the ulcer eats its way through, the aperture gradually narrows to a point, which corresponds to the centre of the ulcer as it reaches the peritoneum. The peritoneum inflames at this point, and perforation is completed by the separation of a slough. An important sanitary effort is here made by adhesion of the peritoneum surrounding the slough to some opposed surface, such as the liver, pancreas, colon, or abdominal wall. Fistulous openings may be thus established, so that food escapes from the stomach when such passages communicate with the alimentary canal. A most comprehensive record of such cases exists in *The Edinburgh Monthly Journal* for July and August, 1857, by the late Dr. Murchison. About 13·4 per cent. of cases of ulceration terminate by perforation; and the liability to perforation decreases as life advances (BRINTON). During the ages from fourteen to twenty perforation is most frequent (CRISP).

Hæmorrhage, while it is one of the most frequent and important symptoms of ulcer, is also a mode of fatal termination. It generally occurs soon after a full meal; and about from 3½ to 5 per cent. of the cases prove fatal in this way.

Symptoms of Gastric Ulcer.—"There is disturbance of gastric digestion: at first mere uneasiness and pain; then nausea and vomiting, or regurgitation, that expel the food previously taken; or a tasteless or acid watery secretion. At this stage it is sometimes cut short by perforation, with its sequel of fatal peritonitis. Failing such an accident, the dyspeptic symptoms are complicated by hæmorrhage from the stomach; sometimes a sudden and dangerous gush, oftener a slow and intermittent drain of blood. The anæmia thus produced is generally associated with a cachexia independent of it, the result of inanition by frequent vomiting of food, or by large destruction of the gastric mucous membrane, and consequent impairment of its function. In young females, more or less complete amenorrhœa may be associated with ulceration, with hæmorrhage, or with both. Retaining the liabilities to death by perforation, by hæmorrhage, by vomiting, and by exhaustion, the lesion may end by one of these modes of dying, or by two or more of them in combination. A spontaneous subsidence of these symptoms, in something like the inverse order of their occurrence, may announce a recovery; or a similar amendment may be effected by careful medical treatment" (*Med.-Chir. Review*, p. 159, July, 1856).

The character of the pain is peculiar, at first being little more than a feeling of weight or tightness, dull and continuous. It gradually becomes intensified into a burning or gnawing sensation, which produces a kind of sickening depression. It comes on from two to ten minutes after the ingestion of food, and remains during one or two hours, corresponding to the period of gastric digestion, after which it gradually subsides, or, if vomiting empties the stomach, it invariably ceases. The pain is expressed at the centre of the epigastrium, or at the middle line of the belly, immediately below the extremity of the ensiform cartilage, often confined to a mere spot, and rarely to a space more than two inches in diameter. A dorsal pain is also subsequently established, generally in a few weeks or months after the epigastric pain. It is expressed by a gnawing sensation, interscapular, or from the spine of the eighth or ninth dorsal to that of the first or second lumbar vertebra. Pressure in the epigastric region is sometimes unbearable, and, for obvious reasons, must be applied with the utmost care and delicacy. *Vomiting* usually occurs when the pain reaches its height, and, completely emptying the stomach, generally affords relief (BRINTON).

Treatment.—Chronic ulcers are to be treated by rest to the stomach, and by dietetic rather than by medicinal means. The patient must eat in small quantities, especially of milk compounded with arrow-root, macaroni, semolina, sago, tapioca, biscuit powder, barley meal, Indian meal, or oatmeal gruel. *Iced milk,* combined with one-quarter to one-third of *lime-water,* is particularly recommended, two or three table-spoonfuls of which are to be taken at short intervals; so that about two quarts of milk may be thus used during the day, rendering regular and more bulky meals unnecessary. The lime-water tends to prevent coagulation, and the milk more readily passes unaltered into the intestines, to be digested by them rather than by the stomach. The most complete rest possible ought to be given by stopping the supply of all nutriment by the mouth, and supporting the patient for several days by nutritive enemata. Perfect quiet in the recumbent posture must be observed, the lips and tongue being moistened from time to time by a little water. The patient may thus be kept for eight or nine days, or even longer, on nutrient enemata alone, when the pain, the irritability of stomach and of the system will cease. The substances most useful for enemata are *milk*, strong unsalted *beef-tea*, *raw eggs beaten up in milk*, occasionally a little brandy, and (generally in two enemata daily) ten or twenty minims of *tincture of opium*. The enemata should be as small as possible, from two to six ounces only at a time. The interval to the stomach thus gained of complete rest is of the greatest importance towards success in the treatment of gastric ulcer; and, combined with the restricted diet described, will usually effect a cure in three weeks (Dr. BALTHAZAR W. FOSTER, of Birmingham, *Brit. Med. Journal*, June 3, 1865). Next to dieting, *blood-letting* by two or three *leeches*, applied to the region of the stomach about twice a week, affords obvious benefit. *Blistering* applied to the spine may relieve the dorsal pain. Small lumps of *ice* may be swallowed if the stomach is irritable; and astringent remedies, especially the *salts of metals*, such as a combination of *iron* and *alum*, are of use; and *gallic acid*, *nitric acid*, and *bitter barks* may be administered. Constipation is to be counteracted by enemata. When hæmorrhage occurs, small bits of *ice* may be swallowed, and rest maintained in the horizontal posture. Astringent medicines are to be administered, such as *oil of turpentine*, *acetate of lead and opium*, *alum and tannic acid*. *Oil of turpentine* should be given in doses varying from ten to twenty minims in *cold* water, and repeated more or less frequently according to the urgency of the symptoms. *Ergot* may also be employed. When hæmorrhage is copious, a mixture containing ten minims of *diluted sulphuric acid* and five grains of *gallic acid* in water, taken every two or three hours, is the best remedy (Dr. B. FOSTER). Vomiting may be relieved by *dilute hydrocyanic acid* (five minims), combined with *bicarbonate of soda* (twenty or thirty grains) in solution, repeated every two or three hours. The *subnitrate of bismuth*, in doses of ten to twenty grains, every six or eight hours, alone or combined with five to ten grains of *compound powder of kino*, has a remarkable effect in relieving pain, vomiting, and diarrhœa (BRINTON). *Seller's bismuth mixture* is also an efficient remedy.

HÆMATEMESIS.

LATIN EQ., *Hæmatemesis;* FRENCH EQ., *Hématémèse;* GERMAN EQ., *Bluterbrechen;* ITALIAN EQ., *Ematemesi.*

Definition.—*A discharge of blood from the stomach.*

Pathology.—(*a*.) *Causation.*—Hæmatemesis may arise from causes peculiar to the stomach, as from the effect of vomiting, or from a blow or from injuries, as by fish-bone or other sharp substances. The following is a summary of causes of hæmatemesis:—(1.) Rupture of over-filled blood-vessels, without change of texture. (2.) Venous congestion of gastric mucous membrane. (3.) Impediments to circulation in the liver. (4.) Obstruction of the portal veins by blood-clots. (5.) Pressure of branches of the portal veins, from cirrhosis or enlargement of the gall-ducts. (6.) Pigmentary embolism, from plugging of the capillary vessels of the liver with masses of pigment. (7.) Yellow atrophy of the liver destroying the capillaries. (8.) Rupture of diseased vessels and bursting of varices or aneurisms. (9.) Hæmorrhagic and scorbutic conditions. (10.) Injuries to walls of blood-vessels. In military service in the field the thirsty soldier sometimes suffers from this affection, in consequence of drinking incautiously water containing leeches. New-born children are subject to hæmatemesis from the day of birth till about twelve days old. Except at this early period hæmatemesis is rare till puberty. Both sexes are liable to it; but women suffer more frequently than men, and especially those who are either pregnant or labour under amenorrhœa.

(*b.*) *Morbid Anatomy.*—On opening the stomach of a patient that has died of hæmatemesis, blood may be found in various degrees of consistency, from a pure liquid black or brown to a solid coagulum; and also in the œsophagus and in the intestines. The internal surface of the stomach is almost always coated with a layer of viscid mucus of a red colour, which separates it from the clot. The quantity varies, so that the stomach has been found distended to the utmost, the blood forming a perfect mould of its cavity. In general, the mucous membrane of the stomach is hardly stained, but it is congested, and in some few spots ecchymosed —blood being infiltrated into the subcellular tissue. But even after the most abundant hæmorrhage from the gastric mucous membrane, the source of the bleeding may not be found. The hæmatemesis may be a consequence of ulceration of an artery (coronary), or vein. Sudden death may thus result from ulceration of the stomach involving several vessels. The most frequent cause of ulceration of the blood-vessels, however, is cancer. Hæmatemesis may also be symptomatic of a tumor of the pancreas, or of an enlarged kidney compressing the aorta; or an aneurism of the cœliac artery, which, obstructing the hepatic and splenic arteries, may cause the greater portion of the blood conveyed by those arteries to pass through the gastric artery, thus causing congestion of the mucous membrane of the stomach. Sometimes in symptomatic hæmatemesis the blood thrown up has come from an aneurism of the aorta bursting into the stomach.

Symptoms.—*Hæmatemesis* may be acute or chronic, the chronic form being usually termed *melæna.* The acute form of hæmatemesis may be sudden in its attack, or may be preceded for a few hours by shivering, heat, weight and oppression at the epigastrium, by nausea, headache, and by pains between the shoulders. The buccal and pharyngeal membranes are also said to be sometimes congested, and the gums swollen. A swollen state of the liver or spleen may exist. At length hæmatemesis occurs, and a quantity of blood, black, clotted, and mixed with alimentary matters, is thrown up, sometimes streaming both from the nose and mouth. The symptoms which follow are proportioned to the quantity of blood lost, and are nearly the same as in hæmoptysis.

If the quantity be small, the pain in the epigastrium ceases, and the patient is relieved; if larger, the patient is in some degree relieved, but greatly exhausted; while, if the quantity thrown up, as it often is, be so abundant as to half fill a wash-hand basin or a chamber vessel, the patient becomes pale, a cold perspiration runs down his face, he has an overwhelming sense of sinking, and his pulse becomes frequent and weak. There are instances in which hæmatemesis has proved suddenly fatal. In the greater number of cases a few hours have scarcely elapsed when the epigastric and dorsal pains are renewed, the thirst and shivering return, and the vomiting recurs, often perhaps four or five times in the space of two or three days; a sensation, as of a burning liquid in the stomach, often precedes these subsequent attacks.

Hæmorrhage from the stomach may take place without vomiting, the blood passing into the duodenum, and being ejected by the intestines. The *colour* of the blood thrown up varies according to the time it has continued in the stomach. If poured out rapidly, and immediately rejected, it is often arterial; but accumulated slowly it is of a blackish brown, and clotted. Sometimes a thin layer of coagulated blood forms, which, when thrown up, has been mistaken for a portion of the mucous membrane of the stomach. The chronic form of hæmatemesis has been termed *gastro-melæna.* The blood is not poured out pure, but undergoes some change, so that it resembles chocolate or coffee grounds, and is, in fact, a species of black vomit. This affection usually occurs as the last stage of many diseases, especially if the patient be of a broken and worn-out constitution. The quantity thrown up is often large, amounting to a pint or two in the course of the day, and this may last for several days.

Diagnosis.—The chief difficulty in the diagnosis of *hæmatemesis* is to distinguish it from *hæmoptysis.* The burning heat of the stomach, the black pitchy stools, the absence of cough, and of all the signs furnished by auscultation, sufficiently distinguish it from hæmoptysis. The colour of the blood from the stomach likewise is generally black, dark, clotted, and may be mixed with food, the coagula containing no air, and generally acid in reaction; while that from the lungs is more commonly arterial, bright-red, frothy, and mixed with mucus; not coagulated, and if so, the coagulum generally contains air, and in reaction alkaline. The quantity is also in general greater from the former than the latter viscus, although there are many exceptions to this rule. It should be remembered, also, that blood may pass from the nose into the stomach during sleep, or from the gums or tonsils after lancing. This disease is one of those, also, most easily and most commonly feigned. A microscopic examination will determine whether the matter ejected is blood. The red juice of fruits has been mistaken for blood. The matters thrown up in melæna bear no resemblance to the fluids ejected from the lungs.

Prognosis.—*Hæmatemesis* is devoid of danger when it is *vicarious* and arises from pregnancy, from amenorrhœa, and from suppressed hæmorrhoids When, however, it arises from organic disease of the stomach, from disease of the liver, spleen, or heart, it is always of grave import, although perhaps not immediately fatal. When, also, it is the result of the action of a morbid poison, the danger may be imminent. In melæna the case i always dangerous, but some recover.

The **Treatment** of the acute forms of hæmatemesis is similar in many respects to that of *hæmoptysis.* The great volume of the arteries of the stomach, and their origin almost immediately from the aorta by means of the cœliac artery, are reasons which have been alleged for this affection being but little influenced by general or local bleedings. The vast amount of blood sometimes lost by hæmatemesis renders it necessary to support the patient by acid wines much sooner and to a much greater extent than in hæmoptysis. In melæna the only chance for the patient is a liberal support by wine diet and medicines, and by *opiates* to quiet the stomach. In cases of *hæmatemesis* from *cirrhosis,* or other obstruction to the circulation through the liver, and in the vicarious hæmatemesis of women, *leeches* to the anal region or *os uteri,* are of service. The action of ice, by swallowing small pieces from time to time, is very beneficial, covering at the same time the epigastric region with ice-cold compresses, which require to be renewed frequently. Solutions of *dilute* or *aromatic sulphuric acid,* in doses of ten to twenty minims, or of *alum,* are of service. *Alum* may be given in doses of eight to twelve grains, combined with Batley's solution of *opium,* or with *laudanum* or *sulphuric acid.* In *chronic hæmatemesis, acid. gallici,* gr. x.; *acid. sulph. dil.,* ♏ x.; *aq. distil., f* ℥i., has been recommended by Dr. Brinton. Solution of *pernitrate of iron,* in doses of thirty to forty minims, may be of use. *Ergot* may also be of service (see page 742, under "Hæmoptysis"). *Tannin* is of service, as one of the best astringents in the form of a pill (O. Rees)—three to six grains twice or thrice a-day. The patient must maintain absolute rest in bed, and must not rise to stool. *Ipecacuanha* in doses to produce nausea is of service in arresting hæmorrhage, and restoring heat to patients in the collapse of hæmatemesis (Osborne, Trenor, Graves), in doses of one to two grains every fifteen or thirteen minutes till nausea is felt, when the remedy is to be discontinued, as vomiting is to be avoided. If stimulants are required, *iced champagne* may be given; and when the vital powers are much depressed, *ammon. hydrochlor.,* ʒiss.; *acid. hydrochlor., f*ʒss.; *decoct. hordei co.,* oz. i., may be taken every second or third hour (Copland), in doses of two or three teaspoonfuls.

DYSPEPSIA.

Latin Eq., *Dyspepsia;* French Eq., *Dyspepsie;* German Eq., *Dyspepsie;* Italian Eq., *Dyspepsia.*

Definition.—*Impairment of digestion, arising without perceptible change of structure or lesion of the stomach.*

Pathology.—*Causation.*—As an abnormal functional difficulty the impairment may be due—(1.) To sympathetic relations with other organs themselves in a morbid state, explained by the phenomena of "*reflex action;*" for example, the *nausea* and sometimes *vomiting* which attends irritation of the *lung, brain, liver,* or *uterus.* (2.) To a scanty secretion of the gastric juice, characterised by slowness of digestion and long retention of food by the stomach, prolonged distress after eating, especially weight and uneasiness at the pit of the stomach; decomposition of food in the alimentary canal; the evolution of fetid gases (*pnuematosis*), and the appearance of unaltered ingesta in the stools. Gastric digestion should occupy, on an average, from two to three hours. The dyspepsia of slow digestion may be an *inheritance,* and the conditions which tend to maintain it are—mental over-exertion, prolonged anxiety, especially after meals, gluttony, drunkenness, sedentary habits, and the consumption of more food than the system requires (LEARED). (3.) To the *abnormal quality* of the gastric juice. (4.) To diminished movements of the stomach, so that the ingesta are not sufficiently mixed with the gastric juice (NIEMEYER).

The **Symptoms** brought about by diminished secretion of the gastric juice are similar to those of chronic catarrh and chronic gastric ulcer. The appetite is lessened, and easily satisfied; but however little may be eaten, gas is rapidly generated, and sour, fetid, or rancid eructation of liquid occurs. The patients, suffering from flatulence and consequent distension, are disturbed and depressed. Palpitation of the heart is common, as well as irregularity of the pulse, headache, occasional dimness of vision, and attacks of a peculiar form of giddiness described by Trousseau (*vertigo stomicale*), but which Niemeyer believes to be due to psychical causes. Mental depression is often extremely distressing, varying in degree from slight dejection, ill-humour, or irritable temper, to the extremes of melancholia, or suicidal monomania. "Half the unhappiness in the world," writes Sydney Smith, "proceeds from little stoppages, from a duct choked up, from food pressing in the wrong place, from a vexed duodenum, or an agitated pylorus. The deception practised upon human creatures is curious and entertaining. My friend sups late; he eats some strong soup, then a lobster, then a tart, and he dilutes these esculent varieties with wine. The next day I call upon him. He is going to sell his house in London and to retire into the country. He is alarmed for his eldest daughter's health. His expenses are hourly increasing, and nothing but a timely retreat can save him from ruin. All this is the lobster; and, when over-excited nature has had time to manage this testaceous encumbrance, the daughter recovers, the finances are in good order, and every rural idea effectually excluded from the mind. In the same manner old friendships are destroyed by toasted cheese; and hard salted meat has led to suicide. Unpleasant feelings of the body produce correspondent sensations in the mind, and a great scene of wretchedness is sketched out by a morsel of indigestible and misguided food. Of such infinite consequence to happiness is it to study the body" (*Memoir of the Rev. Sydney Smith,* by Lady Holland, Vol. I., p. 125; London, 1855).

In impaired digestion from over-secretion of gastric juice or its abnormal qualities, pain at the orifice of the stomach is the characteristic symptom, named *cardialgia.* The secretion of a fluid abnormally acid causes an unpleasant sensation about the cardiac orifice, sometimes termed *heartburn.* This fluid may be regurgitated into the mouth. It has a most disagreeable oily, acid or rancid taste, and not only sets the teeth on edge, but, expectorated on any carbonated alkali, causes effervescence. It is said to be principally *lactic acid.* The effects are more or less pain in the stomach, accompanied by distressing flatulence, derangement of the bowels, headache, disturbed sleep with terrifying dreams. The remote effects are—palpitation, gravel or stone, or a gouty or rheumatic state of the constitution, or *uric acid diathesis*, for the urine, small in quantity, is loaded with the lithates (Dr. PROUT). Alterations in the free acidity of the urine are most important signs. In dyspeptic cases attended with torpid digestion, or with acid vomiting, the urine may be more alkaline than usual; so that there may be abnormal acidity as well as abnormal alkalinity in some cases. When the urine is more acid than usual after food, it is also often scanty, and deposits urates and oxalates of lime—conditions associated chiefly with cardialgia, nausea, and frontal headaches. Chloride of sodium is small in amount when digestion is imperfect; and the more freely it is voided in the urine, the more perfectly has digestion been carried on. Oxalates of lime-crystals are common in dyspepsia (PARKES, l. c., p. 333). This form of dyspepsia most commonly occurs in those who live high, eat largely of rich black meats, and drink largely of malt liquors or champagne, which, acting as ferments in the stomach, turn acid, and dispose everything else to undergo the same changes. Some persons, especially those descended from gouty or rheumatic parents, have an idiopathic tendency to this form of impaired digestion. In them the most opposite substances will produce it, as sub-acid fruits, salt meats, pastry—indeed, anything that deranges their enfeebled powers. Tobacco has a poisonous principle which greatly favours the occurrence of this disease, and many persons suffer severely after smoking a few cigars, or even one.

Treatment.—Congestion, catarrh, and functional states associated with what are called dyspeptic symptoms, or "imperfect digestion," are the results of stomach diseases for which the physician is called most frequently to prescribe. When there is reason to believe *congestion* exists, a sparing and easily digested diet is to be prescribed, with total abstinence from fermented drinks; and in cases where catarrhal inflammation prevails the blandest food must be given in very small quantities. In severe cases leeches are to be applied over the region of the stomach, and the patient may sip iced water, or suck small pieces of ice, to relieve thirst. In impaired digestion from any cause, a mode of life tending to improve nutrition is to be aimed at. The necessity of strict attention to diet in all cases of impaired digestion is absolute. If fulness and uneasy sensations are experienced after dinner, less food should be taken at that meal, and more at breakfast; the principle being to apportion the amount of food necessary to sustain the body more evenly over the waking hours than is commonly done. The great fault in the dietetic system of this country consists in most people supporting themselves mainly by dinner. This meal is consequently too large; and the quantity taken at dinner should be resolutely diminished till breakfast is appreciated (LEARED, l. c., p. 150).

Special symptoms, common to various morbid states, require special modes of treatment. Excess of acid is best neutralised by *lime-water, magnesia*, or *alkaline* remedies, selected according to the state of the patient's bowels; and the gastric fermentation which is apt to be established may be checked by *brandy* and *various aromatic spirits. Bicarbonate of potash* and *nitrate of potash*, in the proportion of *eight* parts of the former to *one* part of the latter, is useful in cases of habitual acidity; and all these antacid remedies should be taken about three or four hours after a meal. Pills containing from a quarter to half a minim of *creasote*, given with each meal, will counteract fetid eructations. It checks that fermentation in which acetic and carbonic acids are formed; while *conium* and *belladonna* are the medicines which better than *opium* allay general nervous irritability. In cases of slow digestion, with deficient secretion of the gastric juice, the rules of treatment are—(1.) To let albuminoid food be as liquid as possible. Eggs must be eaten when cooked short of coagulation of the albumen. (2.) To let the day's allowance of food be taken in small quantities at regular intervals. (3.) That by the administration of *alkalies* the food may pass to the intestines, and be digested there, rather than by the stomach. This latter mode of treatment by alkalies, recommended by Chambers, is contrary to that recommended by Dr. Budd. Both are consistent with physiological facts, and the course to be followed must be determined by the nature of the case. Both gave large doses of *bicarbonate of soda*—ʒii. dissolved in a pint of warm water—to counteract the excessive acidity, or to promote the passage of food to the intestines. The amount and kind of food taken is of importance to be attended to in cases of slow digestion. If a fair amount of exercise be taken, the following dietary, slightly modified from that proposed by Dr. Leared, will be found appropriate in such cases:—

Breakfast, (8 a. m.)

Bread (stale), . . 4 oz.	Mutton Chop, or other Meat (cooked) free from fat and skin,..3 oz.	Tea, or warm milk and water and sugar, or other beverage, . . . ¾ pint.

Luncheon, (1 p. m.)

Bread (stale), . . 2 oz.	No solids, such as Meat or Cheese.	Liquid, . . . ½ pint.

Dinner, (5 or 6 p.m.)

Bread (stale), . . 3 oz. Potatoes and other Vegetables, . . 4 oz.	Meat (cooked) free from fat and skin, . 4 oz.	Liquid, not more than half a pint.

Tea or Supper (not sooner than three hours after Dinner).

Bread (stale), . . 2 oz.	No solids, such as Meat or Cheese.	Tea, or weak brandy and water, or sherry and water, or toast and water, to the extent of ½ pint.

The quantity of wine or other fermented liquor, and also of animal diet, should be thus reduced till the disease subsides and the urine is healthy. Soups, tea and coffee, drank, as they usually are, boiling hot, debilitate the coats of the stomach, tend to produce dyspepsia, and are abandoned by many persons from their so often exciting *cardialgia*. The best bread for the dyspeptic is the unfermented bread, baked by the process of the late Dr. Dauglish, entirely from wheaten flour of the best quality. It is known as "*aërated bread,*" because carbonic acid gas is substituted for yeast. It is more easily digested than common household bread. Pastry and "*sweets*" must be strictly forbidden. They are "sweet in the mouth, but bitter in the belly." Forms of indigestion marked by excessive acidity and heartburn may be relieved by *bicarbonate of soda,* in doses of fifteen grains, combined with a few grains of *nitre*, taken two or three times a day. At the same time, free excretions from the liver and bowels must be sustained by occasional small doses of *blue pill* or *podophyllin*, combined with extract of *colocynth* and of *henbane*, while exercise and diet are duly attended to. Weakened digestion from over-fatigue may be restored under the use of *carbonate of ammonia*, conjoined with *compound tincture of gentian*, or with *extract of gentian* in the form of a pill. Extracts of *nux vomica* or *strychnia* are also valuable remedies. *Half a grain of extract of nux vomica, half a grain of sulphate of iron, and four grains of compound colocynth pill*, form a combination which, taken early in the morning, or one hour before dinner, generally induces gentle action of the bowels (LEARED). *Compound rhubarb pill* may be substituted in place of the *compound colocynth pill.* Another useful ingredient in a dinner pill I find is *ipecacuanha*, to the extent of one grain, or half a grain in each, in cases of slow or torpid digestion. Indigestion from habitual drunkenness, or where there is great irritability or sensitiveness of the stomach, is best relieved by the pure bitter infusions, such as *gentian, quassia, hops*, and *calumba*, singly, or combined in a mixture, so that a dose may be taken two or three times a day, an hour before each meal. Grave's tonic I have found especially useful. It consists of the *tinctures of quassia, calumba, compound gentian, cinchonia flava*, of each an ounce, and from two to four drachms of the *liquor morphiæ hydrochloratis;* of this mixture a teaspoonful is to be taken one hour before each meal. *Quassia* may generally be taken as a *cold infusion*, and is thus prescribed by Niemeyer:—"In the evening pour a cupful of cold water over a teaspoonful of *quassia chips;* by the next morning a bitter infusion will have formed—to be taken fasting;" or water may be poured into a bowl made of *quassia-wood*, sold for this purpose, and after standing over night in the bowl, the water is to be taken in the morning. *Hop-bitter* is most agreeably taken in the form of the many bitter ales, such as are brewed by Bass, Allsopps, Ind-Coope, Salt, and the genuine Bavarian beer, brewed all over Germany. Such ales must be got direct from trustworthy brewers who brew from hops, and where no injurious substitute is used instead of hops. For other reasons it is necessary to obtain the ale direct from the brewery. Once it passes into other hands, there is no guarantee that some of the numerous processes for "*stretching*," and otherwise increasing the *quantity*, to the disadvantage of the consumer, will not be practised. *Of the extract of malt* originally prepared by Trommer, as a genuine extract, containing the soluble constituents of the malt and of the bitter of the hop, Niemeyer speaks highly in the treatment of cases of *irritable ingestion*," as having been almost the only nourishment the patients could take. Small doses of *opium*, or of *morphia* in an *ammonia mixture*, may be given at bedtime, so as to secure sleep at night, or Grave's tonic, as above. In prescribing the *mineral acids*, the following general ule ought to be kept in mind—namely, that the influence of *sulphuric cid* is astringent, while that of *hydrochloric acid* promotes digestion, and f *nitric acid* secretion (DR. BENCE JONES).

PYROSIS.

Latin Eq., *Pyrosis;* French Eq., *Pyrosis;* German Eq., *Sodbrennen;*
Italian Eq., *Pirosi.*

Definition.—*Paroxysms of pain at the cardiac or œsophageal region of the stomach, occurring in paroxysms which do not cease till the patient vomits up a limpid colourless fluid like water, cold and insipid to the patient's taste, but which sometimes gives an acid and sometimes an alkaline reaction.*

Patholgoy.—*Causation.*—*Pyrosis* (πυροω, to burn) is also known by the name of water-brash, *fer chaud.* It is a painful disorder of the stomach. It is frequently met with in Scotland and in Ireland; and Linnæus writes that one-half of the inhabitants of Sweden are liable to it. From the large quantities of spirits drank in those countries it has been supposed to be caused by their immoderate use. The disorder is more frequent among women than men, in the proportion of five to one. It seldom occurs except in those who live upon a low and insufficient diet. It often exists in connection with some derangement of the uterine or nervous system, or with organic disease of the stomach, or pancreas, or liver. It is not uncommon in advanced life. *Sarcinæ ventriculi* are sometimes found in the fluid ejected from the stomach.

Symptoms.—Pyrosis usually comes on in the morning and forenoon, when the stomach is empty, with a sense of constriction, as if the stomach were drawn towards the back, or with a severe and burning pain. This *gastrodynia* the patient finds increased by standing or sitting upright; and he seeks relief by bending his body forward, and making pressure on the painful part. The attack may last from a few minutes to the greater part of an hour, when a clear, limpid, tasteless fluid is vomited up, varying in quantity from an ounce to a pint. As soon as this fluid is rejected the pain ceases, and the paroxysm is at an end; but it may occur three or fou times a day. When there is only one attack, it usually comes on befor ten o'clock in the morning. The patient's appetite is generally impaired he complains of thirst, his bowels are generally constipated, his surfac pale, and his body emaciated.

The **Treatment** of *pyrosis* consists in saline remedies, such as a drach of the *sulphate of magnesia,* with fifteen minims of the *tinct. hyoscyami,* thre times a day. Many other medicines have been recommended, as the *tin kino,* and *compound kino* powder, and mixtures of *bismuth,* especially Seller' The *compound tincture of benzoin,* to the extent of one fluid drachm, wit mucilage, is most efficacious (Baillie, Symonds). *Sulphurous acid,* doses ♏xxx. to one fluid drachm, thrice daily, and shortly before mea is advocated by Dr. Lawson (*Practitioner,* Sept., 1868). It is especial useful where *sarcinæ ventriculi* are present in the fluid evacuated. T diet should consist of animal food, and be otherwise nourishing. Oatm and brown bread must be avoided.

SECTION XII.—DISEASES OF THE INTESTINES.

ENTERITIS; ENTERIC, INTESTINAL CATARRH, OR MUCO-ENTERITIS; INFLAMMATION OF THE INTESTINES.

LATIN EQ., *Enteritis;* FRENCH EQ., *Entérite;* GERMAN EQ., *Enteritis;* ITALIAN EQ., *Enteritide.*

Definition.—*Inflammation of the small intestines generally, or limited locally.*

Pathology.—(*a.*) *Causation.*—It is due to cold, local irritation, the influence of exanthemata, irritative poisons, and may be the effects of burns and scalds.

(*b.*) *Morbid Anatomy.*—Although *enteritis* is generally described in text-books, it is a rare disease, and seldom affects the intestine throughout its whole extent. Originally the name was used to describe local peritonitis. With some modifications, explained by peculiarities of structure, the *organic* lesions of the lesser intestinal tract are similar to those described in the stomach; while the functional disorders are indicated by the various ways in which the intestines express irritation, such as by *spasms, colic, flatulence, indigestion, constipation,* and various forms of *fluxes* or *diarrhœa.* The lesions generally express themselves in the ileum and duodenum. *Acute diffuse inflammation* is marked by redness, thickening, and impaired cohesion. The redness is of a *deep venous red,* approaching to blackness, either partial or general in extent, and in dotted, arborescent, or striated patches. It is distinguished from mere passive congestion by the increased arterial vascularity of the submucous tissue. The thickening is generally sensible, and often considerable. The impaired cohesion is not so obvious as in the stomach, but the mucous membrane may be removed much more easily than in health from its attachment to the parts subjacent. In the *chronic forms of diffuse inflammation,* the colour, thickening, and cohesion of the gut are not greatly changed; but the thickness is more considerable, the cohesion of parts is often more tenacious, while the dark venous hue, on subsiding, eaves a greyish or slate-coloured tint, from a deposit of melanic matter in he substance of the membrane. *Chronic catarrh of the intestines* is the most common of diseases. It may be the result of the following condiions, namely:—(1.) Obstruction to the hepatic circulation, and especially o the escape of blood from the portal vein. (2.) Lesions of the respiratory nd circulatory organs, which cause obstruction to the emptying of the *ena cava.* (3.) Disturbance to the external circulation, such as accomanies severe inflammations of the skin, as from burns, or from sudden xposure of the skin to low temperatures, as by travelling in cold weather. 4.) As a result of peritonitis. (5.) As a result of mental excitement. 6.) As a result of local irritations from the use of purgatives; of some inds of vegetable food; the passage of undigested decomposing substances om the stomach into the intestines; or the retention of fæcal masses. .) *Lardaceous* disease of the intestine; septicæmia, and cholera (simple nd malignant). The effusion of fibrinous lymph in the small intestines is an extremely rare occurrence. But it is the rule to find such exudation in cases of *scorbutic dysentery*, when the inflammatory processs seems to extend from the great intestine into the small.

The following are the principal results of enteritis:—(*a.*) *Softening of tissue* occurs in the small intestines similar to that affecting the stomach; but it is much less frequent and is not expressed by any recognisable clinical phenomena. (*b.*) *Glandular lesions and degenerations* are of much more frequent occurrence, if not constant, over limited portions of the intestine. These lesions are peculiar, from the structure of the parts; and the *degenerations* are analogous to those occurring in the stomach (see pp. 821 and 822). As in the stomach, so in the intestines there is to be observed in some cases an entire destruction of the gland-tissue, with actual loss of substance, so that when the mucous membrane is dissected from the muscular parts, it may be viewed as a transparent object with a lens. In other cases there may be no actual loss of bulk or of substance, while the structure is nevertheless greatly altered—(1.) By alterations of the contents of the mucous tubes, without change of form; (2.) by an interstitial growth of fibrinous matter encroaching upon and ultimately obliterating the proper gland-tissue. Thus, externally and in bulk the parts may seem to be unchanged; but they are materially altered in specific weight.

Thus, through lesions or degenerations of the gland-tissue, *atrophy* of the mucous membrane of the intestines becomes expressed in three ways, namely,—(1.) *Positive atrophy*, in which the tissue of the gut wastes, while the glandular texture is altered; (2.) the tissue of the gut may not appear to have wasted; on the contrary, it may have gained in bulk, but the glandular tissue has been changed, or altogether replaced; while, (3.) these two conditions may be co-existent.

The observations which go to prove these statements are alterations in the specific gravity of the mucous membrane of the intestine, associated with characteristic but microscopic morbid appearances. These combined modes of examination, not only in mucous membrane, but in all parts in which I have applied the test, have yielded one characteristic result—namely, that atrophic states of prolonged duration, which are generally described as *granular degenerations* of minute tissue, are for the most part o comparatively low specific gravity, with a chemical reaction under th microscope indicative of the presence of fat. On the contrary, th acute inflammatory conditions of tissue have uniformly high specifi gravities where loss of peculiar minute structures (like the glands o the intestines) is due to destruction from exudation and replacement b it. A specific gravity of 1·032 to 1·033 of the mucous membrane, fre from congestion, may be regarded as a healthy state of the gland-tissu In *positive wasting* throughout, it descends to 1·030; while in conditio of *relative wasting*, where bulk is unchanged and glandular parts a displaced or destroyed by exudative deposits, the range of specific weig of parts examined has been as high as 1·044, and in the large intestin as high as 1·050. The specific gravity of Peyer's patches I have fou to vary from 1·032 to 1·044; and in patches from the same intesti there is often a considerable latitude in the range of the specific weigh of the glands. Atrophic states of Peyer's gland are indicated—(1.) a reticulated condition, void of all glandular elements, which seems occur naturally with the advance of years beyond forty; or to occur after extensive *infarction* during the progress of typhoid fever; the elimination of the product thus taking place without ulceration. (2.) By the remains of the cicatrices of ulceration in the form of dark granular deposits; or of a thin, clear, skin-like membrane, of a pale colour, and with a wrinkled contracted border. These changes are associated with a continuance of the complex morbid processes of constitutional diseases, such as anæmia, and organic lesions leading to impairment of the constitution generally; also with the lesions of local glands or accumulations of glands of the gut (such as the solitary lenticular glands and the patches of Peyer, during the progress of some zymotic diseases), as in *cholera*, *dysentery*, and *typhoid fever*. The morbid conditions in which the glandular apparatus may be found are,—(1.) *Intumescence*, *stuffing*, or *cramming* of the gland, by different kinds of deposit (*debris* of normal elements), associated with redness and vascular congestion of the submucous tissue—conditions which correspond to the so-called "*infarction*" of the older pathologists. (2.) *Softening*, *degeneration*, and *elimination* of the abnormal constituents in various ways. (3.) *Ulceration*, sometimes leading to sloughing of whole patches of glands. (4.) *Collapse of solitary glands*, or otherwise cicatrised remains of ulcerations of solitary and aggregate glands. (5.) *Accumulations of melanic matter*, characteristic of pre-existent and long-continued vascular action, with changes in the tissue from hæmatine (*Pigmentary Degeneration;* see Vol. I., p. 127). (6.) *Lardaceous disease* (see Vol. I., p. 129). Generally, it may be stated that *corpuscular* and *fibrinous* elements co-exist in the various forms in which the glands are found intumescent.

There can be no doubt that many of these changes go on independently of any phenomena of inflammation as commonly understood. It is now well known, also, that the results of the inflammatory process may occur without symptoms (such as *pleuritic effusions*), which yield to appropriate remedies. In such cases the inflammation has been considered and described as latent. Does it not, therefore, appear consistent to associate the phenomena of such latent inflammations with the phenomena described by Goodsir and Redfern as the first stage of lesion in the cartilage-cell; with those of the early degeneration in the epithelium of the uriniferous tubes in Bright's disease; with the degeneration of involuntary muscle described by Quain; with the changes described by Virchow in the cornea, by Bucknill and Skae in the brain; with the early change in the epithelium of the mucous surface of the bronchi, where they lose their cohesion, separate with abnormal rapidity, and ultimately assume forms and characters not to be distinguished from pus; and, lastly, with the phenomena of alteration and degeneration of the minute contents of the glandular parts now described; and consider such changes as expressions of one and the same complex morbid process, which Virchow has described by the name of *parenchymatous inflammation?* (see Vol. I., p. 65, *et seq.*)

The **Symptoms** of *enteritis* partake more or less of those belonging to *intestinal catarrh*, combined with pain, aggravated by pressure and pyrexia. Movements from the bowels become more and more frequent. Pain may be absent at first; and its chief seat is generally about the umbilicus, or the right iliac fossa. Discharges from the bowels relieve for the moment the griping pains. The pulse is excited, and is generally full and strong. Acute enteritis is generally accompanied by fever, as in catarrhal fever from cold.

Diagnosis.—The absence of intense pain and tenderness, of vomiting, of constipation, of excessive vomiting and tympanites, of the small and frequent pulse, all of which are characteristic of peritonitis, is sufficient to distinguish enteritis from that disease.

Treatment.—The treatment of *enteritis* and *intestinal catarrh*, when not arising from a morbid poison, is by leeches to the abdomen, gentle purgative medicines, combined with an opiate, fomentations, and purgative or opiated enemata. After the inflammation has subsided, mild tonics, as the *compound tincture of gentian*, with *nitro-muriatic acid*, may be substituted, with the prospect of recovering the lost tone of the parts. When catarrhal diarrhœa exists, if congestion prevails connected with the hepatic region, a cathartic dose of *calomel*, followed by *castor-oil*, may be necessary. If the stools indicate an acid reaction, *magnesia* may be given with advantage. When membranous films or shreds of coagulable lymph are passed, electro-galvanic applications over the abdomen and dorsal spinal region, combined with *creasote* or *tar*, in the form of a pill, is worthy of a trial (SIMPSON, CUMMING, WOOD). Next to those remedies are such as determine towards the skin. For this purpose combinations of *Dover's* with *James' Powder*, or a *solution of tartar emetic* with *laudanum*, are the most useful medicines; and their beneficial action may be aided by the use of the warm bath, and a flannel roller applied with firmness round the abdomen (CRAIGIE). Where hyperæmia is excessive, accompanied by moderate transudation from the intestine, as after extensive burns, accompanied by great pain, cold external applications are suitable, as well as in the severer forms of catarrhal enteritis (NIEMEYER). The best mode of applying them is by cloths wrung out of cold water and laid over the abdomen. In the chronic form, combined with obstruction, irritating and warm compresses are most suitable. Preissnitz's compress is extensively used for this purpose. A wet towel in folds, covered with a dry one, is laid over the abdomen all night, to be renewed several times daily. The greatest care ought to be given to the management of the diet. It ought to be strictly antiphlogistic, consisting of slops and light puddings. Animal food must be withheld, and only permitted in the form of soup. The safest diets are those of arrow-root, sago, tapioca, barley-meal, gruel of oatmeal, *sowens*,* barley water, toast water, burnt oatcake water. This last-named substance, used as a drink, is said to allay irritation and morbid sensibility of the bowels (CRAIGIE). When the abdominal pains subside, and stools become less frequent and the appetite increases, the diet must be still more scrupulously attended to. Oatmeal porridge boiled to the utmost degree of pulpiness, and not too consistent, is one of the best of diets, which may be alternated with ground rice alone, or combined with barley flour, all of which may be eaten with diluted milk from the cow, or with milk undiluted of town-fed cows.

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* "The husk and some adhering starch separated from oats in the manufacture of oatmeal are sold in Scotland, under the inconsistent name of '*seeds*.' These, if infused in hot water, and allowed to become sourish in this state, yield on expression a mucilaginous liquid, which, on being sufficiently concentrated, forms a firm jelly known by the name of '*sowens*.' Not less than a quart of the *seeds* are to be rubbed for a considerable time with two quarts of hot water, after which the mixture is to be allowed to rest for several days till it becomes sour. It is then strained through a hair sieve, and the strained fluid left to rest till a white sediment subsides. The supernatant fluid is to be poured off, and the sediment washed with cold water; after which it may be either boiled with fresh water, stirring the whole time it is boiling, or it may be dried and prepared when convenient in the same manner as arrow-root. It may be eaten with wine, milk, or lemon-juice and sugar" (PEREIRA, *On Food*, p. 326).

TYPHLITIS.

LATIN EQ., *Inflammatio Cœci Intestinii;* FRENCH EQ., *Typhlite;* GERMAN EQ., *Blinddarmentzündung;* ITALIAN EQ., *Tiflitide.*

Definition.—*Inflammation of the cœcum, which may lead to ulceration of its mucous membrane, and not unfrequently of the entire wall of the bowel.*

Pathology.—*Causation.*—Collections of fæces in the cœcum and ascending colon; or of fæcal matter composed of such crude accumulations as the skins or stones of fruit, portions of unripe fruit or uncooked vegetable substances; or of intestinal concretions, or of balls of lumbrici, may be the first beginnings of cœcal inflammation, preceded by repeated attacks of colic and catarrh, with stomachache and alternate constipation and diarrhœa. When the inflammation is so severe as to involve the entire coats of the bowel with ulceration of the wall of the intestine, the muscular coat becomes paralysed, and unable to propel onwards the fæcal contents. Mucus, or bloody mucus, the result of catarrh, passes by the rectum; but no proper evacuation is obtained. Thus scyballæ tend to accumulate throughout the saccular pouches of the large intestine; and dilatation, with paralysis of the bowel, extends along the gut.

Symptoms.—In most cases there are severe pains in the right iliac region, and characteristic fæcal tumors. The pain is increased by pressure and motion, and is marked by severe exacerbations, with intervals of comparative ease. The fæcal tumor generally takes the position and assumes the shape of the cœcum and ascending colon, and may extend from the right iliac fossa towards the lower margin of the ribs. The inflammation may extend to the peritoneum of the adjoining intestine and abdominal wall, as well as to the connective tissue which unites the ascending colon behind to the iliac fascia (*perityphlitis*). There may then be pains in the right thigh; the psoas and iliacus muscles also being infiltrated, they are unable to contract, or the patient to raise his thigh. All these symptoms, although severe, may pass away after several passages of masses of badly smelling fæces; secondary inflammations cease, and the exudations are gradually absorbed, when pain subsides and the tumor disappears. On he other hand, the disease may be fatal by peritonitis, or by the formaion of a fæcal abscess in the right iliac region (NIEMEYER).

Treatment.—A full dose of *castor-oil* (℥ss. to ℥i.) is indicated if vomiting does not exist. Drastic purgation is not to be thought of. The use of nemas through long rectum tubes are the most efficient means of relief, which may throw up four or five pints of liquid in a continuous stream, o as to soften, crumble down, and set in motion the fæcal collections. *alt*, *castor-oil*, or *turpentine*, or *milk* ought to be added to the fluid injected

(NIEMEYER). Any effusion left may be resolved by the use of *iodide of potassium.* Leeches may be required if pain on pressure continues in the iliac region.

DYSENTERY.

LATIN EQ., *Dysenteria;* FRENCH EQ., *Dysenterie;* GERMAN EQ., *Ruhr*—Syn., *Dysenterie;* ITALIAN EQ., *Dissenteria.*

Definition.—*A febrile disease, accompanied by tormina, by straining, and by scanty mucus or bloody stools, which contain little or no fæcal matter. The minute lenticular and tubular glands of the mucous membrane of the large intestines, with the intertubular connective tissue, are the chief seats of the local lesion, which sometimes extends into the small intestine beyond the ileo-colic valve, as in cases in which* scorbutus *is a predisposing cause. Under some circumstances it is infective.*

Pathology.—(*a.*) *Historical.*—Dysentery has at all times proved one of the most severe scourges of our fleets on foreign stations, and of our armies in the field during campaigns, even in temperate regions. It is sometimes so prevalent that its victims exceed the number of sick from all other diseases put together. It has followed the tracks of all the great armies which have traversed Europe during the Continental wars of the past 200 years. It helped to destroy the British army in Holland in 1748. It decimated the French, Prussian, and Austrian armies in 1792. It was a chief cause of death in the ill-fated Walcheren Expedition in 1809. It cut down the garrison of Mantua in 1811 and 1812. Sir James M'Grigor records how fatal the disease was in the Peninsular campaigns; and we know how disastrous it was to our troops during the first winter they passed in the Crimea, in 1854. "It is the disease of the famished garrisons of besieged towns, of barren encampments, and of fleets navigating tropical seas, where fruits and vegetables cannot be procured. During the Peninsular War, the first Burmese War, and the late war with Russia, *dysentery* was one of the most prevalent and fatal diseases which reduced the strength of the armies" (Sir RANALD MARTIN). The extreme frequency and mortality of this disease in the armies of America during the Civil War of 1861 to 1863 gave it interest and importance to the American physician. More than one-fourth of all the cases of disease reported during the first two years of the war were forms of dysentery and diarrhœa. The annual number of cases for the whole army was greater than three-fourths of the mean strength—the ratio of cases being 765 per 1,000 the first year, 852 per 1,000 the second year; the mortality being at the rate of 12·36 per 1,000; but acute dysentery never prevailed as an epidemic in the camps of the American armies during the war (Dr. CLYMER). It is a dangerous and frequent disease throughout our intertropical possessions (TULLOCH, MARTIN, EWART). In England generally, however, *dysentery* as a cause of death has been decreasing since 1852, although about 200 years ago it was one of the most prevalent and fatal diseases of London. But although the disease is less violent and less fatal, and although the unfavourable hygienic conditions which were wont to bring about dysentery no longer exist, the active endemic conditions which favour, promote, or are congenial to its development are only dormant, and not eradicated. Dysentery is still sometimes brought about in this country just as in the days of Sydenham or Willis.

(*b.*) *Causation.*—But in no respect do we find that the dysentery of to-day differs from the description given by Sydenham more than a hundred and thirty years ago. When we look to the history of dysentery, and to the nature of its lesions—to its re-appearance from time to time among us, with the same identical characters—there are strong grounds for believing that there is something specific in the nature of the poison which produces some forms of dysentery, just as specific as that of *small-pox*, *typhus fever*, *typhoid fever*, *yellow fever*, *scarlatina*, *ague*, or *diphtheria*.

The records of Sir Alexander Tulloch and Dr. Joseph Ewart show, that the *prevalence and mortality* of dysentery in various countries may be stated in the following order of its greatest severity:—(1.) Western Africa; (2.) Ceylon; (3.) Tenasserim Provinces; (4.) Madras, Bombay, and Bengal; (5.) Nova Scotia and New Brunswick, with Windward and Leeward command; (6.) Malta. As a general proposition, it may be stated that there is no country where malarial fever exists in which dysentery is not an endemic and prevailing disease. In the East and West Indies, in China, the Ionian Islands, Gibraltar, Malta, the Canadas, Holland, the coasts of Africa, as well as in many parts of France, of the Peninsula, of the continent of America, and of the eastern parts of Great Britain, the prevalence of intermittent or remittent fevers and of dysentery is notorious. This connection is so intimate that a given number of persons exposed to the action of paludal miasmata—for example, a boat's crew sent ashore in a tropical climate—the probabilities are that of the men returning on board part will be seized with dysentery and part with intermittent fever. Paludal fever and dysentery are not only conjoined in locality, but they may co-exist, precede, or follow each other in the same individual, so that the fever frequently ends in dysentery, or the dysentery in remittent fever. This proof of the common nature of these diseases is corroborated by every writer of any note, more especially by those who have detailed the diseases of our armies. But dysentery prevails where there is no other evidence of the presence of malaria. Nevertheless, the evidence in favour of malaria being the common, though probably not the sole cause of dysentery, appears to be much the stronger. Dysentery is less common in the hotter than in the colder months, or arises under circumstances less favourable to vegetable decomposition. In India and China it is from the middle of November to the latter end of February, or when remittent fever changes into intermittent, that dysentery prevails. It seems to be recognised that there is a directly exciting action of malaria (quite apart from that indirect action of undermining the general health), the importance of which as a predisposing cause of dysentery cannot be over-estimated (GRANT, W. C. MACLEAN). Our knowledge of the predisposing causes of dysentery is derived from what has occurred in the military and naval medical service of the Queen. From the sufferings of our troops we learn that exposure to the night air, to wet, or to fatigue, together with intemperance and often improper diet incident to the life of a soldier, especially on active service in the field, have at all times furnished conditions powerfully predisposing to dysentery. The effects of *salt* diet in the production of dysentery being less well-known than other predisposing causes, it may be well to state that, in the Windward and Leeward command, when the rations issued to the troops consisted of salt provisions five days in the week, the mortality from diseases of the stomach and bowels among the *officers* was as two to four per cent., while that among the *soldiers* was as 20·7, or a tenfold ratio. On the contrary, in Jamaica, when salt provisions were issued to the troops only two days in the week, the mortality from the same diseases approximated so nearly between these two ranks as to be almost an equality. Corresponding facts to these have been observed in Gibraltar, on the coast of Africa, and at St. Helena. The Sierra Leone Commissioners on the western coast of Africa, were also of opinion that the large proportion of salt rations mainly contributed to the sickness and mortality from diseases of the stomach and bowels in the form of dysentery and diarrhœa. A marked reduction, to a *tenth* part of its former ratio, took place in the deaths from this class of diseases subsequent to the introduction of fresh meat diet. In 1797 the victualling of the navy was changed, greatly improved, and consequently (immediate to the change) the health of the seamen improved strikingly. *Scurvy, typhoid fever, dysentery,* and *ulcer* (which up to the period of change had produced great havoc), became comparatively rare in occurrence and light in impression;" and, it may now be added, are hardly known except by name* (Dr. WILSON). An insufficient diet was the main predisposing cause of the dysentery which prevailed in London at the Millbank Penitentiary shortly after its completion. This prison is built on a marsh below the level of the Thames at high-water, the river being banked out by a narrow causeway. As long as the prisoners were allowed a full and ample diet they appear to have resisted the action of the poison, and to have enjoyed good health. No sooner, however, was the quantity and quality of their dietary lowered, than dysentery of a very fatal character broke out, and made it necessary to clear that establishment of all its inmates. In the dysentery so well described by Dr. Clouston in the Cumberland and Westmoreland Asylum, the predisposing and exciting causes seem traceable to a diet deficient in fresh vegetable food in constitutions of feeble nervous power, in aged, weak, and paralysed persons, exposed to the poison of animal effluvia, in the form of decomposing and undiluted human excreta spread upon the land. The lesions were characteristic of the scorbutic form of dysentery; and the premonitory symptoms were similar to those described by Dr. Barker as peculiar to poisoning by sewage gases. Such cases were not contagious. From the MS. Notes of Dr. Alexander Grant regarding dysentery in India, to which I am kindly permitted to refer, I find that he, too, is of opinion that sewage miasm (animal effluvia poison?) may be a direct exciting cause of diarrhœa and of dysentery. In cachectic states, whether from malaria, syphilis, or scorbutus, the exhaustive effects of long-continued heat have a markedly predisposing influence in favouring the production of dysentery; and in the treatment of the disease the existence of any such cachexia must be inquired into, and, if possible, counteracted. The proportions in which the different ages suffer from dysentery is not quite determined, but the mortality seems to fall principally on men advanced in life (TULLOCH).

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* As Dr. Christison justly observes, the salt meat of military and naval rations is not the same as the salt meat of civil life. The former is highly salted, in order to keep for two or more years in every climate. Its nutritive value is thus greatly over-rated, and its nutritive constituents are still further diminished by the process of washing out in water before it can be eaten. Thus, besides the irritant effects of the salt diet in producing dysentery, another element exists as a cause of disease—namely, the insufficient nutrition which the salt ration diet is able to impart.

Besides unwholesome solid food, water of an impure kind favours the development of dysentery. Drained from swamps, and used for drinking and cooking purposes (as it was on the Chinese coasts), it exerted a marked influence both in exciting and in maintaining dysenteric symptoms. Many other predisposing causes favour the development and propagation of dysentery amongst soldiers in active service—namely, long marches in hot weather, bivouacking at night in the open air (often extremely cold both absolutely and relatively to the day), want of sufficient clothes and bedding. The causes of *chronic camp dysentery*, as it prevailed in the United States armies during the war of 1861 to 1863, are stated to be the long-continued co-operative action of the following agencies:—Scorbutic taint from defective diet; paludal toxæmia; filth and overcrowding of camps and barracks; excessive and prolonged heat, and physical fatigue during the prolonged and active campaigns. It does not seem to be so clearly understood as it ought to be, that some forms of dysentery are undoubtedly contagious, and capable of being propagated from person to person. Being a frequent complication or concomitant of contagious fevers, it has been believed to inherit similar contagious properties. In the severe form of dysentery (for which the old Infantry Barracks at Secunderabad, in the Deccan, have long been notorious) it has been observed that men labouring under other diseases, who happened to be exposed to the putrid effluvia of the excretions of dysenteric patients, were often severely affected by the disease (W. C. MACLEAN). There is, therefore, good reason to believe that the *exuviæ* of dysenteric patients, passed by stool, may (like those of typhoid fever) propagate the disease. The observations of Budd and Goodeve give support to this view; and Niemeyer also entertains this belief.

(*c.*) *Morbid Anatomy.*—It has been usual to describe cases of *dysentery* as being either *acute* or *chronic;* but there are cases belonging to a third class, which may be termed *complex.* Of these in their order,—

(1.) In *acute cases of dysentery* the specific lesion does not confine itself to the tissues of the mucous membrane only. The serous covering of the intestines, or even such solid viscera as the liver, spleen, kidneys, are generally involved ultimately in the diseased process. Ulceration or sloughing of large portions of mucous membrane and exudation go on together. The accounts of the morbid anatomy of dysentery are especially confusing. While the disease has been recognised during life, and defined as a febrile disease accompanied by tormina, and followed by straining and scanty mucus or bloody stools containing little or no fæcal matter, yet the local lesions associated with these conditions have not been clearly defined nor uniformly described. By some the disease has been regarded as a process of rapid, and at first of superficial inflammation, leading speedily to mortification of the mucous membrane of the intestine, unattended by any special disease of the solitary glands—that even in the slightest variety of dysentery the mucous membrane is only swollen and red, and may be removed in the form of a pulp from beneath the furfuraceous and vesicular epithelium. In after stages and in severer forms the mucous membrane becomes gelatinous, and is easily separable, or it passes into a state of sphacelus, black, friable, and offensive. Up till 1844, Chomel, Cruveilhier and Rokitansky taught that ulceration had no essential part in the disease-process which constitutes dysentery, and that it was of rare occurrence. It was not till Dr. Parkes published his admirable description of the morbid anatomy of dysentery, as he saw it in India, that we had any definite knowledge regarding its tropical forms. He not only showed the very early implication of the glandular apparatus of the great intestine in dysenteric inflammation, but he established the fact that, while ulceration occurs with great rapidity, a case never presents true dysenteric symptoms without ulceration being present. In 1843–44, at Moulmein, in India, he investigated fifty cases of dysentery in Europeans and twenty in Asiatics. He concluded from them that—(1.) Certain alterations in the glands of the mucous membrane of the large intestine, and sometimes of the ileum, constitute the earliest lesion in dysentery. (2.) That in all cases, when not too far advanced, the mucous membrane presented the appearance of numerous whitish round elevations, of a size varying from a millet-seed to a size so minute that a lens only can show the lesion. These elevations were hard, and being pierced gave forth a white excretion. Many of these had a black speck in the centre, and were surrounded by a vascular circle. (3.) That exudation sometimes occurred in points beneath the mucous surface; that such points of exudation had a white appearance, with contents similar to those of the solitary glands. The mucous membrane over these points could be easily rubbed off, leaving an ulcer (PARKES, *On the Dysentery and Hepatitis of India*). The observations of Parkes were thus opposed to the views held previously, and led to extended investigation, and, *inter alia*, it was determined that differences of climate do not cause any essential difference in the structural changes which accompany dysentery. The observations of Craigie and Abercrombie in Scotland, in 1837, prove this; and those of Baly, in 1847, as regards England. Cheyne, Graves, and Mayne have demonstrated the same fact as to Ireland. In the records of epidemic dysentery at Prague and elsewhere (as described by Finger and others), the observation holds true as regards the dysentery of Europe generally; and by comparing these records with well recorded cases of dysentery in the tropics, alike in civil and in military life, it will be seen that the dysentery of tropical and temperate climates does not differ *as to its anatomical signs* in any essential particular. The descriptions of the disease in our own country, as given by Cheyne, Craigie, Abercrombie, and Baly, all agree in recording the inflamed condition of the mucous membrane of the colon, with its small round ulcers, pulpy softening or sphacelus of some portions, and ulcers of various forms left by the separation of the sloughs, and enlarged firm tubercles, which no doubt were inflamed solitary glands. Again, Sir John Pringle, M. Broussais, and other historians of dysentery found the same lesions in the dysentery of the camps in the continental campaigns of

Europe; and Broussais expressly states his belief "that the ulcers of the large intestine had their origin in the solitary glands." Thus "tubercles," "pustules," and "small-pox-like elevations" (now known to be lesions of the solitary glands) of the mucous membrane have been most minutely described by Hewson, Pringle, and Davis. The last of these observers describes in graphic language the fatal dysentery of the Walcheren Expedition, and shows that its anatomical characters are similar in all respects to the forms of dysentery which have been described in this country.

The descriptions of the morbid anatomy of dysentery having thus been so much at variance with each other, several questions arise, namely,—(1.) Whether different epidemics are characterised by distinct local lesions? (2.) Whether two or more distinct diseases have not been confounded under the one name of dysentery? (3.) Whether the various local lesions described by different writers are only so many varieties, forms, or types of the same disease-process. This latter view is the one most consistent with observation; and it is in accordance with what we know of the history of *true yellow fever*, *remittent fever*, *diphtheria*, and the like. There is some evidence, also, to show that there is a lesion of the colon not belonging to true dysentery—a *colonitis*, in which the connective tissue of the gut beneath the mucous membrane is implicated in the first instance, rather than the glandular tubes and vesicles. The result is a diffuse gangrenous inflammation of the mucous membrane, the resulting ulcers not differing from the ulcers originating in the glands by any characters at present recognisable (COPLAND, PARKES).

In this country it has been held that the lesion in dysentery is confined, for the most part, to the colon and rectum; and that in tropical dysentery the whole course of the colon, and sometimes a considerable portion of the small intestines, are implicated. Lesions so extensive, while they are common in India, are rare in this country; yet they do occur, and are not uncommon in the south of Europe, in Turkey, and the coasts of the Mediterranean. But, except when the case is associated with *scorbutus*, the small intestines are not involved. Therefore, as regards the extent of the lesion, there is no constant or distinctive characteristic between tropical dysentery and the dysentery of more temperate climates. In both geographical regions the anatomical changes comprehend redness of the mucous membrane preceding further changes; loss of substance of the mucous glands by pulpy softening of tissue, sloughing, or ulceration; the detachment of diphtheritic casts of the intestine, exuviæ, or sloughs of exuded material. The type of the inflammatory lesion is essentially diphtheritic (NIEMEYER). The mucous structures are infiltrated with a fibrinous exudation, which enters deeply into the tissue, and which eventually dies, with loss of substance of the intestine. The structure of the colon in the healthy state differs in many important particulars from that of the small intestine. It is remarkable for the absence of folds and villi, and for the presence of more or less dilated sacculi, which give form and shape to the excrement. The minute tubular glands are thicker in proportion to their length than those of the small intestine; and the intertubular connective tissue is considerable,—a structure which takes an important share in the lesions of dysentery. These tubular glands are lined by columnar, cylindrical, and transition forms of epithelium (ALLEN THOMSON, PARKES). The tubular glands radiate round the solitary glands, indicated by a spot, which corresponds to the solitary gland below. These solitary glands have thick walls, and are said to be more abundant in the cœcum and rectum than in any other part of the great intestine. This statement leads to the question which has been mooted in relation to other solitary gland lesions, namely,—"Are these lesions of so-called solitary glands really due to the enlargement of previously existing solitary glands or their germs? or, Are they new formations altogether?" A similar question is at issue regarding the granulations on the eyelids and conjunctivæ, associated with *purulent ophthalmia* and *granular lids* (see p. 440, Vol. I., Sixth Edition of this work, STROMEYER, FRANK, MARSTON), in which we have *new formations* of vesicular-like granulations, as well as enlarged follicles; but these are more numerous than the glands in the healthy state. Observations somewhat similar have been made regarding the vesicular glands of the stomach (HANDFIELD JONES). It may be that not a few of the "tubercle nodules," the "pustules," the "small-pox-like elevations," and what we call solitary or lenticular glands, are in reality new formations altogether, resulting from increased cell-growth within the meshes of the connective tissue which binds the mucous gland tubes together, and connects them with the submucous muscular layer. In this respect their formation would be analogous to, and not to be distinguished histologically from, a newly formed grey tubercle nodule. So independent have these lesions been believed to be, that one observer of Indian dysentery (MURRAY) described a "pustular form of dysentery," which he believed to be in all respects analogous to small-pox on the skin, beginning with the formation of an independent papule, and the development of a subsequent pustule. This view of the subject is of some importance in pathology. It is related to the specific nature of dysentery and the poison cast off from the mucous membrane, by which it is believed that the disease is propagated like typhoid fever and cholera (Dr. WILLIAM BUDD).

There can now be no doubt that the anatomical signs of true dysentery are inflammation of the solitary lenticular follicles of the large intestine, tending in the first instance to infarction (*i.e.*, intumescence and congestion), and subsequently to ulceration and destruction of the gland tissue. The disease, extending by a similar process, ultimately involves the tubular glands of the general mucous membrane, which tend to soften and to be cast off as an exuvium or slough, exposing the submucous connective tissue, or even the muscular coat of the gut. It is the mucous membrane of the great intestine, and especially of the rectum and lower portion of the colon, which is the seat of these characteristic lesions. The exudative process is generally diffuse, involving the whole of the tissues of the mucous membrane in a diphtheritic process of infiltration. The diseased part looks as if it were covered with a bran-like coating, especially over the summits of the folds of the mucous membrane, which are deeply reddened by ecchymosis and injection, and infiltrated by the grey-like exudation. This bran-like coating cannot be scraped off without loss of substance; and the submucous connective tissue below is œdematous and swollen.

In the *scorbutic form of dysentery*, or in dysentery occurring in persons whose nervous or vital powers are feeble, or below *par* (as in the aged, infirm, or in the paralysis of the insane, conditions in some respects similar in their influence to *scorbutus*), a diphtheritic exudation covers not only the mucous surface of the colon, but (as an almost constant and pathognomonic sign) the same lesion is seen covering the mucous surface of the small intestine, extending upwards from the ileo-colic valve. This exudation grows or is laid down in the course of the blood-vessels ramifying from the mesenteric attachment transversely across the surface of the gut, and occupying especially the prominences of transverse rugæ (PARKES). This form of dysentery prevailed to a great extent among the soldiers who died at Scutari during that period of the Russian War when scorbutus prevailed. Some of the cases recorded by Davis in his description of the dysentery so fatal to the British troops in the famous Walcheren Expedition were of this nature. Finger, of Prague, Maine, of Dublin, and Baly, have recorded similar cases. One of the best accounts of the scorbutic form of dysentery of recent occurrence in this country is that which Dr. Clouston records in the Cumberland and Westmoreland Asylum, which he believed to have been caused by the effluvia from a field irrigated with sewage. The regular diet of the asylum inmates consisted of 24 ounces of animal food, 14 pints of milk, 16 ounces of suet dumpling, 7 pints of oatmeal porridge, 78 ounces of bread, and 7 pints of tea per week (*Med. Times*, June 3, 1865). No fresh vegetable diet seems to have been provided for; and diarrhœa does not seem to have been uncommon. In typical cases of the outbreak all the abdominal organs were found healthy until the small *intestine* was reached, which was normal to within five or six feet of the cœcum. The mucous membrane appeared reddened there in small spots or rings round the gut. Six inches farther down—*i.e.*, nearer the cœcum, the redness was universal, the membrane thickened and corrugated into folds, like small *valvulæ conniventes.* A few inches farther on, a yellowish dirty-looking deposit was seen over the mucous membrane in rings, very thin where it began, but gradually becoming thicker and more continuous, till near the cœcum it was one-eighth of an inch in thickness. The swelling of the mucous membrane also increased downwards, and the folds running across the gut became more prominent. These, with their coating of deposit, made the inside of the bowel look like a series of thick transverse ridges, covering its entire surface. This deposit was soft on the surface, but got more firm towards the mucous membrane, with which it incorporated itself, so that it could not be scraped off without leaving the fibrous covering of the muscular coat exposed as a highly vascular, raw surface. It was a soft lymphy-looking substance, yet towards the mucous membrane, it assumed the consistence and appearance of a soft fibrous membrane. The *cœcum* was found in the same state as the lower part of the small intestine, with two or three ragged ulcerations the size of beans. In the *ascending colon* the ulcerations were deeper and larger; but the lymphy deposit on the surface of the mucous membrane became thicker and more feculent in colour. Towards the *transverse colon* the inside of the gut was one mass of large irregular ulcers, with patches of deposit between them. The colour of the surface was almost black, and this continued down to the very lower part of the rectum. The mesenteric glands opposite the affected parts of the small and large intestines were enlarged and dark-coloured, and on section were soft and pulpy in consistency. Such were the general appearances in a case that had lasted for about a month (*Med. Times and Gazette*, June 10, 1865, p. 598).

Another classic description of the morbid anatomy of *scorbutic dysentery* has been given by the late Dr. Baly in his Gulstonian Lectures for 1847. He described *two* different forms of lesions amongst convicts at the Millbank Prison; and these forms he believed to correspond with two degrees of severity of symptoms during life:—(1.) A swollen condition of the solitary glands, forming round prominences on the surface of the mucous membrane, of various sizes; in colour pale, or red round the base, and dotted at the summit with a vascular spot. These appearances would occur about the eighteenth or twentieth day. At an earlier period the congestion round the glands would be more intense, while at a later period the summits of the prominences would become disorganised. Minute yellow sloughs subsequently form, which, becoming detached, leave an ulcer previously occupied by the gland. The mucous membrane around participates in the process. It is red, tumid, and covered with an aphthous layer of lymph, to the extent of one or two inches around, with three or four solitary glands prominent in the midst. The ulcers which form result from sloughs separating, and the disease still remains *not severe* as regards the amount of tissue involved; but *as to duration*, the illness may be prolonged and tedious, and the solitary gland cavities may enlarge very much—as large as a horse-bean. (2.) In more severe forms a greater variety and extent of tissue is involved, especially of the tubular glands. There is greater redness, tumefaction, and softening of tissue; and with the change of colour and of texture the secretions are greatly altered. The clear mucus is highly charged with albumen, and subsequently with blood.* These changes occupy principally the prominences of the transverse rugæ. The exudation on the free surface in the recent condition may form a thin opaque membrane three or four lines thick. It is homogeneous and of considerable consistence, so that it may be detached, and raised in flakes from the subjacent surface. With the exception of increased vascularity, the subjacent mucous surface appears still unchanged. A microscopic examination of this exudation shows that it varies with the severity of the case to some extent. In mild cases it is simply particles of epithelium mixed with amorphous granules. In more severe forms the exudation consists of fine germs with nuclei, mixed with elongated cell-forms (connective-tissue cells); and the examination of carefully prepared sections shows that such exudation mainly commences in the tubular glands, and by germination subsequently spreads over the mucous surface. In Dr. Clouston's cases, examination of the fibrinous layer (over the small intestine), in the fresh state, showed its structure to consist "of nucleated cells like pus-cells, puriform cells with nucleus, and a fibrinous material between them." The blood-vessels underneath the

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* Some account of the chemistry of the stools in dysentery has been given by Oesterlen, who describes an excessive elimination of water and of albumen. It is very desirable that this observation should be verified in India. The stools should be collected free from urine; and the albumen should be estimated separately from any insoluble sediment. If in severe cases two and a half ounces of albumen are passed in twenty-four hours by stool, it is impossible to over-estimate the importance of such an occurrence.

"fibrous layer were enlarged and very tortuous, and on the free surface of the mucous membrane, between the villi, they could be seen torn and open-mouthed" (*Med. Times*, l. c.)

Exuviæ, or casts of the intestine, may be thrown off in large masses or shreds, leaving a raw vascular surface underneath; and in some respects this process and these casts are analogous to similar phenomena in *croup*, *diphtheria*, *dysmenorrhœa*, and *typhoid fever*. By carefully examining the evacuations, important information may be obtained as to the nature of the process going on in the intestines. Goodeve, of Calcutta, has made some valuable observations of this kind. The evacuations should be washed with water, to get rid of the fæcal matters entirely, and to leave the sediment, which is the product of the colonic disease, free from bile, fæcal matter, and offensive smell. This sediment consists of ropy, gelatinous, branny, or thready mucus; lymph in the shreds or granular masses; pus; fæces; and sloughs. Patches, half an inch or an inch or more in size, are cast off as sloughs. These exuviæ are thin, membranous, and sometimes infiltrated with pus; or they are thick and of a yellowish-brown colour. It is not till after the *eighth* or *twelfth* day that such sloughs are cast off. They may be similar to those cast off from Peyer's patches in typhoid fever. After they are cast off the symptoms diminish, and the patient often gets well rapidly. The following conditions are indicated by the various kinds of *exuviæ* cast off in dysentery and recovered by the washing process of Goodeve:—(1.) *Ecchymosed sloughs* indicate abraded or minute ulcers; intestinal apoplexy; (2.) *compact grey or light-yellow sloughs* indicate acute phlegmonous dysentery; (3.) *thick pus infiltrated mucus* indicates erysipelatous dysentery; (4.) *ragged sloughs* denote gangrene; (5.) *dark olive sloughs* denote secondary gangrene of mucous coat; (6.) *thin black, plain, or tubular sloughs* denote primary gangrene of mucous coat; (7.) *shreddy sloughs* denote gangrene in either mucous or cellular coat; (8.) *molecular or putrilage* indicate disintegration of tissue; (9.) *flaky epithelial sloughs* denote commencing gangrene of mucous membrane; (10.) *shaggy sloughs* indicate violent inflammatory action; (11.) *free filamentous* (*simple*) *sloughs* denote primary gangrene in submucous tissue; (12.) *free filamentous* (*pus-infiltrated*) *sloughs* denote submucous cellulitis; (13.) *ring-shaped sloughs* denote ring-shaped ulceration in mucous folds; (14.) *discoid sloughs* indicate circular ulcers in ecchymosed patches.

During the shedding of the shreds the patients are much griped, and they pass with straining fifteen or even twenty times a day the sanguinolent masses, or slimy mucus in small quantities, generally without fæcal matter. Then a period of cure and improvement supervenes, with diminution of the fæcal discharges—not simply by resolution, but by termination of the specific sloughs or lesions which have formed in the course of the disease. When these membranous flakes are not shed, but retained and ultimately separated in large pieces, there is considerable danger attending the process. Discharges of blood and fatal hæmorrhages may ensue. Changes go on between the intertubular connective tissue and the substance of this exudation, which tend to its organisation or supply with blood, and subsequently to its destruction, separation, or removal by ulceration. In vertical sections, down to and through the mucous membrane, I have seen fine blood-vessels, in loops and bulbous cœcal ends, shooting upwards beyond the mucous surface into the exudation; and when such exudation was forcibly removed from off the mucous surface, the membrane on which it lay was seen to be highly vascular, and numerous minute ruptured vessels showed their torn mouths. Dr. Morehead records a similar observation as regards the connection of the exudation with the subjacent mucous tissue, "through the medium of what appeared to be small capillary vessels, the mucous membrane beneath being vascular" (*Researches on Disease in India*, p. 241). In a case that proved fatal at the end of six weeks, in which the gut was so friable that almost the least current of water tore it up, Dr. Clouston noticed that "in the rectum blood-clots projected from the open mouths of arteries." When such a state of vascular action in the mucous membrane and exudation has existed for a lengthened period, the tissue of the gut becomes greatly thickened, and less coherent. These thickened portions grow luxuriantly, just as isolated patches in a field of green corn grow more luxuriantly than others, being supplied with a greater amount of nutritive material, as the thickened patches of mucous membrane are supplied with more enlarged, more tortuous, and more extensively distributed loops of blood-vessels. These thickened masses continue to fungate and grow from a hardened base, from which the numerous blood-vessels pass into the growing masses. In this way the warty condition of the mucous membrane in chronic cases undoubtedly results. The new material evinces a disposition to contract. The calibre or bore of the gut gradually diminishes, and its texture becomes so brittle that slight force in pulling up a piece of such intestine out of its place at a *post-mortem* examination will readily cause it to *break* asunder.

With such varied lesions, the formation of ulcers does not take place in any uniform mode. The following is a summary of the processes from which ulceration may proceed:—(1.) After intumescence, softening, and simple ulceration of one or of several lenticular solitary glands. (2.) After intumescence, softening, and sphacelus of many solitary lenticular glands and the intervening tissue in one mass. (3.) After softening of the tubular structure and the detachment of sloughs, ulceration follows the intumescence and germination of growths from the tubes which cover the surface as a "croupous," "catarrhal," or "diphtheritic" exudation, to which the name of "aphthous erosion" has been applied. (4.) After submucous inflammation and new growths, with fibrinous and mucinous effusion. (5.) After intertubular inflammation, and inflammation surrounding the base of inflamed glands. (6.) After the formation of submucou abscess. (7.) By changes of an ulcerative nature, commencing in th vascular exudation itself, as in scorbutic cases.

The *circular* ulcers for the most part originate in solitary gland (Parkes, Baly), or in circular patches of tubes (Morehead), similarly t the stomach ulceration. Or such circular ulcers may result from both, a when a solitary gland is destroyed it carries with its destruction some the adjacent tubes. In such cases the colon presents prominent little masse about the size of a pea, which burst readily on pressure, and give for pus or milk-like fluid contents. Such abscesses may open spontaneous upon the mucous surface through the short canal leading from the gla (now an abscess), imbedded in the submucous tissue, between the tubul glands. They undermine the tubular gland substance, and carry off shreds or patches of surrounding tissue. They may be seen in all stages, and almost symmetrically arranged in a double row along the colon (BLEEKER, MOREHEAD). Many of these little abscess cavities are formed below patches of thick exudation (HASPEL). The *transverse ulcers* are due to the transverse arrangement of folds, on which the exudation and textures ulcerate; and I have known the transverse *rupturing* of very thick exudation mistaken at *post-mortem* examinations for ulceration, on seeing the raw vascular surface of the tissue exposed below at the bottom of the rent.

(2.) *In Chronic Dysentery* the exudation undergoes various changes. It may be thrown off from the mucous surface altogether, leaving the surface bare and raw looking, as if ulcerated; but a close inspection will show that the surface is entire and highly vascular. If it is not thrown off, it may undergo a considerable amount of organisation, after which a process of ulceration may be established upon its surface, just as in any other soft tissue. This ulcerative process may extend through the whole exudation, even to the surface of the mucous membrane, which it may penetrate and involve the tissues of the intestine in the ulcerative process close to the peritoneal coat. Perforation of the peritoneum is by no means uncommon. Another very constant morbid change consists in the deposit of black granular matter on some parts of the mucous membrane. It may be regarded as the result of excessive vascular action, and of subsequent changes in the extravasated blood which mark the site of the melanic spot ("Pigmentary Degeneration," see p. 127).

The sigmoid flexure of the colon is the most frequently and the most extensively diseased, the lesion being most expressed towards the rectum. In very severe cases the exudation extends over the whole extent of the mucous surface of the *colon*, which appears covered with black, grumous, carbonised-looking masses, even to the upper part. An *appearance* of ulceration often extends in lines across the gut, so as to embrace its whole calibre in some parts. When the gut is opened in the usual way after death, and extended on a flat surface, the change from the hitherto curved condition of the intestine is so great as to cause rupture and separation between thick masses of exudation, giving rise to the appearance which has sometimes been described as ulceration. In long-continued chronic cases the rectum is generally studded over with punched-out-looking ulcers, with bloodless bases and thin anæmic edges; and the melanotic deposit, already noticed, is here seen in the greatest abundance. Evidence of healed ulcers, with partially renewed mucous tissue covering them, are not uncommon in this locality, their place being indicated by the amount of black matter. The gland tissue, however, is not reproduced in the cicatrix substance.

(3.) *In Complex Cases of Dysentery* there are a variety of lesions, the pathological significance of which as to extent, form, origin, and locality, renders such cases complex. The lesions are,—(1.) Extension of the dysenteric process over the mucous membrane of the small intestine. (2.) Deposits and ulcerations in the glands of Peyer, as well as in the general tubular structure near the ileo-cœcal valve. (3.) Atrophy of the glandular parts of the mucous membrane of the alimentary canal. (4.) Secondary lesions of serous membranes. (5.) Secondary lesions of solid viscera in the cavities of the *abdomen* and *thorax*. (6.) Secondary lesions due to the syphilitic, scorbutic, typhus, or tuberculous states, or to the influence of malaria.

In some rapid and acute cases of *dysentery* (five out of twenty-eight cases, as observed by Dr. BALY) it has been noticed that the process by which the *dysenteric lesions* were developed in the large intestine extended beyond the ileo-cœcal valve into the small intestine. As much as the lower two-thirds of the ileum have been involved in this process, while the upper portion has been found intensely congested. In one case of *dysentery*, Cheyne found an exudation of lymph extending nearly over the whole of the jejunum. If the stomach participates, the mucous membrane may be diffusely inflamed, or of a red or violet colour, its surface granulated, and its texture broken by the slightest touch. More commonly the colour of the mucous membrane is natural, but on its surface a number of ecchymosed spots, or small ulcers, are seen, with edges so sharp, clean, and perpendicular, that they appear as if made with a punch. In other cases the tubular glands, as well as the solitary and aggregate glands of Peyer, have shown various stages of morbid action. The absorbent mesenteric glands are rarely affected (BALY); but, except in cases of secondary hepatic abscess, they were found enlarged and inflamed in all cases of Indian dysentery (PARKES), and in dysentery associated with *scorbutus*. By far the most common condition (in chronic *dysentery* especially) is that which is due to atrophy of the mucous membrane. As an atrophic change, it may be ascribed to the general wasting (marasmic) processes which take place to a great extent throughout the system in such cases. In this complex state the mucous membrane of the *small intestine* appears pale, thin, and worn,—a condition which pervades the greater part of the alimentary canal, which is especially made manifest in the living as well as in the dead by the condition of the mucous membrane of the mouth. On turning down the lips, the mucous glands are seen distinctly projecting through the thin pale labial and buccal mucous membrane. When such cases ar examined after death, the structure of the solitary glands and of Peyer' patches are degenerated and wasted; no gland-cells are to be seen, an their place is supplied by fibroid tissue, with some vascular injection roun the reticulated spaces. In other instances a deposit of black pigmen surrounds the locality of the glands, which indicates the long-continue process of vascular action previous to their atrophy. Associated with th general atrophic state, some gland-patches may be observed in an app rently opposite state—that is, distended, and sometimes engorged; bu on examination, their contents appear to be undergoing a molecula melanotic, and generally fatty degeneration, probably preparatory to co plete destruction of the gland-element. These two apparently opposi conditions, co-existing in the same cases, appear to indicate that the o condition is but the antecedent of the other; and that the atrophy a degeneration are the last result of a series of morbid processes commenci in the engorged gland-cavities. In parts of the mucous tissue which hibited the opposite conditions of extreme hypertrophy and atrophy, t specific gravity of the former indicated 1·046, while the thin and wast part of the intestine indicated a specific gravity of 1·036 to 1·030. Th is now abundance of evidence to show that in some endemic cases, or epidemics of *dysentery* in some places, there is a tendency to the secondary affections of organs or parts, during or subsequent to the development of the dysenteric process. Some look upon these secondary processes in relation to the dysentery as in the relation of effect following cause; or that there is an immediate and direct connection between the primary dysenteric process and the secondary lesion. Such a relationship has not been shown to exist in all cases; and it is more probable that the *dysenteric* process, when it operates on the system during a protracted period, predisposes, as many other morbid states do, to the development of secondary local lesions in distant parts. The *arachnoid*, the *pleuræ*, the *pericardium*, and the *peritoneum* have each and all been the seat of opacities or of fluid exudations in dysenteric cases.

Of morbid states of the *solid viscera* associated with dysentery, by far the most frequent complication is with the *kidneys* and the *liver*. With regard to the kidneys, their relation to the bowel affection is as yet obscure. In mild cases proceeding to a favourable termination, there is no albumen in the urine and no casts. When the dysentery is severe, it continues some time before albumen appears in the urine, and then its occurrence is preceded and attended by putridity of the copious stools, by *status nervosus*, collapse, and paralytic phenomena. If the renal affection occurs early, so much more severe is the case, and death usually speedily ensues. The kidneys are highly congested, the tubes loaded with exudation cells and detritus (ZIMMERMAN, *Syden. Society Year-Book* for 1861). The association of *hepatic disease* with *dysentery* seems to be most frequent in the East Indies, and in such climates as have a similar influence (MARTIN). In the Bombay army, out of thirty fatal cases of *dysentery*, twelve were attended with hepatic abscess (MOREHEAD). Macpherson, Sir James M'Grigor, Parkes, and Henry Marshall, gave similar statistical results of their experience at Calcutta, Moulmein, and Ceylon. The French surgeons in the province of Oran, in Algeria, state that hepatitis and consequent abscess were frequently coincident with dysentery. Parkes observes that if the functional morbid state of the liver is to be judged of by chemical analysis of the secretion of that viscus, the liver is found to be diseased, more or less, in every case of dysentery. W. J. Moore (Assistant-Surgeon of the Bombay army) has collected a valuable series of statistics, which sets in a stronger light than hitherto the relation of hepatic *lesions* to dysentery. The records of five independent observers show *lesions* of the liver (not being abscesses) in about 57 per cent. of the cases; while the number of cases in which *abscess* was observed averages about 18 per cent. (GRANT'S *Annals of Military and Naval Surgery*, Vol. I., p. 227). The tendency to hepatic complication was found in Algeria to increase with age, and with the length of service in that country. It appears, however, that hepatic *abscess* is but rarely associated with dysentery in natives of those warm climates; and amongst British subjects in their native climate it seems equally rare. In the Millbank Prison, "out of many hundred cases, not one has been complicated with hepatic *abscess*." It does not appear, however, that the influence of the climate of the East alone on Europeans tends to the hepatic complication, for "in the Peninsular army, under the Duke of Wellington, the spleen, the liver, and the mesentery were generally found diseased in cases of dysentery; so were these viscera in the epidemic dysentery of Ireland" (MARTIN). In the dysentery of the allied armies in the hospitals of Scutari and the Crimea, during the Russian War of 1854 to '57, hepatic abscess was of rare occurrence. Budd attempts to explain how hepatic abscess is a *consequence* of dysentery through the vitiation of the portal blood from the morbid intestines. But the evidence tends rather to show that the hepatic lesion and the dysentery are each excited by the same cause. If Budd's theory were correct, we ought to have liver abscess a common occurrence after ulcerations of typhoid fever, and after those of tuberculous lesions of the intestines; but we do not find that abscess of the liver is usual in such cases.

Regarding hepatic complication in dysentery, the following conclusions may be stated:—(1.) That dysentery, in a great number of cases, more than a half, commences and runs its course complicated by obvious functional hepatic disease. (2.) That the hepatic disorder and the dysentery acknowledge a common cause and disease-process. (3.) That about 18 per cent. of the fatal cases of *dysentery* are complicated with hepatic abscess, and about 57 per cent. with other hepatic lesions. (4.) That in a few of these cases ulceration of the intestine may be the primary disease, and the source of the hepatic abscess by the infective or simply mechanical phenomena of thrombosis and embolism in connection with the pelvic veins and veins of the mesocolon. The occurrence of hepatic abscess with dysentery has been generally viewed as a result of phlebitis; but Dr. Parkes, after the most careful investigation, never found the slightest trace of inflammation in the small veins of the intestine, and no direct proof has been advanced of the mediation of the portal blood in the process. In conclusion, the preference must be given to that view which regards the two diseased processes, dysentery and abscess of the liver, as running their course together, dependent upon one and the same cause. In hot climates abscess of the liver very frequently occurs associated with remittent fevers, or consecutive to them, without dissection exhibiting any ulceration of the mucous membrane of the intestine (HENOCH, *Brit. and For. Med.-Chir. Review*, July, 1854; Dr. MOREHEAD, in *British Medical Journal*, March 20, 1869). The comparative frequency of hepatic abscesses in cases of dysentery varies from 13 per cent. in Calcutta General Hospital (MACPHERSON), to nearly 60 per cent. in Madras Presidency, (ANNESLEY).

Too much attention and importance seem to have been put upon abscess of the liver *per se*, irrespective of other obviously morbid conditions of that organ—*e.g.*, impaired function, congestion, enlargement. To regard secondary hepatic abscess as due to absorption of pus or other morbific matter from ulcerating mucous membrane, or to a true phlebitis, is to take too narrow a view of the relation of liver disease to dysentery; for, if we are to judge by the condition of the bile alone, the liver is diseased (in function, at least) in every case of dysentery (PARKES). The contrast of the results seen in hot climates compared with those seen in colder climates, is remarkable. Baly's experience yielded him no abscesses of the liver. Finger, of Prague, dissected 231 case of dysentery between 1846 and 1848, and found no abscess of the liver. Broussais records seventeen dissections of dysentery in camp during 1805 and 1806, and no abscesses of the liver. Rokitansky never found the liver visibly diseased in cases of dysentery; and in China, where dysentery, as a rule, is very fatal to Europeans, the rarity of hepatic abscess is said to have been remarkable (Dr. WILSON in *Records of Hospital Ship "Minden"*). On the whole, it will be seen that the association of dysentery with hepatic abscess is not equally frequent in all countries, nor in all epidemics. It seems to have been most frequent in the climate of the East Indies, and in the Bombay army especially (MOREHEAD, PARKES). There are some epidemics in Europe in which the hepatic lesion has been observed—*e.g.*, in Dublin, 1818, it was observed in four out of thirty cases (CHEYNE). It would therefore appear that the *poison* which causes dysentery has at some times and places the power of establishing hepatic complication so severe as to lead to abscess; at other times and places it seems to be less virulent or infective.

The *spleen* and *pancreas* may also be found diseased. The splenic complication is one of the most fatal in the dysentery of the East Indies (TWINING). These viscera are found either enlarged and softened, or enlarged and indurated, the spleen being sometimes the seat of abscess. When dysentery follows upon, or is associated with intermittent fever, the spleen will frequently become enlarged, indicated in the outset by general anæmia, or splenic cachexia, with a low asthenic type of dysentery.

Of *thoracic viscera* the lungs have sometimes exhibited a great tendency to secondary morbid processes. This was especially the case in the dysentery of the allied armies during the Russian War of 1854–57, where otherwise pulmonic lesions were rare. The pulmonic lesions were as follows:—(1.) More or less extensive inflammation of the bronchial membrane, the finer ramifications of the tubes being filled with frothy mucus and pus-like exudation, and associated with extensive vesicular bronchitis: there were also well-marked spots of lobular pneumonia. (2.) Exudations into the pulmonary parenchyma, chiefly in the form of isolated deposits of considerable density, disseminated through the substance of the lungs. These masses passed into a purulent condition, and microscopically they were composed of broken-up cells, granular matter, and pus-elements.

The last class of conditions which render cases of dysentery complex is the alliance of other disease-processes with it. Such cases are of protracted duration; the associated lesions and the organs attacked are numerous, and there is every variety of kind, degree, and extent of the co-existent affections. Many disease-processes may be observed to co-exist in one patient; which tends greatly to multiply the number of the anatomical local lesions, and thereby still more to complicate the case. Dysentery "is found to complicate readily in all climates with the prevailing fevers." Within the tropics it is frequently associated with *remittent* and *intermittent fevers;* in the geographical region of *typhus fever* it is a most frequent complication under various circumstances, and becomes capable of propagation from person to person; and lastly, it is also occasionally complicated with *scurvy*. The *scorbutic complication* is developed in cases of *dysentery* when the supply of food has been deficient in fresh vegetables, or when it consists in whole or in greater part of salted meat. Such a complication has been known to arise among prisoners of war living entirely on fresh (animal) diet (Sir GILBERT BLANE). "The most terrible instance of suffering from this cause was that of the European portion of the force employed in Ava during the first Burmese War, where they were for six and a half months fed on salt rations, and where 48 per cent. perished within ten months, principally by dysentery with the scorbutic state" (Sir Ranald Martin). Such disasters have since been equalled, if not surpassed, by the sufferings of our troops in the camp before Sebastopol during the winters of 1854 and 1855, under the influence of exposure, fatigue, and continued rations of salt meat and green coffee; in the chronic camp dysentery of the American armies during the war of 1861 to 1863. The morbid anatomy of the lesions seems to bring the cases under "*Complex forms of Dysentery.*" There were—(1.) Follicular lesions of the colon, presenting all the transition forms of simple enlargement of the solitary follicles, rupture of the same, and the formation of rounded or oval ulcers, extending nearly or quite to the muscular coat, looking as if they had been cut out with a punch, on a greyish or yellowish-grey base, and sometimes filled with mucus, at other times pus. The enlarged follicles are occasionally the seat of pigment-deposits; and in some cases an areola of pigment, deposited in and among the glands of Lieberkühn, surrounds the enlarged and solitary black follicles. This was found generally in patients who had died of some other disease, as intercurrent camp fever, &c. The colon is usually more or less thickened, even to the amount of a quarter of an inch. Its texture, when cut into, is frequently tough and lardaceous, and often softened. The colour of the mucous membrane of the colon is seldom natural, being ash or slate-coloured, or greenish-red, reddish-brown, or reddish-black; at times there are patches of congestion. (2.) The extension and agglomeration of the follicular ulcers, by burrowing in the submucous connective tissue, destroy large portions of the mucous membrane by vast erosions. The mucous layer, containing the glands of Lieberkühn, undermined by the extension of the ulcer, not unfrequently hangs in shreds like a fringe from its edge, the undermined portion being occasionally destroyed by ulceration, but more frequently by sloughing. In such cases the mucous membrane is generally of a slate, dark-red, brownish or greenish-brown colour; the base of the ulcers is yellowish or yellowish-brown, often with brown or blackish sloughs adhering to their surface or edges. This group represents a more advanced stage of the disease. (3.) In addition to the lesions of the first and second groups, the surface of the large intestine is more or less coated with a yellowish, or greenish-yellow, or brownish-yellow croupous, pseudo-plastic, caco-plastic, or false membrane, similar to the membrane formed in the air-passages in diphtheria, which sometimes extends to the small intestines, and is generally found in those who have died during the supervention of acute dysenteric symptoms. Examination by the microscope shows it to be composed of innumerable round cells (lymph-cells, pus-cells), held together by an adhesive granular matrix, more or less resembling coagulated fibrine. The origin of this membrane may be traced to a rapid multiplication of epithelial cells and superficial connective tissue corpuscles of the diseased mucous membrane. (4.) Two forms of ulceration are observed in the colon: in the first, the process begins in the closed follicles; in the second, in the intestinal epithelium or the glandular layer. The closed follicles enlarge by multiplication of their cellular elements till they project as little tumors above the surface. The tumor, having enlarged to a certain extent, ruptures; its cellular elements escape, and a minute ulcer is formed. The cells or corpuscles of the connective tissue surrounding the enlarged follicle now multiply, and the ulcer spreads by the superficial cells floating off into the intestinal cavity, while a new base is continually formed by the multiplication of the subjacent cells. The second form of ulceration begins by an abrasion or denudation of epithelium at some point which does not correspond to the position of a solitary follicle. The follicles of Lieberkühn are next destroyed, and the ulcer spreads in the connective tissue by the process just described. Such ulcers are rarer than those of the first kind, and probably are always secondary to them.

Metastatic abscesses, resembling those in pyæmia after gunshot injuries, are sometimes found in the liver, lungs, and spleen. In seventeen cases the kidneys were more or less diseased; their weight was somewhat increased, and the cortical portion abnormally light and granular. Microscopical examination showed the tubuli uriniferi filled with granules and detached epithelium (WOODWARD, *American Medical Times*, Feb., 1863).

Symptoms.—(*a.*) *Types and Forms of Dysentery.*—These have been variously described as—(1.) *The purely inflammatory, acute, hyper-acute,* or *sthenic* form. In this form, while the phenomena indicate acute and severe inflammatory action, there is no tendency to the great depression of the nervous, circulatory, and muscular functions, which gives a marked character to, (2.) the *asthenic* forms, in which, besides the depression of the functions just noticed, there is much greater tendency to spread by infection or under an epidemic influence. These asthenic forms are sometimes described as *adynamic, typhoid, malignant, bilious, intermittent,* or *remittent,* according as certain phenomena prevail characteristic of these states.

(*b.*) *Acute Dysentery.*—An ordinary attack generally commences with *diarrhœa;* but in twelve or twenty-four hours disagreeable feelings begin to attend the frequent loose discharges from the bowels. These are irregular pains, commonly called "gripes," along the course of the large intestine, sometimes described as "shooting," or "cutting," and technically known as *tormina.* They are momentarily relieved by discharges from the bowels. But after a short time a sense of heat ascends from the rectum, and pain extends to the epigastrium, till the whole abdomen is painful. There is a frequently returning inclination to go to stool: the griping and straining continue without the patient being able to pass anything more than a little bloody mucus. These symptoms are generally aggravated during the night and early morning, and leave behind them the exhausting sensation that there has always remained in the bowel something which has yet to be discharged. This feeling is technically called *tenesmus,* and ultimately becomes the most striking feature in the case. The acute pain in the abdomen, although it may extend to the iliac regions or flanks, generally concentrates itself about the rectum. The discharges from the bowels are at first scanty, consisting of mucus and blood, or bloody slime, as it is sometimes called. As the disease progresses, the evacuations become more copious, tinged with bile, and carrying off shreds of the exudation thrown out on the mucous surface of the intestine. Hardened balls of fæces, called *scybalæ*, are sometimes discharged: these, however, are seldom seen in tropical dysentery; and if much feculent matter passes, there is always considerable relief. When the disease is fully established, the discharges exhale an odour different from the smell of fæces, which is almost peculiar to dysentery; and very offensive. It is important to observe the character of the discharges, and especially as to the relative amount of blood, mucus, and shreds of exuviæ (see page 849, *ante*). If the disease advances, besides the constitutional symptoms becoming aggravated, more blood and mucus appear in the discharges from the intestines, together with shreds or large sloughs of exudation, which are often erroneously described as pieces of mucous membrane. In very acute cases, going on rapidly to an unfavourable termination, a great change often takes place in the nature of the stools, which become suddenly copious, serous, of a reddish-brown colour with black spots, attended with a putrid offensive odour which pervades the whole house in which the patient is. In the acute dysentery of Lower Bengal, the patient is not unfrequently carried off by copious discharges of blood (W. C. MACLEAN). The shreds which are passed in the stools are not mucous membrane, but, like the dysmenorrhœal membrane which forms on the internal surface of the uterus, the dysenteric slough varies in consistence, thickness, and strength. It may be washed perfectly white in water, and its minute histology shows no character of a mucous membrane. The hardened balls of fæces are much more rarely seen than they have been described to be.

In the dysentery described by Dr. Clouston some of the patients had ordinary diarrhœa (diarrhœa of irritation ?), from periods varying from two or three hours up to twenty-four hours, before blood appeared in the stools. In some cases there was great pain in the abdomen for twenty-four hours before the diarrhœa set in (evidence of irritation ?). In other cases there was scarcely any pain at any period of the disease. Dr. Clouston distinguished two classes of scorbutic dysentery cases. "In *the first* the patient had two or three loose stools, or at once began to pass glairy mucus mixed with blood, in small quantities at a time, from the bowel. The patient had no pain, no fever, no want of appetite, and he refused to believe he was ill. This would continue for a day or two, and then the blood would increase in quantity, and the stools would become more frequent. Pain would begin to be felt in the region of the rectum, and the pulse would mount up by ten or twelve beats. For days the patient would be at stool every hour or two, and of course would become weaker His tongue was then seen to be coated with a dirty yellowish-white fur but the appetite for such forms of nourishment as milk, strong beef-tea calves'-foot jelly made with wine, was still good. Solid food was no relished. The stools would then be seen to be coated with a semi-fibrinou semi-purulent-looking membrane. The tongue would then become clear and glazed, and beef-steaky; the evacuations became feculent, mixed wit pus, the latter element becoming gradually less as the patient advanced i his slow convalescence. In *the second class* of cases the patient had fro the first great pain in the abdomen, of a griping kind, a hot skin, and pulse over 100; the dejections were copious, and frequent, and water while they were largely mixed with blood. In many cases there was sickness; in all, loss of appetite. After some days the tongue and mouth would become dry, and parched, and black, the features pinched, the pulse small and quick, and death soon ensued. In some cases the stools would after a time become membranous and shreddy, and then purulent, till the patient was more gradually weakened and exhausted. One such case lived six weeks, another two months. In one only of this class of cases (out of seventeen) did the patient recover. All the cases had the following features in common:—Bloody stools at first, tending to become purulent; intense fetor of the evacuations during the whole of the disease; no scybala, and great thirst" (*Med. Times and Gazette*, June 10, 1865). These were cases of (scorbutic) dysentery, caused by the poison of animal effluvia from decomposing human excreta (undiluted) acting on constitutions in which vegetable diet seems to have been deficient, and in whom the nervous power was below *par*.

Prognosis.—In *acute cases* death frequently takes place within the first ten or twelve days; but the disease may terminate gradually and spontaneously, or, as the result of appropriate treatment, by the end of the third or fourth week. On the other hand, the disease may not end so favourably and early, but may advance unchecked; the morbid changes being slow in progress, often extending over several months, when the case passes into *chronic dysentery*—one of the most hopeless and intractable forms of disease which the physician has to treat. Under the influence of slow morbid changes, the wasting of the tissues of the patient progresses steadily, till a human form, literally reduced to the state of a living skeleton, whose bones are held together by skin and ligament, is all that remains. The skin acquires a dry, bran-like, furfuraceous aspect, and the epithelium desquamates in scales and powdery particles. During the progress of such chronic cases various intercurrent morbid states become developed, not necessarily connected with the primary affection, but forming secondary lesions to the disease, and constituting the third form in which *dysentery* must be studied, namely—*complex cases* in which there are various secondary lesions regarded by some as directly connected with the primary affection. There are also secondary lesions connected with antecedent forms of disease, which sustain a renewed impulse to their development by the dysenteric state. These secondary lesions may be shortly stated to consist of—(1.) Lesions of the small intestines, and of various solid viscera more or less connected with the dysenteric state; and, (2.) lesions which may be referred to the co-existence of certain morbid states of the patient with the dysenteric condition, such, for instance, as the *typhus*, *scorbutic*, and the *scrofulous* state.

The prognosis in dysentery depends much on the country in which the disease occurs, and on the combination of circumstances predisposing to the disease, not less than on the form or type which the disease may assume. Dysentery is a disease which varies considerably in different countries and localities, and sometimes in apparent accordance with the exciting cause. Sporadic cases, which now and then occur in our large towns, are not generally so violent, and are less fatal than the epidemic cases and those which occur in tropical climates. The effects on the constitution are no less varied and severe. In hot climates it is calculated that the deaths vary from one in nine to one in twenty; and on actual service the chances of recovery are much diminished. In all returns, however, the total deaths recorded give a faint idea and inaccurate representation of the real mortality and loss of strength resulting from dysentery. Dysentery is a most formidable disease, on account of its oftentimes insidious nature, from its tendency to recur, and from the after-influences it exerts on particular organs and on the system at large. For these reasons almost all writers on the diseases prevalent in tropical climates place dysentery at the top of the list of severe affections, and refer to it as the cause and origin of many of those chronic and intractable abdominal diseases which so often afflict Europeans resident in tropical climates, and which entail most varied forms of impaired health when they return to European countries. If it were possible to trace out the men who were invalided from the army and navy services from the effects of this disease, it would be found that the mortality is very much greater than is represented by tabular returns. It is a malady which once fairly engrafted on the system, never leaves it till life itself becomes extinct (BRYSON and others). It is sometimes also insidious in its mode of attack and progress; and there is such a desire, on the part of soldiers especially, to avoid the restraints of hospitals, that the disease is sometimes beyond the power of medicine before coming under treatment, especially in tropical commands (TULLOCH). In the cases described by Dr. Clouston as those of the second class (see p. 858, *ante*), the chances of recovery are hopeless from the first, and all complications with scorbutus are very unfavourable. There may be diseases of a more rapidly fatal character, but there are few which entail so great an amount of suffering. When once the disease has passed into the chronic form, it slowly but not the less surely continues, by a most loathsome process, to exhaust the vital energies, until death relieves the patient of an existence rendered almost intolerable by pain, debility, and the offensive nature of the discharges (BRYSON). If the disease proves fatal in the *chronic form*, the patient generally becomes rapidly altered and prostrated by his sufferings, is strikingly emaciated, and often earnestly prays to be relieved from a life disgusting to himself and entirely despaired of by others. Death begins at the heart. The patient, on the other hand, may in a few rare instances recover; the local symptoms gradually yield, till his health and strength are ultimately restored in a moderate degree. Convalescence is slow, rarely complete; and there is perhaps no disease which makes so persistent and pernicious an impression on the human constitution as dysentery. When the skin is dry, and of a pungent heat, the tongue furred, and the thirst urgent, the urine scanty and high-coloured, and the pulse increasing in frequency—these are symptoms of increasing danger. Throughout the disease there is febrile distress, the nights are passed without sleep, or when it is obtained it is in short periods, dreamy and disturbed; and when the patient awakes he is unrefreshed and his spirits low and desponding. In the majority of cases the disease takes a favourable turn between the sixth and tenth days; the symptoms are then mitigated, the pain ceases, the number of stools diminish, and the flow of urine is restored. On the contrary, if it terminates fatally in this stage, hiccough, vomiting, a small and rapid pulse, and pale sharp features, denote the approach of death. The intellect, however, is perfect, and the patient, deploring the fate which he sees inevitably to await him, dies after a short agony.

Diagnosis.—It is difficult, perhaps impossible, in the first stage, to distinguish dysentery from diarrhœa; but the blood, the number of the stools, and small quantity of fæcal matter passed will, in times when dysentery is prevalent, indicate the true nature of the disease.

Treatment.—It is the obvious duty of the physician to direct his attention, in the first instance, to the *prevention* of the disease. He must inquire especially as to the conditions of the diet, that it be sufficient as to its animal and vegetable elements, and of good quality. Next, he ought to ensure the means of detecting the disease early—for time is of the greatest importance in its cure. He must remove the patient from the sphere of action of any of the predisposing or exciting causes, and see that his surroundings are free of all those circumstances which co-operate in aggravating the disease, the chief of which are overcrowding, bad ventilation, bad food, exposure, and intemperance. With regard to medicinal agents my friend and colleague, Professor Maclean, has written me the result of his extensive experience in India and China:—He is of opinion that the first thing to bear in mind in the treatment of tropical dysentery is, that the appearance of strength in the patient, given by the acuteness of the symptoms, is delusive. Under the use of antiphlogistic treatment the strength of the patient is apt to fail suddenly; and this is often the case, even when the treatment has been more conservative in its character. It was once the custom in India to deplete freely in this disease, either by a general bleeding or by the repeated application of leeches; but the most judicious and successful practitioners in India rarely bleed now, *even in the most sthenic forms of the disease,* and confine the use of leeches within the narrowest limits. It is certain, too, that *mercury* is yearly less and less used in India than it was; and there is much evidence to show that a corresponding reduction in the mortality of the disease has been the result. The objections to its use are numerous, —it entails great suffering on the patient, if pushed to ptyalism, aggravating his miseries, and too often permanently injuring his constitution; it has no specific action on the disease, and its cholagogue effects can be attained by remedies which are not open to such objections as can be brought against mercury. In sloughing dysentery it is followed by the worst results; and the observations of clinical observers in India have shown that individuals under the influence of mercury are not only not exempt from attacks of the disease, but are peculiarly prone to be affected by it. This is the case in a very marked degree in Asiatics (MOREHEAD and MACLEAN).

Ipecacuanha, or the *radix anti-dysenterica,* has long been used in South America in the cure of dysentery,—whence, indeed, it came. It was much used in India until the mercurial notions of James Johnston prevailed. It was again used by Dr. Twining, of Bengal, by whom it was strongly recommended, and also by Dr. Mortimer, of Madras. Twining combined it with blue pill and gentian, and used it chiefly in small and oft-repeated doses. In South America the practice has always been to administer an infusion of the bruised root,—ʒii. being infused over night in ℥iv. of water, and given early in the morning. In Peru it is given in doses of ʒss. to ʒi. of the powdered root in a little syrup and water. This practice of giving ipecacuanha in large doses has lately been revived in India with encouraging success, and, Dr. Maclean believes, with the greatest number of cures. It appears to act on the portal capillaries, and on those of the mucous membrane of the bowels, and to determine powerfully to the skin. *It is usually given in doses of half a drachm or a drachm, either in pills or bolus, or suspended in mucilage, according to the fancy of the patient. It is advisable to give an opiate half an hour before, and to withhold all drink for some hours.* Unless there be hepatic complication, it seldom happens that much vomiting is caused by these large doses; on the contrary, they are often tolerated when smaller doses are rejected. The dose should be repeated in about six hours. A sufficient interval should be allowed to intervene between the doses of ipecacuanha, to admit of the patient being sustained by nourishment adapted to the stage of the disease. Dr. Cornish, of the Madras army, has shown from official documents, that the mortality from acute dysentery in Southern India under mercurial treatment was 7·1 per cent. Since the general introduction of ipecacuanha in full doses it has fallen to 1·3. Dr. Ewart, of Bengal, has shown that equally good results have followed the same system in that Presidency. During the forty-two years, from 1812 to 1853-54, the mortality among European troops in the Bengal Presidency amounted to 88·2 in the thousand. But during 1860, when large doses of ipecacuanha were administered, the mortality was only 28·87 in the thousand. Although highly useful in some conditions, it is not to be regarded as a specific in all forms of the disease. It is more effectual in the sporadic and mild acute cases than in the chronic forms. Its use should be limited to vigorous patients with acute catarrhal diarrhœa or dysentery; or with the preliminary catarrhal stage of diphtheritic dysentery (Woodward). The large dose method of its administration, which now prevails, is to give as early in the disease as possible grs. xxv. to xxx. of *ipecacuanha* in as a small quantity of fluid as possible. A preliminary dose of *opium* may be of service in enabling the stomach to retain the *ipecacuanha.* It can be swallowed in the form of a *bolus*, by wrapping it in soluble tissue paper. The patient should then remain perfectly still in bed, and abstain from fluid for at least three hours. If thirst is urgent it may be appeased by sucking small bits of ice, or taking not more than a teaspoonful of iced water. In from eight to ten hours, from 10 to 15 grains may be again administered, with the same precautions as before. The beneficial results are manifested by the *tormina* and *tenesmus* subsiding, the motions becoming feculent, blood and slime disappearing; and often, after profuse perspiration, the patient falls into a tranquil sleep and awakes refreshed. The *ipecacuanha* may require to be continued in diminished doses for several days, with sufficient intervals between each dose to admit of food being taken; and for several nights after the stools appear normal, grs. x. to xii. of *ipecacuanha* should be still given at bedtime. Astringents in any shape during the acute stage are not only useless but dangerous (Maclean, Docker, Waring). *Blood-letting* has now been totally superseded and rendered unnecessary by the use of *ipecacuanha. Ergot* has been used in an enema to the extent of 12 or 15 grains in some bland fluid; or in 6-grain doses by the stomach, in cases of epidemic dysentery, with the beneficial result of reducing the quantity of blood in the stools (Dr. Gros, *Practitioner*, Nov., 1868). *Opium* is not to be regarded as a specific or curative agent, either in acute or in chronic fluxes. Its use should be restricted to the fulfilment of two indications, namely :—(1.) to relieve pain; (2.) to procure sleep. Chronic opium intoxication is to be avoided, by suspending its administration, or by alternating it with other remedies. Hypodermic injections of *morphia* give the best anodyne or soporific effects. It is most frequently indicated in cases of acute gastro-intestinal catarrh. In alcoholic conditions it may be of advantage to combine *chloroform* with the *morphia*, repeating the dose after an hour's interval. *Dover's powder,—Pulv. ipecacuanha composita,* is of service in full doses of grs. x. to xv. at bedtime, followed up by a five-grain pill of the same, taken every four or six hours for two or three days, or till relief is obtained. In smaller doses, frequently repeated, it may be combined advantageously with *nitrate of silver* (Waring).

In mild cases, but where the pains were excruciating and attended with tenesmus, the warm bath generally gives instantaneous relief in cases following chills, if adopted sufficiently early. Dr. Maclean directs that it be brought to the bedside, and kept at a high temperature (not under 99° or 100° Fahr.), the patient to remain in it till he feels faint. He is then to be carefully and quickly dried, put to bed, and have grs. xv. to xx. of *ipecacuanha*. . Woodward objects to the hot bath except in chronic fluxes. *Leeches*, to the number of six to twelve, applied round the verge of the anus, often affords sensible relief to the *tormina* and *tenesmus*, by unloading the portal and hæmorrhoidal veins (Waring).

In subacute and chronic dysentery, "no remedy has proved more useful than *nitrate of silver*, in doses of half a grain to one and a half grain daily, reduced to a fine powder, and conjoined with Dover's powder in the form of a pill (Dr. Waring). It has also been extensively used in the form of enema, as follows :—First throw up into the transverse colon, by means of a flexible stomach-pump tube, introduced to the extent of six or seven inches, *very cautiously and gently*, enemeta of warm water, or milk and water, to the extent of three, four, or six pints, so as to bring away any fæcal accumulations. Then follow up this practice by the injection of two and a half to three pints of distilled water, holding in solution gr. xv. of *nitrate of silver*" (Hare, Waring).

In *chronic* cases a combination of *sulphate of copper* and of *opium* is often highly serviceable (J. Brown, Raleigh, Waring), in the following formula :—

℞ *Cupri Sulph.*, gr. $\frac{1}{4}$ to $\frac{1}{2}$; *Pulv. Opii*, gr. $\frac{1}{2}$. *Make a pill or powder, of which three are to be taken daily.*

Five grains of *Dover's powder* may be substituted for the *opium*.

Solution of the pernitrate of iron is highly commended by Professor Maclean. It is astringent and tonic in doses of ♏x. to ♏xv. diluted in water. It may also be used as an injection.

Nux vomica, combined with *opium* and *iron*, may be of use (Graves). In *malarial* dysentery, full doses of *quinine* (not less than twenty grains) ought to be given in acute cases before giving *ipecacuanha*, and it should be continued till there is evidence of *cinchonism*. The two drugs should then be given in alternate doses till the characteristic good results of each are produced (MACLEAN). The bark of the root of *calotropis gigantea* (or *mudar*) has been recently used in India, and is said to be an excellent substitute for *ipecacuanha*. It is used in doses of a scruple to a drachm, is a reliable cholagogue, and sedative to the muscular fibres of the intestine, rapidly allaying pain, tenesmus, and irritation (DURANT, *Ind. Med. Journ.*, May, 1866). On the Continent the neutral salts and mild purgative medicines are highly spoken of. *Sulphate of soda* is to be preferred to *sulphate of magnesia* (MÉRY, J. J. WOODWARD). It induces a decided increase in the hepatic secretion, which the Epsom salt fails to do (RUTHERFORD and VIGNALL). *Phosphate of soda* and *Rochelle salts* are, for similar reasons, to be preferred to the Epsom salt, which is the least desirable of saline purgatives in dysentery. *Castor-oil* is similarly objectionable. It has no stimulating action on the hepatic secretion. *In the treatment of chronic dysentery* in the Seamen's Hospital, London, the Senior Physician, Dr. C. H. Ralfe, states that having watched 200 cases, he recorded in 1876 that those who lingered the longest, or at length did well, had little or nothing to owe to therapeutics, an opinion concurred in by Dr. Stephen Ward. He now abstains from action and special treatment, except to meet occasional and urgent symptoms, and places more reliance on the good effects of *rest* and *diet*. He has, however, *systematised* the administration of *castor-oil*, by giving it on stated days, at least twice a week. It removes retained scybalous fæcal matter and irritating discharges, and prevents their accumulation. Some had *bismuth* mixture in addition, others had *ipecacuanha* (12 to 20 grains), in single doses, repeated, if necessary, on second or third day whenever a relapse or return to the acute form was threatened. Sixteen out of thirty-eight cases were discharged cured; in nine of whom the duration of the disease prior to admission had averaged 18 months, and none less than five months. In painless diarrhœa and in chronic fluxes *bismuth* is of use *after evacuants*. From *mercurials* no advantages are to be expected that can counterbalance the serious objections to their use; but the benefit of the *alterative* effects of the remedy may be obtained by minute doses of *corrosive sublimate*. In certain cases of chronic dysentery Parkes found it most useful commencing with doses of one eighth to one sixth of a grain in combination with preparations of *cinchona*. It increases the hepatic secretion. Ringer gives the 100th of a grain every hour or every two hours, alike in acute and chronic dysentery.

Dr. Bryson writes that he has seen all the astringents, both mineral and vegetable, mercury both internally and externally, with many other medicines, tried without any benefit; but there were some means which were useful in relieving the more urgent and distressing symptoms. Amongst these he mentions *a well-regulated farinaceous diet*, *opium suppositories*, *anodynes*, *astringent injections*, in combination with *opium*, *cascarilla*, *resinous astringents*, and the application of *leeches to the rectum* when tenesmus was distressing, or over the course of the colon when there was deep-seated pain. An injection of warm starch (two ounces) with *laudanum* in it, will often give great relief. As much nourishment should be given in a liquid form as the patient can be got to take. Milk boiled with flour or arrow-root and barley-meal should be taken as often as possible, night and day. It should be taken cold, even with ice, and in small quantities at a time; and small pieces of ice not only allay sickness and nausea, but seem to soothe the irritability of the intestines. Strong beef-tea, or Liebig's extract of flesh, are most useful. The value of a change of climate as a curative measure is forcibly illustrated by Dr. Bryson. He says that the crews of vessels improve in health almost immediately after quitting the station where dysentery prevailed. Where sewage is applied to the soil by surface irrigation, it ought to be diluted largely with water, and deodorised by *carbolic acid.* In the scorbutic form we have a valuable remedy in the *Bael fruit,* which contains a large quantity of *tannin,* with vegetable mucus, a bitter principle, and a vegetable acid. It is much used in Bengal; and in the scorbutic form Dr. Maclean has seen it successful when all other measures have failed.

HÆMORRHAGE FROM THE INTESTINES.

LATIN EQ., *Hæmorrhagia;* FRENCH EQ., *Hémorrhagie;* GERMAN EQ., *Blutung*—Syn., *Hæmorrhagie;* ITALIAN EQ., *Emorragia.*

Definition.—*Loss of blood from some portion of the mucous membrane of the alimentary canal below the stomach. It may have its seat in the small intestines, or in the large, or in both, but probably never flows from the whole length of the canal.*

Pathology.—(*a.*) *Causation.*—The same causes which produce *hæmatemesis* will tend to bring about *intestinal* hæmorrhages. Excessive congestion and mechanical obstructions of the portal circulation from diseases of the liver, spleen, or valvular disease of the heart, and especially *cirrhosis* of the liver, are active causes of intestinal hæmorrhages. They are also apt to occur from *lardaceous disease of the blood-vessels,* and from ulcers, such as the *tuberculous, lardaceous, typhoid, or dysenteric,* or from rupture of aneurism into the bowel. Hæmorrhages also occur in the course of some specific diseases, such as *yellow fever, scorbutus,* and *malarial fever;* also from the effects of *sulphuretted hydrogen gas*—stated on the authority of the late Dr. Robert Williams, of St. Thomas's Hospital, to be a cause of epidemic hæmorrhage from the bowels among the workmen in the mines of Anzin[*] (*Encyclop. Metropolitana,* Art. "Medicine"). Children are very liable to slight hæmorrhage from the bowels while teething, and at other periods of infancy. It is most common, however, in the adult. *Hæmorrhoids* are a frequent source of loss of blood. They are caused by plethora of the abdominal vessels. Persons who indulge largely in hot tea or coffee, or who drink to excess of fermented liquors of any kind, or who habitually consume more food than is required for the support of the body,

[*] I am indebted to Dr. J. J. Woodward, of the United States army, for drawing my attention to this statement of Dr. Williams, for which I have not been able to find any other authority; Dr. Woodward refers to an epidemic described by Hallé in *Journ. de Méd.-Chir. Pharm.,* Tome IX., Paris, 1805; pp. 1, 71, and 138, in which the stools were black and green, but in which the presence of blood was not established or suggested (*Med. and Surg. History of the War of Rebellion,* Part II., Vol. I., *Medical History,* p. 358).

are liable to this affection. Hæmorrhoids often affect those who ride much on horseback, and likewise pregnant women. In a few instances they have been met with in children of six and seven years of age, but twenty to fifty-five is the more common period of life when they occur. Both sexes are equally subject to them. The causes of escape of blood from the hæmorrhoidal veins are—(1.) Collection of fæces in the rectum (constipation), tumors in the pelvis, or a gravid uterus (pregnancy). (2.) Congestion from cirrhosis of the liver obstructing the portal vein, or over-distension of it, especially from excessive use of fermented drinks and alcohol. (3.) Lung diseases, where the capillaries are compressed and atrophied, and also heart affections, tend to impede the flow of blood through the hæmorrhoidal vessels.

(*b.*) *Morbid Anatomy.*—If the hæmorrhage be considerable, the mucous membrane is blanched and colourless; or a part of the mucous membrane may be congested, and perhaps infiltrated at the affected portion; but as the hæmorrhages from the upper part of the bowel are generally capillary, the exact source or point of lesion can rarely be discovered after death. Sometimes there is ecchymosis, a considerable extent of the mucous membrane being suffused with blood. The mesenteric vessels may be gorged. When hæmorrhage takes place from ulcers, blood-coagula adhere to the edges of the ulcers which have bled, and the surface of the ulcer seems suffused with blood. The blood may be fluid, but generally it is loosely coagulated, of a chocolate brown colour, or of a dark tarry colour and consistence. *Varicosities of the rectum,* sometimes termed *blind piles,* are also sources of intestinal hæmorrhage; and the term "*hæmorrhoids*" is applied to certain bleeding tumors which form round the edge of the anus, and also at the lower portions of the rectum as far down as, but still above, the internal sphincter. The latter are called *internal* piles, and the former *external;* but the general name of *hæmorrhoid* may be taken to signify "venous dilatations and hæmorrhages occurring in or from the rectum" (NIEMEYER). The *internal tumors* or *piles* form between the inner sphincter and external edge of the rectum. They consist of a number of small soft hemispherical tumors, four to five lines in diameter, of a violet tint, and formed by venous dilatations in a diffuse thick bluish net, from which single *varices* at last appear, which may completely surround the anus. At first the varices are small, but with a broad base appearing and disappearing at intervals. At length they may attain the size of a cherry, or even larger. They are apt to be pressed through the anus every time the bowels are evacuated, and, drawing the mucous membrane after them, eventually form an elongated pedicle, so as to remain outside the anus. Once formed they never disappear. These tumors may rupture and much blood escape from them. When their walls are bluish, thin, and delicate, they may inflame and become indurated; or may ulcerate or form small abscesses, which, should they burst and cicatrise, a radical cure is brought about. But the inflammation thus excited may extend to the veins which form the venous plexus of the lower part of the rectum, and these vessels, especially those that are varicose, may become impervious and obliterated from the formation of *plugs*, *thrombi*, or *phlebolithes* in them. The mucous membrane of the rectum covering these tumors is, by the succession of inflammation and of sanguineous infiltration, at length rendered so vascular as to bleed on the slightest friction. The *external piles* are formed by the action which the sphincter exerts over the tumors thus formed at the edge of the anus, so that at each act of defecation they become compressed at its orifice, are pressed outwards, and are thus progressively elongated. They at length hang pendulous external to the anus, and in time become pediculated, hard, and fibrous. When hæmorrhoids are complicated with *prolapsus ani*, the fibres of the *sphincter* and of the *elevator ani* muscles become atrophied, wasted, and their action impaired. In the early stages the blood or lymph effused may be absorbed, and the disease subside. At a more advanced stage some become indurated and of little sensibility, while others are soft, bleed profusely, are intensely painful, and sometimes rupture or ulcerate (hæmorrhoidal ulcers). The mucous membrane, which is greatly vascular, is sometimes swollen, fissured, or ulcerated, and these fissures may penetrate so deep as to occasion rectal fistula.

Symptoms.—Hæmorrhage from the intestines may assume one of two forms—that in which the blood poured out is pure, and that in which the blood is black, pitchy, tarry-like, and grumous, when the disease is termed *melœna.* The hæmorrhage may be sudden, or preceded by a series of symptoms, as pain in the back and loins as low down as the sacrum, descending even to the thighs. The patient may suffer from colic, flatulence, loss of appetite, and other symptoms of indigestion, while the bowels may be constipated or open. Hæmorrhage from the large intestine is not uncommon, and is far from being attended with those grave consequences attached to that from the small intestines, although the quantity discharged is often great. From the large intestine the hæmorrhage may be periodical, and a relief to persons subject to headache. There are instances, however, in which the quantity passed is so great that the patient becomes completely anæmic. In consequence of the descent of the gut, a patient may pass about half a pint of blood every other day, for many weeks together, till he not only becomes sallow and dropsical, but is unable to move from bed. From the generally innocent tendency of hæmorrhage from these parts, it is of course intended to exclude those cases in which it proceeds from *diseased heart, dysentery, scurvy*, or *enteric fever*, as well as from organic disease of the intestine itself, such as *cancer* or *malignant tumors.* Hæmorrhoidal tumors produce many unpleasant symptoms, the least of which are the sensation of a foreign body just within the anus, and pain on the patient passing a stool, which is generally hard, constipated, and tinged with blood. Sometimes the hæmorrhoids are so numerous as to fill up the rectum, and should they descend so as to be grasped by the sphincter, the pain is often exquisite, and the patient is obliged to return the part with his finger, otherwise it becomes strangulated. When the tumors have become large and tense, pain is constant; and the patient can neither walk, lie, sit, nor stand. Even a pultaceous stool causes much pain on passage, so that the patient refrains as long as possible from going to stool. When inflammation attacks the hæmorrhoidal tumors the pain is often so severe as to extend to the perinæum and testicles in the male, and to the vagina, uterus, and bladder in the female. These pains are much augmented on every movement, even by lifting up the leg, turning in bed, by sneezing or by coughing. In the worst cases every attempt at defecation is distressing, and dreaded by the patient. Even sleep is at last lost, and a grave dysuria may still further add to the torment of the sufferer. The quantity of blood lost may be trifling, but in severe cases it often amounts to many ounces daily. In the former instance the patient suffers little, except from the local irritation; but in the latter he loses flesh, becomes exceedingly nervous, and may sink into a state of melancholia.

Diagnosis.—The only conditions with which intestinal hæmorrhage can be confounded, are those which result from *hæmorrhoids*, and their diagnosis is rendered certain by an examination, except in cases where they are excessively high in the gut.

Prognosis.—Hæmorrhage from the small intestines, although not necessarily fatal, is an unfavourable sign, and always symptomatic of serious disease. Hæmorrhage from the large intestines, if idiopathic, is of less moment; but if it be symptomatic, and results from *disease of the heart or spleen*, from *dysentery*, or from *organic disease of the intestine itself*, the prognosis is grave in proportion to the intractable nature of the primary affection. *Piles* have seldom any dangerous tendency, unless they cause *fistula* or *phlebitis*. When operated on, however, more especially internal piles (as by ligature), they have been known to produce accidents, such as *septicæmia* or *phlebitis*, which have terminated fatally.

Treatment.—*Ergot* is a useful remedy. (See under *Hæmoptysis*.) Dr. Macgregor, of Glasgow, records a case of *persistent periodical hæmorrhage from the bowels* which yielded to *ergot* when all other remedies had failed (*Glas. Med. Journal*, June, 1867). In hæmorrhage from the bowels in enteric fever, the *tincture of the perchloride of iron* is often useful, and if associated with much arterial action, it may be combined with *digitalis*, as in the following formula:—

℞ *Tinct. Ferri Perchlorid.*, ♏xxx.; *Tinct. Digitalis*, ♏xv.; *Aq. Menth. Pip.*, ℥iss., repeated every four hours (WARING).

In the event of failure, the *iron tincture* may be used as an enema, combined with opium, in the following formula:—

℞ *Liq. Ferri Perchloridi*, ♏xv.; *Morphiæ Hydrochlor.*, gr. ½; *Aq. Tepid*, ℥iv.; *Misce* (Dr. JOHN HARLEY).

In cases where there is a tendency to syncope from intestinal hæmorrhage, *oil of turpentine* (♏x. to ♏xv.) every half-hour or hour has proved an effectual remedy (Dr. JOHN HARLEY). The treatment of hæmorrhoids consists—(1.) In fomenting them with hot water, with the free use of Castile soap, when they are in the acute stage of inflammation, and of a dark red appearance, throbbing, and painfully burning, as if a red-hot coal had been applied to them. In this condition dusting with *calomel* has been found to give relief (BIDDLE, ATKINSON, *Practitioner*, Vol. XXIII., 1879, p. 104). (2.) If the pains continue excessive, a few leeches may be applied to the margin of the anus; and, at the same time, the *bitartrate of potash*, combined with *sulphur* and *guaiacum*, in the proportion of one part *sulphur* and of *guaiacum* to two of *cream of tartar*, a teaspoonful being taken three or four times a day, which ought to keep the bowels gently relaxed. Numerous cases have now been published which confirm the value of *glycerine*, in doses of a dessert-spoonful twice a day, in the treatment of internal and external hæmorrhoids (Dr. DAVID YOUNG, *Practitioner*, l. c., p. 251). The protruded bowel ought also to be carefully washed with a sponge and cold water *after* defecation, and the use of paper entirely discarded. To relieve the weight and tension after use of soap and fomentation, *compound gall ointment* and *belladonna extract*, in equal parts, should be freely applied every three or four hours till the pain subsides. In the chronic stage, the common *pitch ointment* is the best application (F. P. ATKINSON, l. c.) Stringent rules ought to be laid down as to diet, which should be light, and the patient limited to French and Rhenish wines. Ablution with cold water should be practised morning and evening; and some persons are sufficiently sthenic even to bear with advantage an injection of cold water after each evacuation of the bowel. In *prolapsus ani* and hæmorrhoids, Professor Cleland recommends the use of *liquor bismuthi* as an enema. A dessert-spoonful of the *liquor* is to be mixed with half a wine-glassful of *fluid starch*, and, after getting into bed and returning the bowel to its place, to introduce this enema and retain it.

OBSTRUCTION OF THE INTESTINES; ILEUS.

LATIN EQ., *Obstructio;* FRENCH EQ., *Obstruction;* GERMAN EQ., *Verstopfung;* ITALIAN EQ., *Ostruzion.*

Definition.—*An obstacle or impediment which obstructs the passage of contents through the bowel.*

Pathology.—(*a.*) *Causation,* and (*b.*) *Morbid Anatomy.*—The causes of intestinal obstruction are mainly—(*a.*) *Intus-susception,* of which the varieties are, *ileo-cœcal, iliac, colic, jejunal,* stated in the order of their comparative frequency; and they furnish more than half the cases of obstruction. (*b.*) *Obstructions due to bands, adhesions, diverticula, gall-stones, and lesions,* such as rupture of *mesentery,* and other peritoneal lesions. The small intestine is the seat of the obstacle in 94·53 per cent. of cases. (*c.*) *Obstruction due to strictures, tumors,* particularly in connection with uterus or ovaries, *volvulus, or twistings of the bowel and mesentery* (*internal herniæ*), give about 87·36 per cent. of cases involving the large intestine. Other sources of obstruction are to be found in accumulation of fæcal matter (stercoraceous tumors) and heterogeneous substances forming masses or concretions.

Intus-susception is the accidental insertion or protrusion of an upper into a lower segment of bowel. It occurs more frequently in infancy and childhood than at any other period of life. Half the ileo-cæcal intus-susceptions are infants under seven years of age, many but a few months old; and the average ages of the ileo-cæcal, jejunal, and colic respectively, are as 18·57, 34·6, and 31·4 years (Dr. BRINTON). Invaginations of the small intestines are so frequently found after death, in comparatively young and well nourished subjects, that it is probable they are formed with great facility during life, giving rise to temporary bowel derangement; but that they soon become disentangled again by the normal peristaltic movements (LOUIS, BAILLIE, MACINTOSH, HODGKIN). I have frequently observed such invaginations invariably in the small intestines, not associated with symptoms during life, and easily reduced by traction. They have occurred generally in cases where the irritability of the bowel had been increased during life by diarrhœa, with or without ulcerations of the intestines. Cases of intus-susception *in the adult* are rare—so rare, that in one of the largest civil hospitals in London (Guy's), Dr. Wilks records that "he has never seen but one case of intus-susception *in an adult*, and in this case the obstruction was never complete, and death did not occur for some weeks" (*Pathological Anatomy*, p. 292). In the *Transactions of the Pathological Society of London*, extending over the first fifteen years of its existence, there are only seven cases of intus-susception *in the adult* on record—no two of which occurred in the individual experience of any one man. The ages of these seven cases are respectively eighteen, twenty-five, thirty-two, thirty-four, forty-one, and two at forty-four years of age. In one case the symptoms continued for three months, and at last ended in recovery after the passage of a portion of ileum (*containing a polypoid tumor*) by the rectum. In another case the symptoms continued during four months, and terminated fatally by exhaustion. In my own experience I have never made a *post-mortem* examination of a case of intus-susception *in an adult*, nor have I ever seen a case of intus-susception *in an adult* during life. In the Museum of the Army Medical Department at Netley there are preparations showing the lesions and morbid relations of the parts preserved, from at least eight cases occurring in soldiers at ages varying from twenty to forty-two years of age. A case recorded by Dr. Todd, in the *Army Reports* for 1864, p. 532, is of interest, inasmuch as the dissection made by me at Netley shows that the intus-susception was associated with a large polypus growing from the mucous surface of the small intestine. The history of the case further shows that it was preceded and accompanied by severe diarrhœa. In the course of examination of the parts sent to Netley, my then coadjutor (Dr. Davidson) discovered that the mucous membrane of the *caput cæcum* was infested by the minute parasite known as the *trichocephalus dispar*. This parasite—a very minute round worm—had its head-end, of hair-like fineness, firmly fixed to the mucous membrane of the intestines, while the rest of the body was coiled upon itself and hidden amongst the mucous secretion of the gut. The natural history of this entozoön shows that it has often been associated with severe epidemics of diarrhœa. Indeed, its discovery more than a hundred years ago (1760-61) was made during the prevalence of a severe epidemic of diarrhœa (*morbus mucosus*) amongst the soldiers of the French army, associated with its presence in the *caput cæcum* of those who died. Five cases of intus-susception are recorded in the *Transactions of the Pathological Society* which are associated with *polypoid tumors* of the intestine at or near the site of lesion. *Seeds*, *fruit-stones*, and *lumbrici* may also get into the *appendix cæci*, and cause a fatal result by inflammation, ulceration, or stricture. *Gall-stones* may be arrested in the jejunum, and just above the *ileo-colic valve stercoraceous tumors* may entirely arrest all passage through the bowels until they are broken up. Such masses are almost always present in persons of costive habits, or who may be deterred from defecation from any cause, such as pregnancy, old age, hysteria, unusual sensitiveness in accomplishing the act, ulcerations, fissures of the anus, or piles. These fæcal accumulations are to be sought for in the region of the cæcum, transverse colon and sigmoid flexure, and especially in the groins. They may attain a great size, accumulating slowly. They are usually soft, kneadable, irregularly nodulated in form, and painless, except for the local enteritis they may occasion when impacted in a series of sacculi. They may cause sciatica or œdema of a limb, or prevent its being extended. Liquid fæces and a species of diarrhœa (with small pellet-like scyballæ) may coexist through a small channel running through these accumulations. *Hernia, twists,* or *strangulation by rotation of the bowel* may also cause obstruction. They usually follow sudden exertion; and may occur by the bowel—(1.) revolving on its own axis; (2.) being rolled on its mesentery; (3.) being rolled on another coil of intestine. Very little torsion is required to establish obstruction. They are most commonly met with in the sigmoid flexure, ascending colon, cæcum, and lower part of the ileum. Enteritis (of which twists may also be a result) soon follows, with a rapidly fatal issue. Adhesions and bands of lymph may constrict a portion of bowel, and cause obstruction. They are found most frequently just opposite the promontory of the sacrum; and often in females from inflammation of some of their pelvic organs, as when the pedicle of an ovarian tumor ensnares and imprisons the gut; or when the vermiform appendage adheres and incloses a loop of intestine. But these are lesions which are only revealed after death (Dr. G. H. B. MACLEOD, *Brit. Med. Journ.*, 1876, p. 645).

The *volvulus,* or portion of gut where an intus-susception exists, consists —(1.) Of the external portion formed by that part of the bowel into which the other has slipped; (2.) of the middle; and, (3.) of the internal part, composed of the reflection of the invaginated portions. As it is not always easy to follow the anatomical relations of the several layers of structures composing an intus-susception, it may be of use to give a diagrammatic outline of the relation of the serous and mucous coats of the intestine in such lesions. It is of practical importance to remember that although the parts are greatly displaced, yet the anatomical relations of the serous and mucous surfaces of the intestine are never altered. Textures of the same anatomical character are always in contact one with another, and the channel of the gut along its mucous surface is always open. That such is the case may be understood by taking the leg of a long stocking from which the toe-end has been cut off, so that the stocking may be converted into a continuous tube open at both ends. If a portion of the stocking be then drawn into the other, a correct imitation of the relation of surfaces in an *intus-susception* will be obtained. In the diagram (Fig. 56) the tube, *a b*, may be continuously traced, as indicated by the arrows. The dotted line corresponds to the *serous* surface, and the thick dark line represents the *mucous* surface of an intestine inclosing an *intus-susception.* From the outer to the *innermost* surface at the site of lesion, on cutting through one layer (*the first* of the inclosing gut) a MUCOUS

SURFACE is reached, which has a *cul-de-sac* reflection at *c*. Thus, the MUCOUS surfaces of the including and the included portions of intestine are in constant apposition, rubbing against each other. On cutting through the *second* layer of the *intus-susception*, a SEROUS SURFACE is reached which has a *cul-de-sac* reflection at *d*. Thus, the SEROUS surfaces of the includ*ing* and the includ*ed* portions of intestine are in constant apposition, rubbing against each other. At *e* the mucous canal is always more or less open in cases of simple *intus-susception;* and the orifice is invariably turned or curved to one side. It may be so firmly applied against the mucous surface of the including intestine (by the dragging of the mesentery which has been included) that the orifice may be closed, like a valve, by simple apposition and compression. In consequence of the lateral attachment of the mesentery to a line along the serous surface of the intestine, a portion of mesentery equivalent to the extent of the serous surfaces in apposition is also dragged into the containing gut, and acts as an efficient and increasing cause of obstruction. In consequence of the one-sided attachment of the mesentery, and the dragging of its parts, the included gut necessarily takes the form of a curve lying within the including intestine. It thus appears highly corrugated over its mucous surface, dragged to one side, and curved upon itself, as it lies exposed on cutting into the outermost layer of intestine. The orifice of the contained or invaginated intestine is thus turned upwards on itself, and is not to be found at what appears to be the lowermost part of the extreme end of the included portion of bowel. This dragging of the mesentery necessarily obstructs

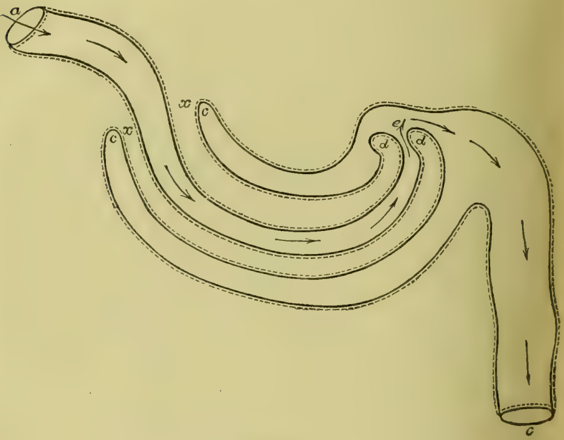

Fig. 56.

the flow of blood in the mesenteric vessels, leads to increasing congestion and the gradual effusion of blood between its layers, seen after death in the form of compact indurated masses of a dark colour when strangulation becomes complete. Blood is also gradually effused from the mucous surface of the gut. Combined with other symptoms, as strangulation increases blood continues to exude from the mucous surface of the intestine, so that small flocculi, and fluid blood mixed with mucus and free from fæcal matter, pass *per rectum* as long as the canal remains open. These symptoms are justly regarded as pathognomonic of simple intus-susception not completely closed. In some cases the pressure of the inclosed bowel on the containing gut, and the dragging of the included mesentery, are so intense that the invaginated portion has actually effected an opening by ulceration through the inclosing bowel, and so projected into the cavity of the peritoneum before death by peritonitis took place. Four specimens in the Museum of the Army Medical Department at Netley illustrate this fact in the pathology of intus-susception. One has no history; another is quoted as an example of the bad effects of purgation; the third is from a woman aged twenty-four, who had been ill ten days, when an extensive opening occurred through the inclosing bowel, and peritonitis of a severe form speedily proved fatal. A fourth occurred in a soldier aged twenty-two, in which a large mass of gut is involved, and its history is significant of the injurious influence of purgation in such cases. The man is recorded to have had persistent diarrhœa, flatulence, bloody stools, and other symptoms of intus-susception; but he was nevertheless alleged to be a malingerer. He was treated with purgatives, and lived long enough for the end of the included gut to wear a hole, by pressure and rubbing, through the substance of the containing bowel. *Post-mortem* examination, combined with a study of the phenomena of intus-susception during life, shows that the increase of the lesion takes place mainly at the expense of the *external containing* portion of the bowel; and therefore, it can readily be understood how some fixed point in the bowel is the first starting-point of an invagination. Most frequently (56 per cent., BRINTON) it is the ileum and cæcum which pass into the colon, then the colon passes into itself, so that the *appendix vermiformis cœci* becomes included. Two orifices then exist at the extreme end of the invagination—one is that of the lesser bowel, the ileo-cæcal valve, the other is the entrance into the *appendix cœci.* Two preparations in the Museum of the Army Medical Department show this arrangement of the parts. In 32 per cent. (BRINTON) the small intestine forms all the layers. In 12 per cent. (BRINTON) the colon is exclusively involved; and the rectum scarcely ever forms more than the outer layer.

In all the dissections of *invagination* whose history I have examined, they have either been associated with the diarrhœa of irritation (as from worms, undigested masses of food), or with cerebral lesions (as in the cases of children in whom invaginations are very common), or with ulcers of the intestines, or polypoid growths. In some cases no cause could be assigned for the affection; in others the disease appeared to have been excited by accidents, by carrying heavy weights, by taking injudiciously large meals or improper food, by the irritation of drastic purgatives, or by the presence of worms, or by spasms, such as result from enteritis or from lead colic (Dr. PEACOCK, *Trans. Path. Soc.*, Vol. XV., p. 117). As to how the lesion commences some notion may be obtained, and the physiology of the process comprehended, by experiments on the intestines of animals while under chloroform, or just after having been killed. If a portion of the small intestine be pinched with a pair of forceps, active circular constriction of the gut immediately commences at the site of irritation. This constriction continues for some time, and advances onwards, under the influence of the usual peristaltic action of the intestines (*vermicular-like movements*). Wave upon wave of constrictions may be made in this way to follow each other in succession, so long as the vital irritability of the intestine continues. If the advance of the constriction onwards is impeded by any cause, such as an undigested mass of food, a scybalous portion of fæces, a foreign body, a polypoid growth, or even another constriction; and, if the onward motion of the bowel fails to dislodge the obstruction, a partial invagination readily occurs; but where the obstruction is necessarily localised (as from ulcers, polypoid growths, or fixed parasites) permanent invagination commencing in the vicinity of such local lesions is still more readily induced. The mere weight of a polypoid growth necessarily favours the occurrence of invagination, by dragging down the bowel to which it is attached, and so inverting its coats. In 5 per cent. of the cases the intus-susception is so caused (BRINTON). It is then generally situated above the ileo-colic valve, the polypus having made its way through it when the spasmodic contraction of the valve would effectually prevent the spontaneous return of the invagination. So also undue distension of a portion of bowel with gas will facilitate the entrance of a wave of constriction and formation of a volvulus.

The **Symptoms** indicate obstruction and inflammation. *In the child* they mainly consist of restlessness, sudden fits of crying, and straining as if at stool, sickness, and anxiety of countenance; a discharge of mucus, more or less mixed with blood, and free from fæcal matter. These phenomena are generally, but not invariably, preceded by a sudden and violent action of the bowels. A physical examination of the belly may disclose a tumor or swelling of the intestine; and sometimes the invaginated part can be reached with the finger introduced into the rectum. *In the adult* the symptoms in cases of complete strangulation of the gut by strictures, like internal strangulation from bands of lymph, or twisting of the gut round such constrictions, come on suddenly, and if the stricture be not relieved, the case proves fatal about the fifth or sixth day. On the other hand, in cases of intus-susception the symptoms of the incarceration are by no means sudden nor rapid in their progress; or, rather, they are comparatively slower in their development and progress to a fatal issue than cases of complete obstruction by strangulation are. In cases of intus-susception the impediment from the first is partial, and in some cases the obstruction is never complete, although the symptoms may extend over many days, or weeks, or even months. One case is on record in the *Transactions of the Pathological Society*, in which the symptoms of incarceration were present during four months, and although adhesions had formed between the serous coats of the invagination, yet obstruction of the intestinal canal was never complete.

A summary of the prominent phenomena may be stated as follows, and regarded as pathognomonic of intus-susception: Diarrhœa is generally the first indication of illness. In connection with this diarrhœa and its prevalence, the existence of parasites must not be overlooked, inasmuch as they may be associated with the prevalence of diarrhœa in an epidemic form. Collapse, nausea, and diarrhœa are the next symptoms to supervene, followed by tenesmus, or a feeling of fulness in the rectum, and after going two or three times to stool *blood only* is observed *to pass*, and similar discharges of blood continue. An enormous discharge of fæces may sometimes take place after an enema, but generally accompanied by a *large flow of blood.* So long as the patient lives *he continues to pass blood at stool,* and, on occasions, scybala. Small portions of hardened fæces may continue to be got rid of, but every effort is attended by *the usual flow of blood.* Purgative remedies aggravate the symptoms. Death is comparatively slow, and hence the extensive coagula which are found in the cavities of the heart. In cases of *complete obstruction,* from *internal herniæ, stricture,* or other causes, the following phenomena are typical: "A person, perhaps hitherto healthy, experiences a sudden constipation, attended with disproportionate uneasiness, or flatulence, soon merging into pain and distension of the belly with violent rolling movement of the intestines. The distension increasing, nausea and vomiting supervene, and, gradually becoming more frequent, end by rejecting not merely any casual alimentary contents of the stomach, or the greenish, bilious, alkaline fluid commonly thrown up when this organ is unoccupied by food, but a fluid of greater opacity, colour, and consistence, with a distinctly fæcal odour. A further aggravation of these symptoms now conducts the malady to its termination. This, if fatal, is usually preceded locally by signs of paralysis, inflammation, or even rupture of the distended bowel, and constitutionally by exhaustion or collapse replacing a febrile reaction. In other cases, the obstacle being removed by nature or art (if by the former, rarely before life is in extreme danger), the symptoms subside with comparative celerity. The pain, distension, and vomiting cease; the bowels are relieved by copious stools; and the patient (if not placed in further peril by any of those conditions incidental or consecutive to obstruction) is rapidly restored to comparative health" (BRINTON, *Intestinal Obstruction*, p. 6).

The most remarkable and most characteristic symptom of *intestinal obstruction* is the fæcal vomiting (especially combined with constipation), explained by a doctrine that remained almost unquestioned since the time of Galen, until the observations and experiments of Dr. Brinton exposed the error, and established the pathology of intestinal obstruction on a rational basis. It was supposed that fæcal vomiting was effected by an *anti*-peristaltic movement of the intestinal canal; that, at a certain stage of obstruction, the natural peristaltic action of the bowel above the occluded point was reversed, so that instead of proceeding towards the anus, as heretofore, it took the contrary direction, thus impelling the intestinal contents in a similarly retrograde course, returning them to the stomach, whence they were vomited. Brinton showed by experimental and incidental proof that the notion of *anti*-peristalsis was contradicted by direct observation. A careful study of the phenomena of intestinal obstruction, as witnessed in the human subject, and as artificially produced in experiments on animals, led him to the following explanation:—

"The movement proper to the healthy intestine is a circular constriction or peristalsis which, travelling slowly and intermittently down its muscular wall, propels its contents in a direction from the stomach to the anus. And when any part of the intestine has its cavity obliterated by an immovable mechanical obstacle, its contents, propelled by such a peristalsis, are stopped at the obstructed point. Here they gradually accumulate, so as first to fill and then to distend a variable length of the canal with a more or less liquid mass. But a peristalsis engaging the wall of a closed tube filled with liquid, and falling short of obliterating its calibre, sets up two currents in that liquid—one at the surface or periphery of the tube, having the direction of the peristalsis itself, and one in its centre or axis, having precisely the reverse course. Those particles of the liquid which are in contact with the inner surface of the tube are propelled onwards by the muscular contraction of its wall. And this propulsion is necessarily accompanied by a backward current in those particles which occupy the axis or centre of the canal."

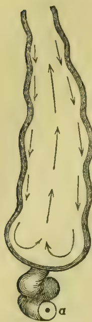

Fig. 57.

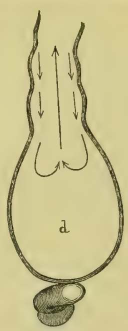

Fig. 58.[*]

Fæcal vomiting is thus shown to result from the reflux of the intestinal peristalsis—a backward current in the liquids occupying the centre of the tube. The ordinary course of intestinal obstruction thus divides itself into two stages. In the first stage the healthy actions of the bowel are continued; in the second stage they are arrested or utterly and permanently annihilated by paralysis, enteritis, or peritonitis. In the first stage, abnormal *distension* of the intestine can generally be felt through the yielding wall of the belly—as a condition of fæcal vomiting—from the commencement, and continuing through all the stages a persistent physical sign. Hence the accumulation of intestinal contents immediately above the obstructed point may be detected as a slight fulness by palpation, and as a much more definite dulness by percussion, when many of the other indications of obstruction are scarcely perceptible, or even absent. The movements of the obstructed intestine, accompanied with violent gurgling, may thus be traced rising visibly against the walls of the belly covering the obstructed tube, "in coils that may be fancifully compared to those of a writhing serpent," until paralysis and collapse usher in the fatal issue.

The character of the pain in *obstruction* varies from a feeling of weight to violent suffering. It is sometimes sudden, and often rises to great intensity in a very short time. It is distinct from the burning pain of peritonitis. It is usually intense in intus-susception, and in the impaction of gall-stones; somewhat less marked in the obstruction produced by twisting of the bowel, or by bands and adhesions; scanty in the obstruction of stricture, and almost absent in the obstruction caused by the impaction of fæces in the large intestine (BRINTON). The seat, character, and the amount of pain may coincide very exactly with the seat of lesion. It is at first like colic, centring about the umbilicus; but when inflammation is developed the accumulation of fluids and air causes much distress from the great distension: and the pain is then widely diffused. If with symptoms of *obstruction* we find an elongated cylindrical (sausage-like) tender movable tumor, lying in the axis of the bowel, or across the lower part of the belly; and if we discover on inserting the finger into the rectum a body which feels like the *os uteri*, which recedes from and again returns on the tip of the finger, the diagnosis of an invagination is complete (Dr. G. H. B. MACLEOD, *Brit. Med. Journ.*, 1876, p. 645).

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[*] Figs. 57 and 58: Diagram to illustrate the peristalsis of an obstructed bowel (after Dr. Brinton).—Fig. 57. Stage of moderate distension, with forward and backward currents, as indicated by the arrows traversing the whole tube above the obstacle; (*a.*) Contracted segment of intestine below the obstacle. Fig. 58. Stage of extreme distension, in which (*d.*) the dilated and paralysed segment above the obstacle is scarcely engaged by either of these currents.

Diagnosis is mainly differential between intus-susception and other forms of alvine obstruction. The colic is extremely severe, and the remissions of pain are followed by exacerbations, which increase in violence with each repetition. Physical diagnosis ought to be had recourse to in every case. From hernia cases of *intus-susception* are to be distinguished by the absence of the hernial tumor at the respective abdominal apertures where hernia is usual. These apertures should be carefully examined. A tumor within the abdomen is a physical sign of the greatest value; and is not often absent, though easily overlooked, as it may be of small size (BRINTON). The condition of the rectum ought always to be ascertained by a digital examination. The vomiting is less urgent in proportion as the obstruction is lower down in the bowel; if the duodenum is involved, vomiting is almost incessant. Dr. Osborne, of Bitterne, brought me the stomach and intestines of a child who died of this affection; and the relations of the parts involved were peculiar in this respect, that in the colon (which contained the volvulus) the mesentery of the duodenum and the *adjacent wall* of that portion of gut were dragged in. Vomiting was a marked symptom.

It is necessary to distinguish between ileo-cœcal, or colic invagination, and that of the small intestine. The former is distinguished from the latter by—

"(1.) The prominence of tenesmus, which is rarely present in any marked degree where the small intestine only is implicated; (2.) the greater size and fixation, as well as the different site of the tumor, which, if large, generally proceeds towards the left side of the hypogastric or left iliac region; (3.) the subordinate share taken by hæmorrhage, which, instead of copious bleeding by stool and vomit, is often little more than a scanty admixture, scarcely sufficient to tinge the mucus passed from the bowels, with violent and frequent straining by the patient; (4.) the still more subordinate share taken by obstruction, which not only seems to be often anticipated by death, as regards any complete symptoms of its presence, but to be really absent, owing to the patulous state of the axis of the invagination; (5.) the presence of the end of the invagination in the rectum" (BRINTON).

Obstruction of the small intestine is chiefly characterised by—(1.) The umbilical seat of the pain, which is also early and severe; (2.) vomiting is early, severe, and frequent: obstruction is more rapid, constant, and complete in the small than in the large intestine.

Prognosis is not always hopeless, but is nevertheless most grave. Exclusive of hernia, Dr. Brinton estimated (from an analysis of 12,000 necropsies) that obstructions of the intestine cause about 1 in every 280 deaths from all diseases indifferently; and the chief varieties of obstructions have to each other the following proportionate frequency:—Intus-susceptions or invaginations, 43 per cent.; obstructions by bands, adhesions, diverticula, or peritoneum, external to the bowel, 31½ per cent.; strictures (including a few tumors) involving the intestinal wall, 17½ per cent.; torsion of the bowel on its axis, 8 per cent. The cases which recover are almost invariably chronic or protracted ones. The longer he lives the better are the prospects of recovery. In the fifteenth volume of the *Transactions of the Pathological Society of London*, Dr. Peacock gives an account of the passage of a large piece of bowel by the rectum in a case of invagination, followed by recovery. He also refers to eighty-eight cases of intus-susception in which portions of bowel are reported to have passed by the rectum. Those which end by expulsion of the *volvulus* have a duration of from twice to thrice as long as the fatal cases; and it is not till the second, third, or even sixth week that the remission of symptoms occurs which announces the relief of the obstruction, and which often precedes by a day or two the first healthy alvine evacuations.

The distension of the involved bowel measures not only the danger, but the rapidity of the fatal issue; and the cases of most serious import are those which, commencing in the small intestine, involve the ileo-colic valve. The majority of fatal cases are those in which the cæcum and ascending colon have swallowed up, as it were, a large portion of the small intestine. The danger may not be at first appreciated, because the obstruction is never complete at first, as we know from the nature of the lesion as well as from the accurate history of cases. The bowel at first is merely incarcerated by the invagination, and it is not until the middle and internal portions with their contained mesentery become compressed, constricted, and ultimately strangulated, that complete obstruction ensues. Till this occurs, the continuous expulsion of bloody mucus from the central tube of the inversion goes on. Inflammation and sloughing thus commence as the parts become subjected to more and more increasing pressure. The early establishment of acute inflammation is the worst feature which can arise in a case, and points to an early fatal issue. If the obstruction is high up and not relieved, death may take place within a week; but low down the patient may survive for weeks.

Two forms of inflammation prevail—namely, one serous, between the opposed peritoneal surfaces, commencing at the angle of reflection of the middle on the external layer (*x*, *x*, *x*, in the diagram, p. 872), at the part where the one portion slips into the other. It is here that the peritoneal surfaces of the invagination commence to adhere; and up to the period of adhesion the ancient remedy, inculcated by Hippocrates, of injecting air into the great gut by a long tube, introduced *per rectum*, has effected the greatest number of cures—forcing, by gentle, persistent, and equal pressure, the invagination backwards, and so causing it to be undone. The other form of inflammation takes place between the two opposed mucous surfaces (*c*, *c*, in the diagram). At this angle of reflection an abundant white leucorrhœal-like secretion (corpuscular elements of inflammation with mucin) commences very early to be discharged by the rubbing of the opposed surfaces, and eventually the inflammation (mucous and serous) may be so destructive, that ulceration and sloughing of the whole invagination may be the consequence. In this way continuity of the canal may be restored—the middle part of the volvulus having passed, *per rectum*, as a slough. Dr. Brinton believes this favourable termination occurs in not less than one in every two or three cases of intus-susception. On an average, separation of the slough is not complete before the eighth day, and the dead portion of bowel is rarely expelled *per anum* before the tenth or twelfth day, and is sometimes only prevented by the death of the patient on the fifth or sixth day. Of such cases eighty-eight are now on record—an analysis of which, by Dr. Peacock, is to be seen in *Path. Soc. Trans.*, Vol. XV., p. 114.

Treatment.—In all cases of *intus-susception*, whether occurring in children or in adults, the administration of purgative medicines tends to aggravate the lesion and the symptoms. Accordingly the rule of practice is absolute—namely, "to withhold all purgative medicines from the commencement in cases of intus-susception." The bowel being incarcerated, the stimulus of purgation proceeding from above downwards is quite unable to undo the incarceration of an intus-susception. A purgative acts injuriously as a stimulus which cannot be obeyed; and its obvious tendency is to increase the peristaltic action of the bowels, and therefore to increase still more the invagination. That such is the case will be readily understood by a physiological consideration of the phenomena of intus-susception; for although it is not always easy to account for the first beginning of an invagination, yet physiology enables us to understand how, an invagination once begun, the lesion tends to increase—(1.) From the peristaltic action of the bowel, greatly stimulated and increased by irritation of every kind, so long as tonic irritability continues. (2.) From the spasmodic action of the part of the gut *above* the invagination, preventing spontaneous return. (3.) From the invagination being thus completed, it continues permanent, tenesmus occurs, and the violent and repeated contractions of the abdominal muscles tend still more to maintain and increase the lesion. The constant motion and pressure of parts one upon another in some cases is so great that the end of the invagination has been known to penetrate through the walls of the inclosing bowel, so as to appear in the cavity of the peritoneum. The chief indications are—(1.) *To prevent distension*, by reducing, in every possible way, the quantity of food and drink, restricting the latter to small but frequent sips (preferably through a long straw or tube) of cool iced liquids. Food is to be given, as strong beef-tea, soup, or milk, with equal frequency and caution. Small doses of alcohol (as brandy with water, or soda-water) is to be alternated. But if any repugnance exists to food or drink, or if vomiting is excited by these articles, the amount given must be reduced. Water, milk, and gruel are to be given freely in often-repeated enemata. (2.) *To assuage pain and to mitigate excessive peristalsis* suggest the same kind of remedy—namely, *opium,* which is to be given continuously and alone in the solid form, preferably as an *extract.* The limit of the dose is indicated by the comparative arrest of pain, the approach of narcotism, and decided contraction of the pupil. *Belladonna* is only of use as a remedy to diminish the straining peristalsis. It may be given *combined with opium,* as *two* parts of *extract of opium* to one, two-thirds, or even one-half part of *extract of belladonna* in a pill. *Enemata* are useful as a mechanical aid to removing obstruction. They may gradually distend the bowel at the site of obstruction, so as to effect such a change in its position and arrangement as may release the impacted portion. The administration of enemata is only safe and efficient if undertaken by a person of competent skill. The quantity of fluid must be injected little by little, and must be retained as long as possible; and the patient must resolve to tolerate some pain in reaching that climax of distension at which only enemata are calculated to relieve obstruction. In cases where the *intus-susception* is in the large intestine, inflation of the bowel with air, as originally suggested by Hippocrates two thousand years ago, in his third book (*Περι Παθων*), has of late years been revived and adopted, first in America, and subsequently in this country (GORHAM, in *Guy's Hospital Reports,* and *Med.-Chir. Trans.,* Vol. IX.) This Hippocratic remedy has undoubtedly been more successful than any other. Of twenty-eight cases, the details of which were collected by Dr. Osborne, there were only *seven* recoveries; and three of these were effected by inflation of the colon with air. Dr. Murphy and Mr. Erichsen have borne strong testimony to its value in discussions on the subject; and the former had recourse to the operation on one of his own children with success (MS. Notes of Dr. OSBORNE). For its success, the remedy should be employed at an early period, before there has been time for adhesion between the contiguous surface of the volvulus. My friend, Dr. David Greig, of Dundee, has had recourse to this method of treatment in numerous instances with perfect success, and has published an account of his experience in the *Edin. Monthly Med. Journal* for October, 1864. By means of the ordinary elastic enema tube, fitted to the pipe of a small pair of bellows, he was able to pass a considerable quantity of air into the rectum, continuing the process till the belly showed signs of considerable distension, and even till uneasiness prevailed. Its beneficial action is indicated by relief of the urgent symptoms, such as straining and vomiting; and gradually a fæcal evacuation is obtained from the bowels. At the same time warm fomentations are to be applied to the belly. The use of large enemata, with manipulation, has also been recommended. A long stomach-tube is to be passed as high up the colon as it will go, and the anus being firmly compressed round it, warm water is to be slowly injected, so as to distend the bowel as much as possible. When the fluid is allowed to come away, the abdomen should be pressed upon with the hands, so as to move about the coils of intestine (TANNER). The earlier any of these remedies are had recourse to, the greater will be the chance of recovery; and the patient may be put under the influence of chloroform to facilitate the manipulations. Gastrotomy, *abdominal section,* or, as it is now called, *laparotomy,* has been successful in many cases where other remedies have failed to give relief. Dr. Hilton Fagge and Mr. Howse have related such a case (*Med.-Chir. Trans.,* Vol. LIX., 1876). The operation was advocated by Benjamin Philips (see his paper in *Med.-Chir. Trans.*, Vol. XXXI.) The cases for which it seems suitable are in obstructions from bands, diverticula, also intus-susception, and the like lesions affecting the small intestine. The object of the operation is to divide the cord-like cause of strangulation (BRINTON); or to undo an invagination, all of which may be now easily accomplished under the antiseptic spray. If the bowels be much distended with air they may be punctured with a small trocar and canula. *Stercoraceous accumulations* must be mechanically removed. Repeated enemata of warm water, delivered through a long tube, will gradually permeate and disintegrate the mass; and when it begins to break up, continued ʒii. doses of Epsom salts, with a grain of *quinine*, in *aromatic sulphuric acid* and *infusion of gentian*, will gradually bring the bowels to a normal state of activity.

DIARRHŒA.

LATIN EQ., *Alvus Soluta;* FRENCH EQ., *Diarrhée;* GERMAN EQ., *Durchfall*—Syn., *Diarrhöe;* ITALIAN EQ., *Diarrea.*

Definition.—*A frequent discharge of loose or fluid alvine evacuations, without tormina or tenesmus* (GOOD, COPLAND), *in which the intestinal lesion varies from a mere condition of irritation, expressed by the increased and modified secretion (beyond which it does not progress), to those cases in which there is actual inflammation or other lesion of mucous membrane, the latter being a higher degree of the former, the cases passing from one to another by insensible degrees* (J. J. WOODWARD).

Pathology.—(*a.*) *Causation.*—This affection is rather a consequence or a symptom of certain pathological states than of itself a disease, and, therefore, it is difficult to give it a fixed place in Nosology. In many cases it is a natural effort to get rid of some irritating material which has been passed from the stomach in such a form that it cannot be made available for digestion and nutrition. The diarrhœa, or bowel flux, in such cases is an effort of nature to wash the irritant away. On the other hand, the excessive secretion may be due to local lesions which can be demonstrated after death—*e.g., Catarrhal inflammation, follicular disease, lardaceous disease, enteric fever, and cholera.* The irritant material passed through the stomach, and such local lesions, are causes of increased flux or diarrhœa. It is sometimes a consequence of sudden mental impressions, as of fear, or unpleasant news; or of exposure to cold. It is a characteristic symptom of *malignant cholera*, concurrent with whose presence it has often been observed that there is a widespread prevalence of diarrhœa. The majority of cases are not to be regarded as *miasmatic*, but as local diseases from rritation of the bowel. *Summer cholera* (already considered) is different rom a mere bowel flux. It is a disease affecting the individual in his ntirety. The diarrhœa associated with cholera is a *choleraic diarrhœa,* ubordinated to *malignant cholera,* and part of the phenomena of that isease (*Brit. and Foreign Med.-Chir. Rev.*, Oct., 1869, p. 361). Large odies of men exposed simultaneously to similar morbific influences may resent a number of characteristic phenomena in common, so that the cases of diarrhœa resemble each other as well in the grade of the attack, as in the constitutional condition of the patient and the complications. Hence various grades of diarrhœa become epidemic, from simple looseness without pain to the severest inflammatory forms, with coexisting constitutional disturbances, more especially resulting from *malaria* or *scorbutus* (WOODWARD, l. c.) Thus many agents, of a moral and physical nature, cause diarrhœa; and there are also many morbid poisons, and medicinal substances in excessive doses, which bring about this state. It is generally the immediate result of unwholesome diet, excess in food or drink; animal or mineral or vegetable poisons; cold, wet, fatigue, exposure; and various functional derangements of the biliary and gastro-intestinal apparatus.

(*b.*) *Morbid Anatomy.*—It is often a morbid excess of function, rather than any disease of structure, unassociated with any definite specific lesion beyond simple irritation of the mucous membrane. The lesions whether mild or severe, are generally seated in the cæcum and colon; but more or less extensive tracts, especially of the ileum, are also involved. It is an error to suppose that diarrhœa is confined to the small intestine and dysentery to the large. The real nature of the intestinal lesion in acute diarrhœa has hitherto been very imperfectly considered, or altogether neglected, probably because the disease so rarely terminates fatally in civil life; so that opportunities for *post-mortem* examination are comparatively unfrequent. The diarrhœa which occurs in the course of other disorders is not to be regarded as a mere symptom of the primary affection, but is itself a secondary disease or complication, comparable to other intercurrent irritations and inflammations. There seems to be conclusive evidence to show that much of the intestinal catarrh described by the common name of diarrhœa is associated with an *erythematous* congestion of the mucous surface of the intestine, extending in patches of a few inches to several feet in extent in all parts of the canal, and rarely attended by increased arterial vascularity of the submucous tissue. They are more common in the ileum than the jejunum, and still more frequent in the colon. The large bowel is generally more extensively diseased. When the symptoms of such congestive states are manifest during the progress of other diseases which terminate fatally, there may frequently be observed, besides the congested state of the mucous membrane, a marked increase of vascularity in other parts, such as the gastro-splenic omentum, mesentery, and glands, or infarction of the gastric glands, associated with congestion of the stomach generally. These phenomena, for the most part, are associated with a congested state of the hepatic system; and, occurring in a person otherwise in good health, give rise to symptoms which have been considered as a disease, and variously named *enteria*, *enteritis*, *erythemoidea*, *diarrhœa mucosa*, seu *catarrhosa* vel *catarrhale*. During the autumn and winter months in this country it is common to meet with such cases of disordered bowels in adults; and in children at any season, characterised by frequent fluid alvine discharges, and associated with extensive superficial irritation of the mucous surface.

Cases of diarrhœa with collapse have been described by L. Buhl, E. v. Wahl, v. Recklinghausen, Zalesky, Waldeyer, and Gerald Yeo, associated with the presence of a *bacteria* development in the stomach and intestines under the name of *mycosis intestinalis*. The prominent symptoms were vomiting and diarrhœa, "a brisk catarrh of the mucous membrane of the stomach and the intestines, or both," occurring suddenly during apparently robust health, quickly followed by lividity of hands and face, collapse, and death in from two to six days. The mucous membrane of the pharynx, larynx, and trachea were excessively red. The lungs were filled with blood, and of a dark colour. Œdema of the areolar mediastinal and sub-peritoneal tissue existed, and also of the neck and larynx. The right cavities of the heart were filled with blood. The peritoneal cavity contained much turbid fluid; and the connective tissue along the backbone, progressing downwards and forwards to the sides of the abdomen, was characterised by blood-injection, ecchymosed and œdematous. The stomach and the intestinal canal were similarly injected with venous blood, œdematous and ecchymosed. Necrotic hæmorrhagic patches, or diphtheritic sloughs, were found in the mucous membrane of the stomach and intestines, in which micrococci and bacteria were found in great abundance, as in the sloughs of diphtheritic dysentery. The mesenteric glands were enlarged, and an ecchymosis spread around them and between the layers of the mesentery. The lymphatic glands of the thoracic and abdominal cavities were swollen, thick, and permeated by hæmorrhagic infarction. Furunculoid hæmorrhagic patches—roundish (three-fourths of an inch in diameter)—of increased thickness and prominence, existed upon the mucous surface of the stomach, having an umbilicated depression, and a feebly yellow-coloured centre, resembling a flat scab or commencing ulcer. Similar patches, to the number of fifty-nine or sixty, were found from the pylorus to the ascending colon—the smaller patches in the small intestine resembling œdematous flabby swellings, slightly injected. These lesions were different from those of any known disease. Microscopic sections of the affected parts (proceeding from the innermost surface of the mucous membrane towards the peritoneal coat) showed black particles of granular pigment; and to the *villi* of the affected parts there adhered groups of zooglœa, the larger ones having the central position, with smaller groups extending peripherically towards the healthy tissue. These molecular corpuscles, of oval shape, which could only be recognised by powers magnifying 800 to 1,000 diameters, were mostly imbedded in gelatinous connective tissue. Buds could also be distinguished (but only with the very highest powers) at the sides of some, as in yeast-cells. The entire substance of the *villi* was permeated with these corpuscular groups, and rarely could *mycelium* be noticed outside these groups of fungous structures, known as *schizo-mycetes* (BUHL; see p. 150 to 153, Vol. I.) The submucous tissue was traversed by *mycelium-fibres* of considerable length, but without producing any change in the elements of the tissue itself, except that a considerable number of pus-cells (colourless blood-cells?) were infiltrated into the tissue, and the blood-vessels contained less coloured than colourless blood-corpuscles. The fungous formation had grown from the surface of the mucous membrane towards the interior layer, the thick felty mass of *mycelium-fibres* penetrating the submucous tissue (BUHL). The fungous ibres (hyphens) were also found penetrating the mesenteric and portal veins, floating among the blood-corpuscles. The blood of the whole body ontained abundantly isolated small bodies (*conidia*), and the number of olourless blood-corpuscles was evidently increased (*lecuocythæmia*). For one *white corpuscle*, Buhl recognised 50 *red ones*, and 350 *conidia*. The mesenteric and lymphatic glands were so much infiltrated by the *mycelia* as to obliterate the structural part of the gland. The presence of isolated *conidia* in the general blood, and of *mycelia* in the portal vein, proves that they passed from the mucous membrane through the venous radicles and lymphatic vessels. They were found in varicose lymphatic vessels of the serous membrane of the stomach. The œdema, the hæmorrhage, the exudation into the abdominal cavity, and the diarrhœa were due to the mycelia entering the mesenteric veins, the lymphatic vessels, and glands (Bhul).

In this form of diarrhœa, Dr. Waldeyer recognises evidence of embolic lesions from the enormous quantity of minute *zooglœa*, in connection with the vessels of the skin, heart, liver, intestine, kidneys, and lymphatic glands. The smaller vessels were blocked up by the fungoid elements, which led to hæmorrhagic infiltration of the parts, to which the ecchymoses are referable. Finally, he regards the disease and the diarrhœa as a form of *malignant pustule* (*Milzbran*) transferred from animals to man. One of his patients was a cattle-feeder; and the swelling of the lymphatic glands and spleen, with the carbunculous affections of the mucous membrane of the stomach and bowels, are constant symptoms of affected cattle, of the enteric fever of horses, and of cattle plague. In the blood of these animals "*bacteria*" are said to have been found (*Zeitschrift fur Biologie*, Vol. VI., 1870; Virchow, *Archiv.*, Vol. XXX., p. 366; 52, 1871, Part II., p. 541; and *Handbuch der Speciellen Pathologie*, Vol. II., p. 387). Workers in hair are also liable to this affection (see p. 747, *ante*, also Woodward's *History of the War of the Rebellion*, Med. Vol., Part II., p. 283).

The solitary follicles of the bowel are generally enlarged in diarrhœa to the size of pin-heads. They appear as whitish or yellowish elevations, surrounded by a little circlet of increased vascularity. In the colon they are generally sessile. In the small intestine they may have contracted necks, and so project from the surface of the gut like sago grains (*sago-grain appearance*). The intestinal catarrh of Asiatic cholera furnishes the best examples of this appearance. Tumefaction and hypertrophy of villi and pigmentary deposits (*shaven-beard appearance*) in patches of Peyer are also usual appearances in cases of long continuance. The follicular enlargement is most pronounced in the cæcum and descending colon, where it seems first to commence and to extend upwards into the small intestine. In prolonged cases small ulcers may be seen on the apex of each in the colon, and so the case may pass into dysentery.

Symptoms and forms of Diarrhœa.—Nosologists have arranged the disease into varieties founded on the characters of the discharges; but as these do not depend upon definite pathological states, the classification may, at first sight, appear to be of little use. Nevertheless, the characters of the discharges furnish important indications for treatment. The most common appearances are due to the predominance of fluid feculent matter, or of bile, mucus, serum, chyle, or to undigested masses of food passing unchanged, and giving rise to what is termed a "lientery." But the discharges are more often of a mixed kind, made up of several of those characters occurring in the same patient.

The following forms of diarrhœa require notice:—1. *Diarrhœa of Irritation* comprises most of the cases denominated *feculent* by authors. It is induced by stimulating or irritating substances received into the stomach, excesses in eating or drinking, or even by a small quantity of unwholesome food, or by poisoning from animal effluvia and from certain mineral poisons, or what constitutionally disagrees with the patient. In infants it is brought on by unwholesome conditions of the milk, such as the persistence of *colostrum* in it. Nausea, with severe griping pains before each evacuation, a foul, loaded tongue, copious feculent stools, watery, mucous, or bilious, and becoming frothy, are characteristic phenomena.

2. *Diarrhœa from Increased Vascular Action.*—This variety is caused by whatever induces a greater flow of blood to the intestinal mucous surface, and at the same time lessens or obstructs the cutaneous elimination of fluids; the application of cold to the cutaneous or pulmonary mucous surfaces, or to both at once; cold acid drinks, or ices taken when the body is overheated; suppression of perspiration or of accustomed discharges; checked menstruation or lochial discharge. *The evacuations* are watery or serous, with mixed feculent matter, and exhibit every shade, from a dark brown, greenish-brown, to a pale greyish or whitish colour. They may contain pieces of thick gelatinous mucus, or thin, glairy, stringy mucus; or whitish albuminous flocculi, or large membranous or albuminous shreds or flakes may present a mould of the internal surface of the gut. After acute diarrhœa has lasted a short time, the *odour* of the stools becomes strikingly modified, when they lose their feculent character and become watery or mucous. A nauseous smell prevails in the advanced stages of grave cases; and in fatal cases for a short time before death, the *odour* is so offensive and characteristic as to have been called "*cadaverous.*" Even if the stools are not offensive when voided, they soon become so—putrefaction occurring sooner than in normal stools, on account of the great proportion of water and the absence of bile in them. When irritation predominates toward the upper part of the intestine (duodenum, for instance) the symptoms are an inclination to sickness, speedily followed by copious feculent discharges; the surface of the body is easily affected by cold; the individual is chilly, and may shiver. There is thirst, and a feeling of internal heat over the epigastric region. The functions of the liver are disordered at an early period, as expressed by the dull yellowish colour of the conjunctiva and sallow darkness of the complexion, especially round the eyes. The tongue is moist, but viscid, clammy and furred. The appetite is completely lost in the first instance. The skin is dry, and the palms of the hands and soles of the feet become unpleasantly hot and burning. The bowels become distended with flatus; and there is an uncomfortable sense of distension, incapacity to expel the air, and occasional griping of the bowels, which are constantly producing a rumbling noise (*borborygmi*). The stools, at first large, feculent, and consistent, subsequently become watery, and even mixed with blood; then *tenesmus*, or a tendency to strain at stool, comes on, and may increase as the case may pass on to dysentery. Undigested articles of food are also passed—the characteristic symptom of *lientery* or *diarrhœa crapulosa* of the older authors. The belly is not painful when pressed, as in peritonitis or acute inflammation of the bowel, but there is often a deep-seated sense of uneasiness. This state soon terminates, in general favourably. It is

especially brought on by exposure to changes of temperature in humid and moist weather, by wet feet, damp beds or clothing; by improper dieting at irregular times; by over-feeding, especially in summer; by certain articles of food, imperfectly fermented malt liquors, acid wines, and sour unripe fruits, drastic purgatives, and various mineral poisons. A diarrhœa of *whitish stools* is often extremely persistent, and may reduce the patient to the verge of death. It may supervene after injuries which induce cerebral concussion, or it may come on after dysenteries have been cured. There may be no fever, the appetite may be good, and digestion may seem tolerable, but emaciation and weakness become daily more and more marked. At first the motions only increase to two, in place of the usual single daily one. Afterwards the calls to stool increase, so that, during the twenty-four hours, there may be eight or ten in the day. The desire to evacuate the rectum comes suddenly; and the stools are apt to pass involuntarily, preceded by no premonitory sensations, and consisting merely of two or three table-spoonfuls of muco-gelatinous matter, resembling thick milk or puriform jelly-like fluid. A similar state of mucous membrane lining the small gut gives rise to a white secretion from its surface; so that white milky stools are observed to flow—*Diarrhœa alba* of Hilary—a form of bowel disease sometimes epidemic in Barbadoes. In addition to the symptoms noticed in the former variety, there is, in this form of diarrhœa, a dry, harsh skin, with increased temperature, a flatulent state of the bowels, a small, frequent, constricted, but soft pulse, a tongue furred or loaded towards its root, with red edges and point, and scanty, high coloured urine. In infants this variety is known as the "watery gripes," and often precedes fatal exhaustion.

3. *Diarrhœa with Discharges of Unaltered Ingesta* is essentially an atonic form, very different from the last variety. It corresponds to the "*diarrhœa lienterica*" of the older authors. The most characteristic phenomena are due to the almost total suspension of the digestive, assimilative, and absorbent functions, the *egesta* often differing but little in appearance from the *ingesta*. It occurs more frequently in children before the period of the second dentition than at later periods. It is frequently the consequence of inflammatory irritation of the alimentary mucous surface and disease of the mesenteric glands. It seems as if the stomach, intestines, and mucous membrane of the alimentary canal had lost their true tone or vital energy. The flux results, in the first instance, from impaired digestion. This was a frequent form of diarrhœa amongst the soldiers in the Crimea (LYONS). The soldiers themselves observed it, and were in the habit of saying, "It is of no use eating, as our food passes through us in the same state as it goes in." The appetite is usually voracious; and when this form of diarrhœa continues long, the debility becomes extreme. When death takes place, it is from stupor and exhaustion.

The *quantity* of fluid passed from the bowel is always greater than the normal amount (five to ten ounces). The liquid stools may amount to forty pounds in the twenty-four hours (DALMAS, quoted by WOODWARD, l. c.). The absolute amount of solids is increased.

Diagnosis.—Acute diarrhœa is to be distinguished from the milder form of dysentery by the absence of *tenesmus*. Acute griping pains (*tormina*) are sometimes present.

The **Treatment** of these three forms of diarrhœa may be founded on the following indications:—(1.) When the *tongue is clean*, the pulse quiet, and all constitutional reaction absent; (2.) when the *tongue is white* and coated, the pulse accelerated, some fever present, and the pain or soreness constant and increased by pressure. *The stools* in either case may be black, green, white, or mixed with blood indifferently. When the *tongue is clean*, if the disease be quite insipient, *one* dose of an opiate may be given, combined with a gentle cathartic. The form may be *one grain of opium*, combined with a *drachm of compound rhubarb powder*, or with *three to five grains of calomel*. To remove any offending matter that may be present, the action of the bowels may be aided by *castor-oil*, or a *saline cathartic*, such as a *seidlitz powder* or *compound senna mixture*. Sometimes it may be advisable to omit the *opium*, and to combine *antacid* remedies with the laxative, as in the following prescriptions:—

℞. Sodæ Bicarbonatis, Hydrargyri cum cretâ, ā ā gr. ii. ad gr. v.; Magnesiæ Carbonatis, gr. iii. ad gr. vi.; Pulv. Rhei, gr. v. ad gr. viii.; *misce.*

Or—

℞. Sodæ Bicarbonatis; Pulv. Rhei; Pulv. Calumbæ, ā ā gr. iv. ad gr. vi.; *misce.*

The administration of such a powder may be repeated at intervals—twice or thrice a day; and *ipecacuanha* in small doses (a quarter or a sixth of a grain) may be advantageously combined with each dose. Other more astringent remedies may be administered if diarrhœa persists. In many cases a drachm of *syrup of poppies* after each stool is sufficient to arrest it. In severe diarrhœa a scruple to half a drachm of the *compound chalk powder*, in some aromatic (peppermint or cinnamon water), every four or six hours, may be used whether blood be or be not in the stools. If the opiate and aromatics should prove insufficient, it may be necessary to add to each dose some of the class of pure astringents, as a drachm of the *tincture of kino*, or of *catechu*, or of *hæmatoxylon*, or of *iron*. The following formula (*Diarrhœa Mixture*) has been found of service:—

℞. Conf. Aromat., ʒiij.; Sodæ Carb., ʒiss. (Bicarb.); Tinct. Opii., ℥i.; Æther Chloric, ʒiij.; Ol. Caryoph., ♏ xl.; Mucilag. Acaciæ, ℥i.; Aquæ Destil., ad ℥v.; *misce.* One teaspoonful for a dose. This quantity may be given every two or three hours, or every hour, or every half hour, should the purging continue.

Absolute rest in the recumbent posture must be maintained, and warmth applied to the surface of the abdomen. Bland demulcent food, such as arrow-root, with beef-tea or gruel, may be taken.

There are cases of diarrhœa with a *clean tongue*, which will not yield to *laxative remedies*, nor to *opiates*, *astringents*, or *stimulants*, either singly or combined, which probably depend on a want of tone in the intestine, associated with decomposition of intestinal contents and fetid stools. In these cases *five grains of salicine* every four or six hours have often stopped a diarrhœa that appeared fast hurrying the patient to his grave. *Salicylic acid* and *salicylate of soda* have also been found useful. *Tincture of the sesquichloride of iron* is similarly useful in doses of five to ten minims. In the diarrhœa of *whitish stools,* with frequent calls and sudden desire to evacuate the rectum, when muco-gelatinous matter like a jelly is passed, no remedy is of so much service as the *extract of nux vomica,* to the extent of *a fourth to a half grain dose;* or *strychnia,* to the extent of *one-twelfth* of a grain, in a pill twice or thrice a day, with the *sulphate of iron* and *extract of calumba.* *Tincture of the pernitrate of iron* (♏ x. every half hour) may be given with great benefit (W. C. MACLEAN). It is in diarrhœa of this kind that iron is of so much service. The *ferri ammonia-phosphas* possesses some desirable properties, especially as to solubility and freedom from the inky astringent taste of preparations of iron. Its preparation is given in the note below.* In *fluid extract of coto bark* (prepared by Ferris & Co., Bristol), we have also a useful remedy in doses of five to eight minims. *Black or dark stools* (*melæna*) are not so much due to bile (*atrabilis* of Abernethy) as that such stools, resembling pitch, are principally composed of morbid or impaired secretions from the intestines (HOFFMAN, HOME, GRAVES). The discharge of the black matter is followed by a feeling of relief to the system generally. In cases of true *melæna* (where the dark colour is due to blood), great debility, and sometimes fainting, may follow the evacuations. Stimulating and tonic remedies, such as *turpentine,* are of benefit (GRAVES). When diarrhœa is accompanied by a *white furred tongue,* with pain and soreness, it is necessary to give opiates, combined with some mild purgative. *Half a drachm to a drachm of Epsom salts,* with a *drachm of the syrup of poppies;* or *fifteen minims of the tincture of hyoscyamus;* or, in severe cases, with *three* to *five minims of tincture of opium,* every *four* or *six* hours, are remedies on which, as a general principle, we may very confidently rely. In other cases, *rhubarb, castor-oil,* or any other *mild purgative,* may be substituted for the Epsom salts. In cases of diarrhœa, accompanied by vomiting, *a drachm of syrup of poppies alone,* repeated every half hour, or every hour, for two or three times, may quiet the stomach, and enable it to bear other remedies. Soda-water, or the effervescing draught, with a table-spoonful of *brandy,* with or without a few minims of *tincture of opium,* often remain on the stomach when everything else is rejected.

Most practitioners lay great stress on the colour of the stools, and the necessity of correcting the supposed morbid states of the liver; but the various colours of the stools are in many instances caused rather by morbid secretions from the surface of the mucous membrane of the intestines than by any defective state of the bile in the gall-bladder. In simple diarrhœa, mercury (which is so often given in a routine way) in any form is either unnecessary or injurious in the majority of cases, except as a purgative. It is, however, sometimes necessary, especially in children under four years of age. One general rule may be acted on—namely, that in the adult, whatever be the form of the diarrhœa, if the stools be dark at first, and then become light-coloured, purgative medicines are no longer beneficial. In no instance ought they to be continued longer than is sufficient to remove any irritative substance accumulated in the alimentary canal. *Sulphuric acid,* in doses of the officinal or aromatic diluted drug, of twenty to thirty drops, with water simply, or combined with the *compound tincture of gentian,* has been found a useful remedy. It may be alternated with the *diluted nitro-muriatic acid,* and prescribed in a similar manner. The dietetic treatment should be limited to slops, puddings, and white fish boiled, and the drink to weak *brandy and water,* which acts locally as an astringent, and generally as a diffusible stimulus.

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* *Ferri Ammonia-Phosphas.*—Heat common phosphate of soda to redness. Take of the pyrophosphate of soda so obtained ℥ij. Dissolve in one pint of warm water. Then take of protosulphate of iron ℥iv. Dissolve in twelve ounces of water. Mix the solutions, collect, wash, and dry the precipitate at a gentle heat over a water-bath. Take of this precipitate ℥j., Liq. ammonia P. L., ℥iss., water q. s. Dilute the Liq. ammonia with an equal volume of water, and rub up with the phosphate of iron in a mortar until the latter is dissolved. Then dilute to ℥viij. Filter the solution, and evaporate at a heat not exceeding 120° Fahr., over a water-bath, and proceed as for the other scale preparations of iron. The dose is one fluid drachm (W. H. MOSS, Dispenser, A. H. C.)

LARDACEOUS DISEASE OF THE INTESTINES.*

LATIN EQ., *Morbus Lardaceus;* FRENCH EQ., *Lardacée;* GERMAN EQ., *Speckige oder Wachsartige Degeneration;* ITALIAN EQ., *Lardacea.*

Definition.—*A disease in which the blood-vessels of the villi, and of the mucous glands, especially of the alimentary canal, become altered and added to by an albuminoid material, ultimately infiltrating the tissues and the glands, tending to hæmorrhages, diarrhœa, and ulcerations.*

Pathology.—*Morbid Anatomy.*—Lardaceous disease of the intestines comes next in frequency to that of the spleen, liver, and kidney. In them it especially affects the arterial capillaries of the villi and the surrounding networks of the mucous and submucous tissue (see p. 129, Vol. I.) Its progressive involvement of parts is as follows:—It is seen—(1.) In the points of villi; (2.) involving entire villi; (3.) in the mucous and submucous capillaries of inflamed parts; (4.) as annular infiltration round solitary glands; (5.) as degeneration of the vessels surrounding the glandulæ of Peyer's patches.

Virchow has seen the whole tract of arterial capillaries from the mouth to the anus in a uniform condition of lardaceous disease. I have repeatedly met with this condition in dead soldiers, at the invaliding hospital at Netley. Anæmia of the mucous membrane, with a peculiar glistening or shining aspect of its surface, are the most characteristic features. Otherwise there are no outward signs of the lesion to attract attention. Pallor, anæmia, and atrophy ought to excite suspicion during life; and the application of the iodine reagent, after death, to the morbid points is absolutely necessary for its recognition. The atrophy has advanced so far that Virchow has known the villi to drop off, and the intestine to be bare of villi. The walls of the fine arterial twigs become pellucid, transparent, glistening, rigid, and thick, and a reduction in the size of their calibre is the result. Blood ceases to pass through them, nutrition is impaired, and atrophy occurs, extending over large tracts of bowel,—most decided towards the duodenum. The substance of the villi has frequently been found changed into the lardaceous or albuminoid material. Sometimes the mucous membrane is destroyed, and ulcers are developed which penetrate deeply into the tissue. Peyer's patches have been seen enlarged, as well as the solitary glands (FRERICHS). Lambl has traced the degeneration and destruction of the intestinal epithelium through the substance of the villi, the follicles of Lieberkuhn, and the muscular coat itself. Hayem records lardaceous disease of the intestines in scrofulous children supervening upon chronic suppuration of bone. Similar lardaceous disease existed in other organs, with evidence of having commenced in these organs prior to the intestinal lardaceous lesion; thus showing that the intestines became involved at an advanced stage of the general disease (*Comptes Rendus de la Soc. de Biol. Gar. Med.*, 1866; and *Syden. Society Year-Book* for 1865-66). The lesion was most intense in the lower part of the small gut; and was found also in the large intestine, and in the stomach. Two stages may be recognised—namely, (1.) Lardaceous lesions of the blood-vessels of the mucous membrane of the intestines, and enlargement of the submucous solitary glands; (2.) breaking up of the lardaceous material and of the gland tissue, with destruction of the surrounding mucous membrane. The phenomena during life associated with these stages are of the following kinds: During the first stage the evacuations are fluid, serous, and green; and the mucous membrane is covered by a thick layer of mucus (catarrh), easily removed by washing. The small arteries and capillaries are thickened, especially in the vicinity of Peyer's patches and solitary glands, which are tumid, and form projections varying in size from a millet-seed to a hemp-seed. They seem surrounded by a cup; and the projecting glands have a white semi-transparent aspect, are of firm consistence, and do not collapse if punctured. They give the usual reaction with the iodine solution as the thickened vessels and glands; and the minute vascular ramifications become visible on the pale surface of the gut, indicating an appearance of intense hyperæmia, if the vessels could be filled with blood. The lardaceous lesion commences in the cell-fibres of the small arteries and inner surface of capillaries. The small vessels in the interior of the gland follicles are affected; and the follicles themselves may contain minute concretions of lardaceous matter. Thus the appearance seems analogous to the condition of the spleen, as regards its glomeruli in lardaceous disease of that organ. In the first stage of lardaceous disease of the intestine, the *submucous vessels* are scarcely affected, while the catarrhal changes exist; and as the disease advances there is enlargement, with similar degeneration of the mesenteric glands. *The second stage* involves a transformation of the diseased follicles, and extension of the degeneration through the whole thickness of the mucous membrane. The follicles become yellow, and are afterwards replaced by the ends of thickened vessels lying in a pulpy yellow substance. This condition is sometimes followed by ulceration. The patches of Peyer become reticulated from loss of substance of the glandules; and small ulcers may result in the patches, or in the solitary glands of the intestine. The reticulated sieve-like patches of Peyer are represented by whitish prominent lines, circumscribing depressed spaces—giving an appearance of a fine lace-work or honeycomb, which represents exactly the distribution of the blood-vessels anastomosing and ramifying round the follicles in Peyer's patches. In proportion as finer vessels are implicated, so do the meshes become smaller and smaller, and the lesion extends along larger trunks. Eventually the mucous membrane becomes thin. The iodine reaction follows the reticulation of the meshes of morbid blood-vessels over the patches. The ulcers of solitary follicles have the appearance of small depressions or losses of substance, with neatly-rounded borders, as if a round piece of membrane had been punched out, with a base smooth and granular, formed of submucous tissue. Yellow debris precedes loss of substance, due to molecular disintegration of the lardaceous lesion rather than to ulceration implying pus formation. The destructive process is a granulo-fatty disintegration, through which the follicle or gland entirely disappears—as it does in enteric fever lesions sometimes—without ulceration (see p. 590, Vol. I.) The vessels on section appear as glassy cylinders, with an extremely small opening and thick walls. The lesion eventually extends to the submucous areolar tissue, and even to the boodvessels of fat vesicles, and to smooth muscular fibres. Thus the lesion is traced—(1.) to the blood-vessels; (2.) in the parts supplied, and especially in the gland follicles and surrounding parts.

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* Not recognised by the College of Physicians.

Symptoms.—Diarrhœa which persists, and does not remit in the first stage, is followed by hæmorrhage in the second stage. The stools become gradually liquid, but do not increase in frequency (one or two only daily), which are liquid, greenish, serous, white. There is no colic nor tenderness. Dr. T. Grainger Stewart noticed, for some years previous to 1868, that hæmorrhages from the stomach and intestine occur in cases of lardaceous disease independently of any ulceration of the mucous membrane. The principal symptoms during life are diarrhœa and hæmorrhage (HAYEM); the latter chiefly associated with the ulcerative changes, as erosion of the mucous membrane, and probably proceeding from ruptured vessels surrounding the follicles. The conclusions to which Dr. Grainger Stewart has arrived at regarding hæmorrhage in lardaceous disease are as follow:—(1.) That it is not an unfrequent occurrence. (2.) That next to the spleen the intestinal tract is its most common seat. (3.) That it may occur independent of any ulcerative process. (4.) That it probably depends upon rupture of the capillaries at the affected part. (5.) That the lardaceous disease of the liver does not of itself induce hæmorrhage from the bowels. (6.) That the hæmorrhage from the intestine occurs when the liver is free of the disease. (7.) That the occurrence of hæmorrhage increases the danger of the patient. (8.) That sometimes it comes and goes for years without markedly depressing the vital powers (*Brit. and For. Med.-Chir. Rev.*, 1868, p. 201).

COLIC.

LATIN EQ., *Colum;* FRENCH EQ., *Colique;* GERMAN EQ., *Kolik;* ITALIAN EQ., *Colica.*

Definition.—*A painful affection of some portion of the abdomen, caused by violent contraction of the muscular fibres of some portion of the intestinal canal.*

Pathology.—*Causation.*—The remote causes are indigestion, exposure to cold, or other general cause, and the effect of lead poison. All periods of life, from infancy to old age, are liable to colic. It attacks either sex. It is seldom that persons die of colic; but such instances appear to have occurred; dissection has then shown some portion of the intestines intussuscepted—affording a strong presumption that the beginning of cases of intus-susception may depend on a spasmodic constriction of some part of the intestinal canal. This view is supported by Mr. Blane's experience in veterinary practice. In fatal cases of colic in horses, different portions of the alimentary canal are found strongly contracted, more often the small than the large intestines, which sometimes contain gas. The bladder appears to participate in the spasm, the urine being either frequently ejected or suppressed.

Symptoms.—Colic is usually sudden in its attack; and the patient, without any previous indisposition, is often unexpectedly seized with a severe fixed pain in some part of the abdomen, which is relieved on pressure, so that he either sits doubled up, or rolls on the ground, or lies flat on the belly. In other cases, where much air is secreted, the bowels are greatly distended, and the pain is compared to a twisting or wringing around the navel, accompanied with soreness. The walls of the abdomen participate in the internal spasm, so that the navel is often drawn in towards the back, or the heads of the *recti* muscles are prominent, resembling so many round balls. The bowels are generally constipated, and the stomach may or may not be irritable. In the latter case it often rejects both food and medicine. The pulse is little altered at the commencement of the attack; but if the paroxysm be prolonged, and the patient exhausted by pain, it may be hurried and frequent. The tongue is generally clean, although sometimes white and coated.

Gastralgia, or stomach *colic*, is a severe pain in the stomach, often so completely idiopathic that the slightest cause produces it. One person cannot eat a strawberry, another a gooseberry, another an egg, without suffering. In other cases every sort of diet produces it, so that the patient is racked with pain after every meal. Those affected are usually adults; and women are more frequently the subject of it than men. The attack is generally so sudden, that the patient is unexpectedly seized with a pain, which attains its greatest height on the instant, round or above the umbilicus. It is generally accompanied by sickness or vomiting, by flatulence, and by a confined or sometimes by a purged state of the bowels. It may last from a few minutes to a few hours, and may cease as soon as the stomach is emptied or the bowels have acted. With constipation it generally continues till the bowels are relieved, when it subsides almost as rapidly as it commenced, leaving some soreness. The pulse is natural, there is no fever, and the pain is relieved on pressure—circumstances which readily distinguish it from inflammation. The disease may subside after one attack; but genuine gastralgia may last for months.

Diagnosis.—*Colic* is distinguished from inflammation by the pain being relieved on pressure, and by the quiet state of the pulse.

Prognosis is in every case favourable.

The **Treatment** is by *opiates, chlorodyne stimulants*, and *purgative medicines.*

When the bowels are constipated, *five grains of calomel, fifteen grains of jalap*, and *one grain of opium*, should be administered immediately, and followed by—

℞. Mist. Camphoræ c. Magnesiæ Sulphat., ʒj.; Tinct. Hyoscyami, ♏ xv. to xx.; Tinct. Cardamomi, ʒj. To be repeated every five or six hours, until stools are obtained.

In mild cases a scruple of *rhubarb*, or half an ounce of *castor-oil*, or other mild purgative, combined with a grain of *opium*, may be substituted for *opium, calomel*, and *jalap*. Some have doubted the propriety of administering opiates at the onset of the disease; but it is certian that a mild purgative, combined with a mild narcotic, will effect more than a drastic purgative without such combination. *Opium* in full doses (gr. i. to ii.) is more generally useful in colic than any other remedy. *Enemata* often give immediate relief. Externally, the application of large bags filled with hot *chamomile flowers*, or of *heated sand*, or *heated salt*, or of the *stomach-warmer filled with hot water, are useful.* The *warm bath, fomentations*, or a *large linseed* or *mustard poultice* over the abdomen, are also highly useful auxiliaries. The diet should be sago and arrow-root, with a little brandy; and for some time after the patient has recovered it should be light, and perhaps limited to fish and puddings.

CONSTIPATION.

LATIN EQ., *Alvus Adstricta;* FRENCH EQ., *Constipation;* GERMAN EQ., *Verstopfung;* ITALIAN EQ., *Constipazione.*

Definition.—*A retention of fæces beyond the usual period, so that when they are passed it is with difficulty, and in a comparatively hard indurated state.*

Pathology.—*Causation.*—Constipation is essentially a disease of function. It often exists without the slightest trace of organic lesion. Its *physiological causes* appear to consist—(1.) in want of sensibility of the nerves of the mucous membrane of the alimentary canal to the stimulus of its fæcal contents, so that peristaltic motion downwards is retarded; (2.) to general absence of mucous secretion from the glands of the intestine; (3.) to absorption of the fluid part of the fæces by the colon. It is a question in what portion of the alimentary canal constipation begins. Most authorities have placed its seat exclusively in the large intestines. In *post-mortem* examinations, however, formed, lumpy, hardened fæcal matter is sometimes found in the small intestines. Hence it is manifest that the origin of constipation may be either in the small or the large intestines: often commencing with impaired digestion in the stomach or upper bowel, the lower one ceases to act with its normal energy in the expulsion of abnormal fæces. The *remote causes* of constipation are numerous. Every form of impaired digestion may be a cause. The existence of hæmorrhoids, or piles, is another frequent cause; as well as a too sedentary life, especially if too strictly applied to study. Women labouring under amenorrhœa, or other functional disease of the uterus, are often constipated; and almost every acute disease is occasionally ushered in by constipation. It is a common concomitant of most chronic affections, as dropsy, diabetes, hydrocephalus, pyrosis, rheumatism, or mania. Many articles of diet are causes of constipation; many mechanical accidents also, as stricture of the alimentary canal; and many medicinal substances, as *lead*, *opium*, or other *astringents*. Persons of all ages are liable; but it is most common after the middle period of life; but children and young people up to 16 or 17 years of age are frequent sufferers. Both sexes suffer; but women, from their more sedentary lives, the greater capacity of their colon, and their greater delicacy on these subjects, are most disposed to it. When pregnant, it is a frequent complaint, from the pressure of the enlarged uterus on the colon.

Symptoms.—Most persons in health have one evacuation daily, on getting up in the morning, at the time when the organic sensibility is heightened by repose, or when it is excited by a meal, as after breakfast. If these usual periods be prolonged the fæces become hard, knotty, or scybalous, and ultimately small pellets of fæces may accumulate and form large round balls. When constipation continues, the colon becomes distended, and individual sacculæ attain a greater capacity. The bowel gradually loses its tone, so that its power of contracting upon and expelling the fæces is greatly impaired; and when the bowels are evacuated patients do not experience that sense of complete relief which is desirable after a stool. There is a desire to sit and to void more fæces, which are lumpy and difficult to pass. This retention of fæcal matter may thus cause great distension of the abdomen from its bulk and from the generation of gases, as well as pain, irritation, and a flow of blood from the rectum on passing a stool. In some instances the fæcal matter, whether retained in the *caput coli* or other part of the intestinal canal, causes so much irritation that constipation and diarrhœa co-exist at the same time; the solid matters, being retained, set up an irritative diarrhœa, while the more fluid portions give rise to repeated stools. These diarrhœal evacuations usually contain hard pieces (pellets) of feculent matter. The stools are small in quantity, and do not come away with the rush of diarrhœa. The desire to void them is often urgent, and most apt to occur early in the morning. There may be two or three such attempts before breakfast; and perhaps no more during the remainder of the day. The stools are of a very offensive odour. The looseness is that of a limited portion of the intestine only, and is due to the irritation produced by the presence of hard masses of scybala in the colon, causing catarrh in their immediate vicinity. The mucus thus abundantly excreted acts as a partial solvent of the scybalous matter, a partial disintegration of the mass takes place, and hence the stools always contain innumerable small pieces of hard fæces, from the size of a pea to that of a Barcelona nut. Such are *local symptoms*. The *general symptoms* are not less distressing. The fluid part of the fæces absorbed by the colon from day to day finds its way into the blood, and produces a form of blood-poisoning. The red corpuscles become diseased. They are altered in colour and diminished in numbers. A sallowness of complexion is the result; dark rings appear below the eyes, and the limbs, hands, and feet are cold. A feeling of lethargy is the result. The system is not nourished, and there is a falling off in flesh. Nervous depression co-exists. The pulse is slow and easily compressed. The extremities may feel numb. The organs of digestion and assimilation are lowered in tone. There is loss of memory; a want of power of concentrating the thoughts; a heavy drowsiness supervenes, which never seems to be removed by really sound sleep, and all the functions of the body are carried on in an unsatisfactory way. The patient is tired on very little exertion of any kind. There are also numerous local symptoms produced by the presence of scybala in the colon, which very often are attributed to other causes. Among these may be noted most acute pain on pressure over the situation of the hardened matter, so severe as to simulate peritonitis. If it occurs in women we may observe, as a consequence, ovarian neuralgia, excited by the irritation conveyed to the ovary by the presence of scybala in its immediate neighbourhood, while a predisposing cause exists in the lowered nervous tone consequent upon blood-poisoning. Frequently there is acute pain over the ileo-cæcal valve, and in many, if not the very great majority of instances, *typhlitis, perityphlitis,* or *pelvic cellulitis,* are direct consequences of the irritation produced by scybala lodging in the cul-de-sac. Irritability of the bladder is another local symptom frequently resulting from a similar condition of things in the immediate neighbourhood of that viscus. Displacements of the uterus may be and are caused by large accumulations in the rectum, and in every case are very much aggravated by such a state of matters. The leg too may be unable to be extended. Cases have been also known to occur where an accumulation of this kind has been mistaken for pregnancy, and at other times where it has been looked upon as an ovarian tumor. A constant effect of this condition is the prevention of refreshing sleep at night. The patient always complains of dreaming, and nightmare is a frequent symptom of this torpid state of the bowels. There is no refreshment from sleep, and the remark is generally made that he or she is more tired in the morning than they were on going to bed. In many cases the temperature of the body, rising at evening, and falling again towards morning, may raise the suspicion of typhoid fever. If children are the sufferers, a characteristic train of ailments may result, of which the following may be noted:—(1.) *In a child nine months old,* acute pain on going to bed, subsiding towards morning—wakening from sleep with a start, and shrieking as if in pain—frequent evacuations from the bowels, containing a good deal of mucus mixed with small pieces of feculent matter—falling off in flesh—a blanched, sallow physiognomy—abdominal distension and vomiting after food. (2.) *In a boy aged three,* with prolapsus ani induced by the constipation. The child shrank from going to stool because of the severe pain it produced. Health began to fail, with loss of flesh, feverish towards night, restless and starting up during sleep, and throughout the day listless and easily fatigued. Appetite was fitful. (3.) *In a boy aged six* there occurred a species of delirium after starting from sleep, continuing from a few minutes to an hour or more in the night. At other times he was drowsy and irritable, with loss of appetite and failing strength. The bowels acted every day, and appeared to be in fair order; but the stools were not sufficient in quantity, and not soft enough. (4.) *In a girl of twelve years,* languidness, being easily tired, and inclined to lounge about all day, indifference to usual occupations, and feeling "so weary" were the characteristic first signs. Sleep was disturbed by dreams and nightmare. Had been in the habit of putting off going to stool when she felt the desire to go. Pain existed over the right iliac region, which was tender on pressure. (5.) *In a girl of sixteen years,* from being lively and active, she became languid, lethargic, and desponding; irritable, hysterical, and chlorotic; breathlessness on moving about, and inability to walk a short distance without great fatigue. Feeling of relief after stool is incomplete. Sleep is broken, and there is no refreshment in the morning (Dr. Robert Bell, *Lancet,* February, 1880, p. 283). Besides this general influence of constipated bowels over the healthy state of every function, there are few disorders which are not aggravated by its continuance, few that are not benefited by its removal, while many are cured altogether. It is the superinducing cause of many disorders, especially in the female, such as dysmenorrhœa and uterine displacement. Instances of constipation of two, three, four, five, to perhaps fifteen days are not rare. The quantity of feculent matter discharged in a state of health is about 8 to 10 ounces, but varies with the size and bulk of the person, the quantity and quality of the food he eats, and amount of exercise. In cases of constipation, where the fæces have been retained for a lengthened period, the quantity passed at one motion is often quite extraordinary.

Diagnosis.—The subject of intestinal obstruction must be studied in connection with constipation (see p. 893, *ante*). It ought to be looked on as a suspicious circumstance if children have more than one motion in the day; or if they incline to sit at stool and strain for a long time without relief after having had a motion.

Treatment.—There is no rule of health more important than that the bowels be kept regularly and daily open. *Enemata* are of the first importance, and ought to be much more frequently employed than they are as a habit. They may be simply a pint of warm water, 100° Fahr.; or the same quantity of warm water, with half an ounce of *common salt.* The *common soap* enema (a strong solution of soap) is likewise a valuable remedy; and when the constipation is great, half a pint to a pint of *castor-oil, neat,* may be thrown up. For constipation in children and young persons nothing is so efficient as an *enema* of salt and water, repeated night and morning, or every night, or every other night only, according to the urgency of the symptoms. Large accumulations of scybala (stercoraceous tumors) seem visibly to melt away under their continuous influence. These *enemata* ought afterwards to be followed by one to two drachms of *sulphate of magnesia,* one grain of *quinia sulphatis,* in aromatic *sulphuric acid* in an ounce of *infusion of gentian,* every morning, till the bowels assume a healthy tone. A soap suppository—*i.e.,* a piece of common yellow soap, shaped to an oval form, about half an inch thick at the middle part and an inch and a half long, introduced into the rectum, is also a useful stimulant to the action of a torpid rectum in children. When constipation is *occasional* and *accidental,* any of the milder cathartics, in the absence of an enema, as the *sulphates of soda* or of *magnesia, castor-oil, rhubarb, aloes,* or the *confectio sennæ,* or the *pilulæ colocynthidis comp.,* will in general open the bowels. In obstinate constipation, *tartrate of antimony,* to the extent of one-fourth of a grain, combined with a drachm of *sulphate of magnesia* every hour, may often bring fæces away after nausea supervenes. If, however, the constipation is habitual, the *healthy habit* of a daily evacuation is not easy to re-establish. The remedies, though often successful, yet occasionally fail. In cases of *habitual constipation*, the combination of a tonic with a laxative will often produce a more efficient action than a perseverance in the use of purgative medicines alone. Thus, two grains of the *ferri sulphatis*, or *an ounce and a half of infusion of gentian*, combined with one or two drachms of the *sulphate of magnesia*, given according to the urgency of the case, three times a day, or every six hours, will empty the bowel when the salt alone will fail. In old persons, a combination of aromatics with the purgative, as in the *decoctum aloës*, is a more useful and effective remedy than the same or even a greater quantity of aloes alone. *Aloes* is a useful remedy when the colon seems specially at fault as a result of fever and debilitating diseases, or of sedentary habits and occupations. The watery extract, combined with *essential oil of ginger* and *jalapin;* or with *quinia*, *extract of nux vomica*, *ipecacuanha*, are the most useful combinations. Where *slow digestion*, with a *deficiency of mucous secretion* prevails, the following is a formula for pills, of which one ought to be taken an hour before dinner (and before breakfast also) if required :—

℞ *Pulv. Ipecacuanha*, gr. viii. to xii. ; *Ext. Aloe*, gr. xii. ; *Sulphatis Ferri*, gr. xviii. to xxiv. *Misce, et divide in pil.* xii.

Or, the *ipecacuanha* may be omitted ; or the *aloes* may be combined only with some bitter extract, such as *gentian*. Such pills ought to be continued regularly till the occurrence of a "loose motion," after which one only, an hour before the principal meal, will be sufficient.

Another formula for a pill of a similar nature is of use in habitual constipation, namely :—

℞ *Pil. Rhei. Co.*, *vel Pil. Coloc. Co.*, *vel Aloe*, gr. ii. to iii. ; *Pulv. Capsici*, gr. i. To be taken *with* the *food*, and repeated daily as occasion may require (PAVY).

Drastic purgatives ought never to be given. The diet ought to be especially attended to. More food, both solid and liquid, may require to be taken. When constipation is obstinate, and has resisted the stronger purgative remedies, a full dose of *opium* sometimes seems to relax the spasms of the muscular coat of the bowels, on which the constipation may depend. *Belladonna* also may be given with advantage, in doses of $\frac{1}{6}$ to $\frac{2}{3}$ of a grain every morning at rising, combined with *extract of gentian* (TROUSSEAU) ; or 10 minims of the tincture of belladonna may be similarly taken. All medicines for the relief of constipation ought to be given just before or with the food ; and are to be regarded as acting *with the food* in bringing about the result. Pills or draughts at bedtime are not so useful. In the constipation of children, *sulphate of potass* is recommended alike by Drs. Hillier and West. The following are useful formulæ :—

℞ *Potas. Sulphatis*, gr. xl. ; *Syrup Rhei.*, ℥ss. ; *Aq. Carni Ad.*, ℥iii. ; *Misce.* Dose—A table-spoonful for a child six years of age (HILLER).

℞ *Potas. Sulphatis*, gr. xii. ; *Infus. Rhei*, ʒvss. ; *Tinct. Aurant.*, ʒss. ;

Aq. Carni, ℥ii.; *Misce.* Dose—A table-spoonful for a child three years of age (West).

A severe case of *colic* with *constipation*, in the practice of Dr. Murchison, recovered under the use of one-half grain of the *extract of belladonna* every four hours, with *belladonna ointment to the abdomen*, *a warm bath*, and *castor-oil enemeta* (*Lancet*, Jan. 19, 1871). When constipation arises from torpor of the colon, equal parts of *compound gamboge pill* and of *compound colocynth pill* is recommended by the late Dr. Symonds of Bristol (*Lib. of Medicine*, Vol. IV., p. 139). The *compound extract of colocynth*, or *compound colocynth pill*, variously combined with small doses of *blue pill*, or with *ipecacuanha*, or with *podophyllin*, or with *nux vomica*, is a safe and useful form for the relief of the bowels. *Colchicum* (*tincture of the root*) has been found of service in doses of *ten* drops several times daily (Chapman). When constipation does not yield to the simple treatment which has been mentioned, recourse must be had to more active remedies. Thus *calomel*, gr. v.; *c. jalapæ*, gr. xv., is a dose which rarely fails to produce motions, and this, if necessary, may be followed up four hours after, either by the neutral salts in divided doses, or by a black draught in one dose. If a stronger medicine than the above be necessary, *elaterium* is of greater power, and from $\frac{1}{16}$ to $\frac{1}{2}$ grain, combined with *henbane*, is sufficient to begin with, so as to avoid hypercatharsis. When the stools indicate a deficiency of bile, *inspissated ox-gall* has been recommended. It may be given in the following formula:—

℞. *Fel. Bov. Purificat.*, ℥ii.; *Ol. Carni*, ♏x.; *Magnes. Carb.*, q.s.; *Misce. Divide in pil.* xxxvi. Dose—Two to be taken daily (Waring); or,—

℞. *Fel. Bov. Purificat.*, *Pil. Asafœtid. Co.*, ā ā. gr. xxx.; *Ex. Aloes*, gr. xx.; *Sapon. Dur.*, gr. x.; *Pulv. Ipecac.*, gr. viii.; *Misce. Divide in pil.* xxx. Dose—One or two may be taken daily before dinner (Copland).

The *resin of podophyllin*, to the extent of $\frac{1}{6}$ to $\frac{1}{4}$ of a grain, combined with *compound rhubarb pill*, acts similarly to the combination of *rhubarb pill* with *calomel*. A lengthened period sometimes elapses before it operates. The *tincture of benzoin*, to the extent of gtt. xx. three times a day, will, it is said, keep the bowels active and regular (Hastings, Streetan).

If medicines by the mouth have been insufficient, it may be desirable to hasten their action by enemata.

Sometimes the fæcal matter accumulated in the colon is so large in quantity, and so hard and impacted, that manual assistance is necessary to relieve the patient. Women sometimes suffer for years from constipated bowels; and a contrary state of bowels may take place from the irritation of hardened fæces, when they are much harassed by purging, existing more or less for many months. At length violent tenesmus comes on, with a bearing down most intolerable, much worse than a woman suffers in her confinements. On examining the rectum, a mass of hard matter may be found, which cannot be broken to pieces without the aid of an instrument. This mass consists of a variety of undigested substances, which, when broken down, may be washed away by injections, to the perfect relief of the patient.

Dietetic Treatment.—Patients suffering from constipation should avoid *port wine* or *brandy*, and should eat freely of sub-acid fruits, drink more water, and in some cases eat more food of a kind likely to stimulate the colon, such as bran bread; and *oatmeal*, in the form of cakes, or well-boiled porridge, should be eaten for breakfast. They should go daily at the same hour to stool; for such is the periodical regularity of all the functions of the body, that they are more readily performed at accustomed hours than irregularly.

SECTION XIII.—DISEASES OF THE LIVER.

SIMPLE ENLARGEMENT—*Syn.*, CONGESTION OF THE LIVER.

LATIN EQ., *Amplificatio Simplex*—Idem valet, *Congestio Jecinoris;* FRENCH EQ., *Congestion;* GERMAN EQ., *Einfache Vergröserung*—Syn., *Congestion der Leber;* ITALIAN EQ., *Aumento di Volume Semplice*—Syn., *Congestione del Fegato.*

Definition.—*Simple enlargement of the liver, from fulness or distension of its blood-vessels and bile-ducts.*

Pathology.—(*a.*) *Causation.*—The most frequent cause of hepatic congestion is valvular disease of the heart (soonest in affections of the right side, later in those of the mitral, and latest in those of the aorta); and especially obstruction to the circulation through the right side, interfering with the emptying of the right auricle. These congestions are of a passive kind; and their most marked effect is to produce *ascites*, with or without *anasarca*. If such congestions continue long, they lead to bilious contamination of the blood; and to brown or brownish-green discolorations of the skin, characteristic of torpid cachectic states, arising from these waste materials which should be taken from the blood for the formation of biliary and urinary pigments (Dr. DOBELL). *Congestions of the liver* are brought about by sudden chills, the cold stages of fevers, over-abundant feeding with little exercise, intemperance in alcoholic and malted liquors. Products of faulty digestion are thus at once carried to the liver, and influence its circulation; and so does excessive bodily exercise in the heat of the sun; and especially in Europeans by exposure to unusually high ranges of temperature in tropical climates. In summer and autumn it is not uncommon in our own country; but to Europeans on first landing in India it most commonly occurs, and occasions what is termed a "bilious diarrhœa." Great increase of temperature, combined with change of climate and an unsuitable mode of life, is known to have a direct influence on the functions of the liver, expressed, in the first instance, by an unmistakable ncrease in its secretion. Increased secretion and elimination of bile ead to enlargement. As a complication of other diseases, hepatic disorders and enlargements are of frequent occurrence; and no true estimate has yet been made as to the frequency of hepatic disease in his or in other countries. Like other exciting agents, the prolonged xposure to increased temperature (under conditions such as are experienced by the European in India) ceases to have a stimulant effect, and a depression in the powers of the organ results, corresponding to the previous excitement. In India the duration of exalted hepatic function is not found to be of long duration, but is confined to the earlier years of residence, declining from that time. This increased secretion of bile and its elimination, so long as it lasts, is regarded as a salutary effort of nature to maintain health. It is the decline of the powers of secretion, the arrest of the function, and the more or less sudden suppression of the secretion, which is attended with danger. It always precedes the expression of inflammatory states, and is associated with congestions of an active kind, in which the arterial system becomes prominently excited. This is consistent with what occurs in every other secreting organ. Previous to the expression of the inflammatory process becoming developed in a gland, the secretion at first flows freely under excitement (not necessarily morbid); but so soon as inflammation becomes expressed, the secretion is at the same time dried up. This is well seen in the phenomena of a common cold, when the exciting cause at first merely stimulates the nasal passages, marked by an increased flow of mucus from the nose; but so soon as the inflammatory state becomes fully expressed, the secretion is dried up, and when re-established is altered in its properties. So it is with the liver when the increased flow of bile is suspended, from exposure to cold or some other exciting cause, a warning is given that inflammation is about to be expressed by congestive and exudative processes in the texture of the organ. Albuminoid disease, fatty infiltration, hydatids, cancerous and other growths, hepatitis, especially ending in abscess, cirrhosis in its early stage, syphilis, tuberculosis, scrofula, and lymphatic growths, are all causes of liver enlargements. A "peculiar" enlargement of the liver is also described in connection with chronic jaundice, and with xanthalamia of the skin and other tissues (ADDISON, GULL, *Guy's Hospital Reports*, 2nd Series, Vol. VII., p. 265).

(*b.*) *Morbid Anatomy.*—Congestion is expressed in various forms:—(1.) There may be *congestion from increased secretion of bile*, and the bile-ducts are then gorged with bile; or there may be—(2.) *Passive congestion* of the hepatic veins or of the portal veins; and, (3.) *Active congestion*, chiefly involving the arterial capillaries. The liver is enlarged principally upwards, so as to encroach upon the capacity of the right side of the chest. It is apt to be increased in thickness more than in length. Its covering appears smooth, tense, and glistening, and its substance firm. A section lets free a great amount of blood. The ducts are generally distended with bile, from various causes, such as compression from over-distended veins, frequently brought about by intermittent and remittent fevers. Biliary congestion is also a condition associated with the early stages of *cirrhosis*, when the appearance of the liver is that known by the name of *nutmeg liver*. The distension of the veins seems to be of a passive kind, and to depend on recession of blood from the surface of the body. An ultimate result of hepatic biliary congestion leads to the filling of the hepatic cells with dark yellow matter; and when forms of passive vascular congestion are long associated, ultimate atrophy and degeneration of the hepatic cells ensue. The central part of a hepatic lobule is, in health, distinguished by a red spot, centrally situated, and measuring about half the diameter of the lobule. When this dimension increases it is characteristic of *intra*-lobular congestion—a morbid state in which the hepatic veins are especially engaged. In *inter*-lobular congestion, where the *portal* veins are distended with blood, the centres of the lobules are comparatively pale, while the lobules are surrounded by red vessels. The condition known as the *nutmeg liver* is explained by the various forms and results of congestion, as well as by other morbid changes. It is so named from the resemblance (in combination and arrangement of colours, in a section of the liver) to that seen in the section of a *nutmeg*. In its most marked form a deep red congestion forms patches and streaks, which occupy the central parts of the lobules, and are *partially* surrounded by patches of a greyish or dirty white colour, which are devoid of blood. The light-coloured parts may be caused either by fatty degeneration of the cells, which, enlarging, compress the capillaries and expel or prevent their being filled with blood; or it may be due to an increase from deposit and condensation of the Glissonian capsule in the early stage of that morbid condition known as *cirrhosis*. It is a morbid state in which the capillaries are emptied by compression and contraction of tissue, or in which they have undergone atrophy.

Symptoms.—With excessive secretion of bile, the patient has purging of bilious stools, which scald the anus. There is slight sickness just before the bowels act. A bitter taste is felt in the mouth, and the tongue is foul. In all forms of enlargement the liver bulges on account of its swollen condition, which may be accompanied by uneasiness on exploration, or of weight on getting into the erect posture, or after meals and lying on the left side. The countenance and complexion may be pale, sallow, or dusky-livid. The tongue will be found coated, the bowels constipated, the appetite defective, and there may be nausea, vomiting, and headache. The pulse is slow, compressible, and irregular, or it may be quick and feeble; and, generally speaking, the symptoms are obscure.

Treatment.—A restricted and judicious diet is of first importance, with abstinence from rich dishes and fermented liquors. With regard to medicine, Plummer's pill at bedtime, followed by *Rochelle salts* in a *seidlitz powder*, or *Pullna water*, in the morning, or by the usual black draught, or *compound mixture of senna*, or by such saline purgatives as *sulphate of soda* and *bitartrate of potash* (which cause a drain from the portal system of veins), usually give relief. A purgative dose of *calomel*, especially combined with *compound jalap powder*, is of all combinations the most efficient in diminishing congestion of the liver. After its action the local weight, the pain, the fulness, and other symptoms subside concurrently, with copious bilious stools. In such cases it seems to act by irritating the upper part of the small intestine, propelling onwards the bile as fast as it flows into the duodenum, thus preventing its reabsorption (MURCHISON). The bilious accumulations thus evacuated constitute the "*bilious stools*;" but they are not to be regarded as the result of increased biliary secretion from the liver itself. An active aperient ought to be prescribed every second or third day for ten days or a fortnight after the action of the *calomel* and *jalap* purgation (GRAVES). In *chronic congestion* the external use of *iodine ointment* sufficiently diluted is of service. Dr. Graves bears testimony to the value of setons over the hepatic region. Two leeches every second day to the verge of the anus, and repeated from ten to fifteen times, yield excellent results. *Nitro-muriatic acid* is a valuable remedial agent. *Podophyllin*, in small repeated doses, best relieves the engorgement and torpor of the liver in those who have resided long in tropical climates (GAIRDNER). It may not act for ten, twelve, or even twenty hours, when the purging which results is due to the large amount of bile passed into the bowels. It stimulates the liver to increased secretion. The best mode of administration is to dissolve *podophyllin* in *spirits of wine*, in the proportion of one grain to the ounce, and combine it with *essence of ginger*, in the proportion of ℥iss. to the ounce. A teaspoonful of this mixture in a wine-glass of water every night, or every second or third night, secures all the advantages of *podophyllin* without the disadvantages which result when given in pills (DOBELL, *Brit. Med. Journ.*, May 24, 1879). *Chloride of ammonium* is especially indicated when congestion is the prominent condition. It should be given in doses of grs. v. to xx. twice or thrice a day after food, combined either with acids or alkalies, according to the special indications of the case. The skin must be first induced to become moist and perspirable by some diaphoretic. If it is hot and dry, with a temperature of 103° or 104° Fahr., a sixteenth of a grain of *tartar emetic* should be given every three or four hours till diaphoresis occurs (Dr. WM. STEWART, *Philadelphia Med. Times*, April, 1878, also under *Hepatitis*). When coloured deposits are present in the skin, *chlorate of potass* should be combined with the *chloride of ammonium*. *Benzoate of soda* is another hepatic stimulant of value (Dr. DOBELL). *Taraxicum* diminishes abdominal plethora:—

Ext. Tarax., gr. xxxvi.; *Ext. Aloes*, gr. xii.; *Ext. Acet. Colchici; Pulv. Ipecac. rad.* āā., gr. vi.; *misce. Divide in pil.* xii. Two pills every night.

An electuary, composed of equal parts of *sublimed sulphur* and the powdered gum-resin of *guaiacum*, will be found useful in cases where the congestion of the liver is associated with hæmorrhoids. When dropsy prevails, doses of the diuretic pill of *hydrargyrum*, *squills*, and *digitalis* (a grain and a half of each ingredient), taken night and morning, are attended with benefit. In bilious congestion copious draughts of hot water act as an emetic, diluting the bile, and if some saline purgative be added, the congestion of the liver will be relieved. If pain prevails over the liver, leeches and fomentations are indicated, with perfect rest in the recumbent position—the diet consisting of arrow-root, sago, milk and water, barley-water, and afterwards beef-tea when convalescent.

HEPATITIS.

LATIN EQ., *Hepatitis;* FRENCH EQ., *Hépatite;* GERMAN EQ., *Leberentzündung*—Syn., *Hepatitis;* ITALIAN EQ., *Epatitide.*

Definition.—*Inflammation of the liver.*

Pathology.—(*a.*) *Causation.*—Injuries over the hepatic region. Over-indulgence in highly nitrogenous food, especially in alcoholic beverages, combined with indolent habits and deficient exercise: the influence of malaria, of residence in tropical climates, of dysentery, of syphilis, and of the causes which lead to lardaceous disease and fatty degeneration (see Vol. I., pp. 122 and 129): the action of extreme heat and extreme cold, or sudden chills: the lesions induced by parasites, such as the immature forms of the echinococcus (*hydatid* or the *cysticercus*), or round worms (*ascarides*) ascending the bile-ducts from the intestines: the influence of suppurative phlebitis, especially amongst the hæmorrhoidal veins and those in communication with the portal system.

(*b.*) *Morbid Anatomy.*—Inflammation of the liver, or hepatitis, occurs in various forms, and results in several consequences:—(1.) In simple or granular induration, which has a clinical history of its own. (2.) In a greater or less extent of softening and acute atrophy of the gland, associated with jaundice and febrile phenomena of a malignant typhoid type (FRERICHS). (3.) A form of inflammation which has a tendency to result in suppuration, especially in tropical climates, and after certain forms of dysentery, which is described under the name of *suppurative hepatitis* (ANNESLEY, CAMBAY, HASPEL, BUDD, MOREHEAD, MACLEAN). The inflammation may have its seat in the fibrous envelope of the gland (*perihepatitis*), or in the sheath of the vessels (in Glisson's capsule); or it may attack chiefly the glandular parenchyma, the portal or hepatic veins, or the bile-ducts. Inflammation of the glandular or hepatic parenchyma may be circumscribed, leading to abscess or to granular contraction; or it may be diffusely extended over the entire organ, ending in a true *suppurative hepatitis;* and, according as the process involves all the anatomical elements of the gland, or is limited to the matrix between the lobules, it may give rise to softening and abscess, or to acute yellow atrophy, or to induration or cirrhotic degeneration.

Inflammation of the Capsule of the Liver (*Perihepatitis*) and of Glisson's capsule is rarely accompanied by serious derangements, unless the inflammation extends to the portal or hepatic veins, or causes obstruction of the larger bile-ducts—events of rare occurrence (FRERICHS). It may exist alone in some cases of syphilitic infection; but on examination after death it is most frequently found associated with *interstitial hepatitis.* Peritonitis, disease of the liver itself, or inflammation of neighbouring structures, such as pleurisy, are the usual causes of *perihepatitis.*

Inflammation of the Glandular or Hepatic Parenchyma occurs as a circumscribed process limited to isolated patches, or it is diffuse, extending over the entire organ in a more or less uniform manner. The former variety leads to suppuration and the formation of abscesses. The diffuse form may induce rapid destruction of the glandular elements, with softening and atrophy of the organ, or it may induce induration and cirrhotic degeneration.

In the primary stage of the *acute form of diffuse inflammation of the liver* there occur patches of hyperæmia, while the peripheral portions of the lobules are infiltrated with grey matter; and a juice or fluid serum, rich in albumen, flows from the cut surface of the gland. The capsule in the vicinity of the inflamed part is rough and opaque. Destruction of the glandular cells results in all cases; but whether atrophy of the gland occurs or not depends upon the stage at which the disease becomes fatal, and partly upon the condition of the liver at the commencement of the process of infiltration. It is usually accompanied by a similar condition of the kidneys and spleen, when large quantities of albumen pass off in the urine, and the spleen is in a state of acute tumefaction. How far the affection is one originally of the *hepatic cells*, or whether the process starts from the *interstitial tissue* (to which the disintegration of the liver *cells is secondary*), are points not quite decided. In the former case, the appearance of the hepatic cells being mainly affected consists in their large size and swollen condition, from turgescence with albuminous material. Subsequently the cells disintegrate, and with them the general parenchyma of the inflamed part, leaving a cavity filled with the disintegrated elements of the tissue. The most important *post-mortem* changes to be seen in such livers are softened portions, having an irregular form, of a pale yellow or reddish-brown colour. The outline of the lobules are obliterated, while they are distinct in the firmer portions. The secreting cells of these parts are everywhere destroyed, and their place supplied by numerous oil-globules, granules, and particles of colouring matter. Sometimes the softening follows the ramifications of the portal vein, and the hepatic cells in the softened parts are destroyed, being converted into a granular *debris*, oil-globules, and pigment-molecules. In the former portions the entire cells are loaded with fine granules. The gall-bladder, when treated with chloroform, fails to yield pigment. *Tyrosine* crystals are formed on the cut surface of such livers after some hours' exposure to dry air; and chemical analysis of the parenchyma yields large quantities of *leucine* and *tyrosine*. The symptoms of this form of inflammation are included in the account of *acute* or *yellow atrophy of the liver*, which will be given subsequently.

The **Symptoms** of *perihepatitis* are, superficial tenderness of the hepatic region on pressure, motion, or deep inspiration, without any change in the volume or situation of the organ. Jaundice, as a rule, is absent; and febrile phenomena may not attract attention. When the liver itself is acutely inflamed, a sensation of weight is experienced in the region, with obscure sensations of pain about the right shoulder and scapula. Throbbing may also be experienced; and distress is increased by coughing or movements. The liver may be increased in bulk. Repeated chills or rigors may indicate suppuration, followed by much sweating, prostration, and emaciation, typhoid symptoms, and death.

Treatment.—*Tartar emetic*, in one-eighth or one-fourth grain doses every two or three hours, is of service in the acute parenchymatous inflammation, if given sufficiently early—*i.e.*, within the first three days, when there is much vascular excitement, and a full bounding, unyielding pulse, with a dry hot skin and scanty urine. *Tincture of aconite* may be similarly used. General blood-letting is not to be adopted. Local depletion by leeches over the region of the liver, and also round the anus, so as more directly to unload the portal system, must be at once resorted to in acute cases, followed by fomentations and large linseed meal poultices, made as light and soft as possible, over the hepatic region. Mustard may be added to them, or laudanum may be sprinkled over their surface. *Saline purgation* and *alkalies*, with or without *colchicum*, are also to be freely administered.

Some cases may be best treated by *ipecacuanha*, as in dysentery. In the early stage of acute primary hepatitis, should there be no diarrhœa, a mild purgative should be given at the commencement, to be followed by some mild diaphoretic, such as two drachms of the *liquor ammoniæ acetatis*, with five minims of the *tincture of hyoscyamus*, every half hour. After the skin has been thus made perspirable, the administration of *chloride of ammonium*, should be at once commenced in 20 grain doses twice or thrice daily, carefully noting its effects, after the manner advocated by Surgeon-Major Dr. William Stewart (*Madras Monthly Journal of Medical Science*, 1872, and *Indian Medical Gazette; Philadelphia Med. Times*, April 13, 1878). Dr. Stewart had previously (1870) published a pamphlet at Rangoon, in which he called the attention of the profession in India to the *chloride of ammonium* which has since obtained a great and well-deserved reputation in India and other tropical countries for the treatment of hepatic congestion; and it has been found of great use in this country as well as in India (MURCHISON, *Clin. Lectures on Diseases of the Liver*, 2nd Edition, 1877, pp. 136 and 624). About fifteen minutes after the first dose a sensation of warmth is experienced in the epigastrium, which gradually pervades the abdomen and becomes diffused over the entire cutaneous surface. The patient may feel "light-headed," and at times drowsy, and the acute pain is either removed or limited to some fixed point. A free and equable perspiration takes place over the entire surface in about a quarter of an hour, and with each dose similar effects will occur. Other phenomena may occur which the patient and physician must be prepared for—namely, symptoms referable to the liver or related parts, and variously described as a "shock," or succession of "shocks," in the hepatic region, "as something giving way," a "pricking sensation," a "pulling," "clawing," "working" or "gnawing" sensation. Fomentations or bran poultices are to be applied to the seat of pain. The diet is to be limited to arrow-root, sago, and barley flour made with milk, and barley water may be taken freely. Beef-tea may be added during convalescence. If liver enlargement continue on cessation of the physiological action of the drug, with feeling of stiffness, weight, or uneasiness, it may continue to be given in smaller doses (gr. v. to x.) thrice daily. The patient must be kept in bed, and in the recumbent position. Alcoholic stimulants, fermented drinks, and solid food must be absolutely forbidden, and the diet restricted to the farinaceous food already mentioned. Hot turpentine *epithems* are beneficial. *Iodide of potassium*, in combination with *taraxacum*, is of service in the chronic forms of hepatitis.

ABSCESS OF THE LIVER.

LATIN EQ., *Abscessus;* FRENCH EQ., *Abcès;* GERMAN EQ., *Abscess;*
ITALIAN EQ., *Ascesso.*

Definition.—*Suppurative inflammation, ending in a circumscribed collection of pus, or in several separate abscesses.*

Pathology.—(*a.*) *Causation.*—Abscess of the liver is rare in temperate climates. It has been caused by—(1.) Contusions or wounds; (2.)

Metastatic or *pyœmic* inflammation, as of the portal vein, the irritation of veins from disease about the rectum—*e.g.*, fistula in ano, and after operations for hæmorrhoids. (3.) Inflammation and ulcerative processes in the gastro-intestinal canal, as in dysentery. Combined statistics show the occurrence of liver abscess once in every five and a half deaths from dysentery (J. J. WOODWARD; see p. 854, *ante*). (4.) Inflammation and ulceration of the stomach, gall-bladder, or gall-ducts, or direct irritation in some part of the liver, as from suppurating hydatid cysts.

(*b.*) *Morbid Anatomy.—Suppurative inflammation of the liver* is limited to one or to several isolated portions; and, with the exception of congestive turgidness of the contiguous texture, the remaining portions of gland-tissue are rarely implicated. On making sections of a liver in this condition, in which suppurating *foci* are in various stages of pus formation, it is usual to find discoloured, yellowish, or variously colour-marked spots, all more or less soft, and set in a hyperæmic portion, from which it is fair to infer that such portions present the early or commencing stage of suppurative hepatitis. A microscopic examination of such spots shows the hepatic cells cloudy or granular, and lying in a granular material. A finely granular detritus at last takes the place of the cells, which are thus gradually destroyed. Sometimes the abscess-cavity is inclosed by a cyst, or surrounded by disintegrated discoloured parenchyma; or there is no defined boundary, but the inflammatory process extends till several contiguous *foci*, or inflamed and suppurating portions, unite into one large lesion, or till the pus finds an outlet. The pus is rarely passed into the abdominal cavity. Adhesive inflammation of the capsule covering the abscess almost invariably occurs, so that attachments form to the abdominal walls and adjoining organs. If the abscess bursts into the abdominal cavity, the result is fatal peritonitis. The abscess may perforate the thoracic or abdominal wall superjacent to the liver, and open directly outwards. The locality for such spontaneous opening is usually the space below the ensiform cartilage. The pus may also discharge into the pelvic, inguinal, or sacral regions, close to the spine. Sometimes the abscess tends, in an upward direction, to penetrate the diaphragm, when it generally empties itself into the right pleural cavity; but more often forces its way into the substance of an adherent right lung, by a distinct suppurative process, and in favourable cases passes by a free opening into a bronchus, whence it is discharged. The stomach, the duodenum, and the colon are the principal abdominal organs into which abscesses of the liver otherwise discharge themselves. The hepatic abscesses are sometimes superficial, but more frequently deep-seated, and may be developed in any part of the gland; although they are most frequently found in the posterior portion of the right lobe. In size and in number they vary greatly. They tend to point or empty themselves in the following directions, stated in the order of their relative frequency:—(1.) Through the lungs (from 10 to 30 per cent.); (2.) through into the peritoneum, the stomach, or some part of the intestinal tract (nearly the same proportion); (3.) through the external integuments (in the minority of cases).

Symptoms.—Fever, rigors, headache, and delirium are not uncommon as early phenomena of suppurative inflammation; but there may be no symptoms pointing to disease of the liver, and the difficulties which embarrass the diagnosis of suppurative hepatitis cannot be overrated. In 13 per cent. the disease runs a perfectly latent course, and in only 8 per cent. are symptoms at all well marked (LOUIS). In most cases a correct diagnosis will only be arrived at by not relying upon individual symptoms; but by taking a comprehensive view of the mode of origin and entire clinical history of the case, and after excluding the diseases of the liver and the neighbouring parts which may give rise to symptoms similar to those of hepatitis. Practically the symptoms denote either—(1.) A superficial and adhesive inflammation of the organ; or, (2.) a deep-seated suppurative inflammation of the substance of the liver. The most prominent symptoms of hepatitis are, however, some tumefaction, pain, or uneasiness of the liver, or of the adjoining parts, as the thorax, abdomen, or right shoulder; an affection of the bowels, as diarrhœa or dysentery; and lastly, pyrexia in a continued, remittent, or intermittent form—the presence of the malaria cachexia. Tenderness on firm pressure is more rarely absent than pain. When pain is present it may be acute, or dull and heavy. Acute pain characterises small multiple abscesses; dull and heavy pain, large single ones (MURCHISON). It is aggravated by lying on the right side, apparently from the greater weight pressing on the liver; or, the pain is felt most acutely on turning on the left side, probably from adhesions being dragged on, which have formed to the ribs. In general, the easiest position is on the back, or a little over to the left side; and towards the termination of the disease the patient may be seen lying in a position which he had previously declared himself unable to assume. As the disease advances, the pulse becomes frequent and hard, the skin hot, dry, and constricted, while pain, with a short dry cough, and dyspnœa increase. Gradual enlargement of the organ from day to day can often be made out (MARTIN). Where the abscesses are small, acute hepatitis may exist without pyrexia. Some fever, however, is commonly present, and in general it commences with shivering, vomiting, and purging—symptoms which gradually diminish in a day or two, leaving the patient comparatively free from fever, and the pulse nearly natural. These paroxysms occur at intervals, sometimes returning as regularly as those of intermittent or of remittent fever; at other times the periods are less marked, the chief symptoms being rigors occurring at irregular intervals, frequent pulse, and sweats, the latter chiefly occurring in the night, and so copious as in some instances to pour off the body of the patient. Cases of general suppurative inflammation are insidious in approach, and the destruction of the substance of the liver proceeds silently and rapidly. The existence of the disease is often not known till severe structural changes in the organ manifest themselves, and then they run a rapid and often fatal course. This is especially the case when the inflammation is induced by the combined effects of extreme heat and malaria. The miasmatic affection of the blood, from deranged mucous surfaces, and from contaminated matters brought by the splenic vein, contributes to hyperæmia of the liver, which may remain unnoticed till extensive inflammation develops itself. A burning sensation with a mottled appearance of the skin of the hands and feet; an irritable temper; a capricious appetite; langour and persistent feverishness; frequent, settled, and increasing pains in the shoulder and back, are pathognomonic signs of suppurative inflammation going on in the liver. The fever may be from time to time of a continued or intermittent type, and *the patient emaciates slowly*, becoming sad and desponding. The face, pale and cachectic, has a patched or blotchy appearance. Sometimes there is jaundice, followed by anasarca and ascites,—from compression of the ramifications of the portal vein—intestinal catarrh, bilious diarrhœa, hæmorrhage from the bowels, and dysentery. Icterus is by no means a constant symptom of abscess. It is absent in the majority of cases (LOWE). As in phthisis, the "cheerful hope" may illumine every hour the patient has to live. In other cases depression amounts to despondency, with restlessness and want of sleep. At last delirium may obliterate the past, and throw a veil over the future, till the patient dies. In the midst of these symptoms perhaps an abscess points; and the pus may have formed in the absence of rigors usually indicative of its formation. The patient becomes hectic, his pulse rapid, he is covered with a copious and clammy sweat, and his life in a great measure depends on the part where the abscess points.

Diagnosis.—Hepatic abscesses are especially difficult of diagnosis when complicated with right basic pneumonia; or where there is a large central abscess not pointing in any direction; otherwise the subject of diagnosis is fully set forth in the symptoms already given.

Treatment.—The abstraction of blood is most efficiently accomplished by leeches round the anus. They act more directly on the portal circulation than over the hepatic region. When congestion is known to exist, leeches may be applied with benefit, combined with purgative salines, such as *sulphate of soda* or *Rochelle salts*, and antimony as a diaphoretic, to be followed by *chloride of ammonium*. Hot fomentations applied to the region of the liver are of great service, and the diet should be as limited as is consistent simply with the maintenance of life, as long as acute symptoms exist. *Mercury* should not be given, except as an occasional purgative, when the local inflammation is subdued, and purgatives are only then of use when the intestinal functions are sluggish. It is still a question whether or not an artificial opening ought to be made, in the absence of fluctuation to justify the operation. "In any cases the opening of a liver abscess is a critical proceeding; but it should be resorted to whenever the symptoms give assurance that matter has formed, whenever there is a reasonable prospect of getting at the matter, and when operative proceedings are not so dangerous as to be prohibitory, for it is very desirable to anticipate spontaneous opening by any of the usual channels, as all are dangerous. Into the peritoneum, pleura, or pericardium, the result is of necessity almost always fatal; while even into the lung, stomach, or bowel, the danger is often great; and though followed by temporary amelioration, may end in exhausting disease or purulent contamination of the whole system" (Sir JOSEPH FAYRER). Professor Lister's antiseptic method of procedure appears to me the one which ought to be adopted in opening all hepatic abscesses.

"When there is reason to believe that matter has formed, although the physical signs of it may not be clear, attention should be directed carefully to the evacuations, especially if there be diarrhœa, as the abscess may open into the bowel. This is the probable explanation of recovery in cases where it has been thought that the abscess had either never really formed, or that it had undergone absorption. When opening occurs through the lung or stomach the symptoms are unmistakable. When abscess has formed, the object is to favour its evacuation, to support the strength, and to allay irritation. Much has been written and said about *exploring for liver* abscess, and in certain cases, where doubt exists, the proceeding is justifiable, using a small canula and aspirator, when there is enlargement, tenderness on pressure, which causes nausea and disturbance of the heart's action, such as palpitation or sickness, especially if there have been any chills, increase of temperature, or sweats. In the event of matter being detected at a considerable depth, it may in some cases be aspirated; but it may happen that, on account of its thickness and the presence of flakes of lymph, pus will not pass through the small canula, and no satisfactory result is obtained; but if there be any marked probability of its presence, a large canula may be used and left in after if pus is found, the most careful antiseptic precautions being observed in the subsequent dressings. Whenever the matter lies moderately near the surface, even though a certain amount of liver-tissue intervene, a free incision is best—hæmorrhage, should it occur, soon ceases, and there is much better prospect of a free drain afterwards. A large canula may in some cases be preferable. It would signify little which were selected, provided antiseptic precautions be carefully observed, and the exit be free. In the event of exploration detecting matter deep-seated, but under much liver substance, the canula is preferable to the knife, used with the aspirator, though great care should be taken not to exhaust too rapidly, or with too great force, for fear of injuring the liver tissue surrounding the cavity of the abscess and causing hæmorrhage" (Sir JOSEPH FAYRER, *Lancet*, May 8, 1880, p. 709). "Cases of doubtful liver-abscesses present symptoms varying in every degree ; at the one extreme cases of general cachexia, with irregular slight febrile attacks, exhibit symptoms as frequently attributable to deranged stomach, or bowels, or lungs only, as to the liver itself; while at the other, slight general enlargement of the organ would be found associated with that peculiar form of 'tenderness' in which pressure over the organ produced an indescribable sensation, inducing either faintness, hurried respiration, palpitation, or nausea with retching, or all of these at once. In all this large class of cases it was the custom of Professor W. S. Palmer, of Calcutta, to plunge a long trocar and canula, of small diameter, into any or all parts of the liver, through a valvular opening, examining on the spot the small quantity of extracted matter for pus globules. It was only in exceptional cases that any signs of pus could be detected. When it was so detected the puncture was generally followed by slight inflammatory action at the seat of puncture, which probably ended in adhesion of the organ to the parietes, and so facilitated the future opening of the abscess. When, on the other hand, no pus was found, a good deal of anxiety was felt in the earlier cases, lest the puncture should be followed by any evil results. Such moments of anxiety soon ceased to recur, for it very rarely happened that the patient did not express himself the next day as feeling very much relieved; and in no case did any bad consequences result from such punctures. The relief was frequently only temporary, in which case a second, a third, or a fourth puncture was made at intervals of eight or ten days. In some, however, one puncture sufficed to cure" (*Lancet*, May 8, 1880, p. 709).

To prevent the entrance of pus into the abdominal cavity, the following method has been recommended by Bégin and Recamier:—(1.) Being decided as to the limits of the abscess, the patient is (2.) laid on his back, with the upper part of the body bent forward and the thighs flexed upon the abdomen; (3.) an incision from two to three inches long is to be made over the abscess, dividing the skin, the subcutaneous adipose tissue, the muscles, and the aponeurosis; (4.) the peritoneum is then laid open, as in operating for hernia, by slitting it up on a grooved director to the same extent as the primary incision; (5.) the wound is then to be dressed with charpie, and to remain untouched for three days; (6.) at the end of three days the dressing is to be removed, when the capsule of the liver will be found to have contracted adhesions to the margins of the wound so firm that the abscess may be opened without the danger of pus passing into the abdomen. The following expedient may indicate whether the liver adheres to the abdominal parietes or not. When the liver is large, and the abdominal parietes are thin, it may be made out "by feeling the edge of the liver, or some prominent part of its surface, and marking the place of this with a pen on the surface of the belly. If the liver be adherent to the abdominal parietes, the line or spot so marked will correspond to the edge or prominence of the liver in all positions of the body. If it be not adherent, the liver will slide along the wall of the belly when the patient draws a deep breath, or changes his posture—the liver will fall, for example, towards the left side when he turns from his back over to that side, and the line or spot will no longer correspond to the edge or prominence in question (Budd, *op. cit.*, p. 122).

An interesting case of hepatic abscess is related by Sir Henry Cooper, of Hull, in the *British Medical Journal* for May 23, 1863. He justly takes exception to the method of Bégin and Recamier, because it may provoke the very danger it is desirable to avoid. In all cases in which suppuration has extended so near the surface as to give the sense of fluctuation, irritation and adhesion of contiguous surfaces have taken place. The process also interposes a serious delay at a critical period. He advocates a direct opening into the abscess, *when there is reasonable ground for believing that an external outlet for the matter is the direction taken by the abscess.* Peritoneal connections are sure then to have taken place. Tenderness of the tumor is the most satisfactory indication for the operation, and a hardened base of effused lymph a certain confirmation. Delay exposes the patient to the risk of rupture of the walls of the abscess by coughing, sneezing, or the like, and to the laceration of any adhesions which may have formed; or it allows him to perish unrelieved from constitutional irritation and consequent exhaustion. A middle course was devised and carried out by Dr. Graves, in the Meath Hospital, in a robust man. External elevation, hardness, and pain, eventually confined to one spot in the right hypochondrium (after acute inflammation of the liver), left no doubt of the formation of an abscess. Hardness was followed by deep-seated softness, yet no tendency was shown by the abscess to point outwards. The swelling remained stationary, and the integument of natural colour; but the general strength began to fail, and it became an important question whether the abscess should be opened. It was objected—(1.) That the external tumor was diffuse; (2.) that the exact site of the abscess was uncertain; (3.) that failure to evacuate its contents might prove detrimental. A decision was therefore given *against* operation by the surgeons of the Meath Hospital. Under these circumstances Dr. Graves remembered that he had seen cases where an incision made over a deep-seated abscess had failed to give vent to the matter in the first instance; yet in the course of a few days the pus found its way to the incision, and burst through it. He therefore proposed that an incision about four inches long should be made exactly over the centre of the tumor—that it should be carried through a considerable depth of muscle, and, if possible, be continued *to within about one or two lines of the peritoneum.* This incision was then to be plugged up from the bottom of the wound with lint, and thus kept open, in the hope that pus might tend towards the incision, and finally burst through it. Such an operation was done. The abdominal muscles were found of considerable thickness, and healthy; and although the incision was very deep, yet the situation of the hepatic abscess was not felt more distinctly. It now became evident that no prudent surgeon would have persevered in an attempt to open directly into it. A result was therefore waited for. In two days the patient sneezed, when purulent matter in very large quantity burst through the wound. It appeared that the incision had not been made exactly over the abscess, but rather to one side of it; for the matter did not come from the bottom, but from the side of the wound; and pressure on the liver on that side caused matter to flow out in abundance through that part of the wound which communicated laterally with the abscess. Had the original incision been continued, it might have failed to open into the abscess (*Dublin Hosp. Report,* Vol. IV., p. 40).

After the abscess has opened, strict rest must be enjoined; and sometimes the cavity is very long in closing up. It must be kept from putrefactive changes by antiseptic dressings. Convalescence is always tedious; and sometimes cicatrisation is imperfect, continuing to discharge pus at intervals for years (FRERICHS). It has been argued, as already indicated in the experience of Dr. Palmer of Calcutta, that our efforts should be directed to detecting by acupuncture the seat of an abscess, and evacuating it as soon as possible; and that this method of exploration or of "prospecting the liver," should be commenced as soon as symptoms present themselves indicating the possibility of abscess. It has been argued that because some cases have recovered after such a method of finding and evacuating an abscess, it is a practice which should be made the rule, in place of waiting for some indications that pus has even formed in the liver (MURRAY, CAMERON). The recommendations to the plan are not yet based on statistical records, and rest upon the opinions or impressions of two or three individual men. Certain it is that the plan recommended is opposed to the well-established principles upon which surgical interference rests, and is opposed to all that is known regarding the pathology of hepatic abscess, as set forth in the carefully recorded facts of Waring, Martin, Morehead, Frerichs, Budd, Maclean, and Lowe. It is also stated by Dr. Cameron (the latest exponent of this method of puncture), that in cases of hepatic enlargement, where the trocar has been used more than once in an unsuccessful search for an abscess, the operation was followed by the speedy absorption of an enlargement of the liver, which had resisted all the routine methods of bringing about its dispersion; and that this is a native mode of dispersing hepatic and even splenic enlargements in India (*Lancet*, August 8, 1863, p. 169). *Puncturing parts, or acupuncture* with a very fine needle, is a surgical operation said to be much in use amongst the native Chinese and Japanese *hakims*. But accurate information regarding the results of such operations are not on record. It should be remembered, in dealing with large livers or *probable* hepatic abscess, that an hepatic abscess always begins from multiple foci (ROKITANSKY). We must still look to physicians of large Indian experience for information on this important subject; while the antiseptic method of Lister warrants operative interference now which could not have been carried out with safety before.

The time which elapses after opening an abscess till the patient's recovery is generally from one to two months. When convalescence is established, the functions of the liver remain torpid, and its substance often indurated; subsequently, also, the spleen may be enlarged. In such a state Sir Ranald Martin recommends the use of *nitro-muriatic acid* baths. They promote the depurative functions of the liver, kidneys, bowels, and skin.

"The form and manner of preparing and using the acid bath are as follow:—Take of *hydrochloric acid* three parts; *nitric acid*, two parts; mix the two acids carefully and slowly, so as to avoid evolution of heat; and having waited for twenty minutes, add of distilled water five parts, and mix the whole carefully. *For a general bath in which to immerse the whole body:*—Pour into the bath about five pailfuls of cold water; add two quart bottles, containing sixty-four fluid ounces of the dilute *nitro-muriatic acid*, prepared as above, and then sufficient boiling water to raise the temperature to 96° or 98°. The body is to be quickly and thoroughly dried with warm towels; and afterwards the patient must retire to a well-aired and warm bed. The use of the bath is only to be discontinued when tenderness of the gums or general *malaise* occur; and cuticular irritation from the acid is to be avoided by diminishing its strength. Iron and other forms of tonic remedies may also be administered at the same time, as well as opiates," (MARTIN, *On Climate*, p. 564, *et seq.*)

CIRRHOSIS.

LATIN EQ., *Cirrhosis;* FRENCH EQ., *Cirrhose;* GERMAN EQ., *Cirrhosis;* ITALIAN EQ., *Cirrosi.*

Definition.—*Interstitial inflammation, with increase of the connective-tissue and its subsequent contraction, leading to other alterations and results.*

Pathology.—(*a.*) *Causation.*—The chief cause of cirrhotic induration is the abuse of spirituous liquors. In other words, it is due to the specific action of *alcohol* as an irritant or stimulant poison. The forms of alcoholic drink which are most efficient as an irritant seem to be *gin* in this country and *schnapps* in Germany. Gin is known to be greatly sophisticated, especially with spices, such as *capsicum*, which may also have an influence as an irritant. The disease is more frequent in men than in women, and is rarely seen in childhood (NIEMEYER). Wunderlich relates the cases of two sisters, aged eleven and twelve years respectively, and in each of them the liver was in a condition of *cirrhosis*. On careful inquiry it was discovered that both of them were great *schnapps* drinkers (see also *British Med. Journal*, Nov. 11, 1871, for the account of a case in a boy, a brick-maker, aged eighteen, and a gin-drinker). *Alcohol*, however, is not the only irritant capable of inducing cirrhotic or adhesive inflammation. Numerous products of faulty digestion, or of material such as spices taken into the stomach, may act as irritants of which we know as yet little. Other causes are *syphilis* and *malarious fevers*, especially frequent attacks of intermittent fever. The cirrhosis from syphilis is generally associated with the lardaceous disease about to be described.

(*b.*) *Morbid Anatomy.*—The chronic form of diffuse inflammation expresses itself by simple or granular induration of the substance of the liver, sometimes called *cirrhosis*, *interstitial hepatitis*, *hob-nailed* or *gin-drinker's liver*. The nature of this induration was first demonstrated through the investigations of Kiernan, Hallmann, and Carswell. They showed that an increase of the interlobular connecting tissue of the hepatic parenchyma was the essential feature of cirrhosis. Rokitansky distinguishes two different modes of origin of the granular induration—the one proceeding from a morbid development of the capillary blood-vessels, owing to an excessive secretion of bile; the other due to a chronic inflammation of the hepatic parenchyma, tending to impermeability or obliteration of the finest ramifications of the portal vein. As a rule, the disease only comes under observation when it is more or less completely developed, and when consecutive disorders also associated with it draw attention to the state of the liver. Its commencement is marked by increased consistence of the liver, due to increased and increasing hypertrophy of the areolar framework, that scanty connective-tissue (almost granular in fineness when normal) which, as the continuation of Glisson's capsule, accompanies the hepatic vessels throughout the hepatic parenchyma. The inflammation is characterised by excessive germination of this connective-tissue, without any tendency to free exudation, suppuration, or abscess. The nature of the hepatic cell-contents also affects the consistence of the organ. The gland is softer when the cells are loaded with *fat*, and firmer when they are infiltrated with *albuminoid* material, as when *cirrhosis* is associated with *lardaceous disease*. The amount of blood and of serous infiltration affects the consistence; but the induration and the tenacity of the liver in cirrhosis are mainly due to the increased development of the areolar framework, or connective-tissue, the increased and increasing amount of which displaces more and more the hepatic cells. In the *early stage* the liver may be enlarged or of normal size, rarely smaller than natural; its surface covered by an opaque and thickened capsule, exhibiting flattened projections, varying in size from a pin's head to a pea. Similar nodules are observed on section, separated by narrow strips of grey areolar tissue, more or less vascular—the first beginnings of the cicatricial contraction which eventually strangles the parts. Sometimes the colour is dark from bile-pigment; sometimes pale from fat. In the *advanced form* the liver is reduced in size, particularly the left lobe, which not unfrequently is shrivelled up into a small membranous-like appendage, with a soft flabby rim of connective-tissue at the margin. Semi-globular knobs, more or less prominent, of a uniform, or of unequal size and form, are scattered over the surface. In *lardaceous disease* combined with *cirrhosis* the liver exceeds its normal size. The *serous capsule* is always thickened, and of a greyish-white colour, especially in the depressions between the granulations. Numerous bands of connective-tissue pass from it to neighbouring organs, such as the diaphragm, colon, stomach. On section it has a cartilaginous hardness and tenacity, showing at one place narrow, at another broad lines of connective-tissue surrounding the granulations, and sending streak-like processes into their interior. The granulations are generally dark or pale yellow. The elements of tissue are changed as to,—

(*a.*) *The Condition of the Hepatic Cells,* a large portion of which are destroyed, and their remains are found in the form of small masses of brownish pigment scattered through the filaments of the newly formed connective-tissue. Another portion of the hepatic cells constitutes the substance of the granulations, in which they may remain for a long period intact; but ultimately they become filled with fat and various sorts of pigment. In nearly one-half of the cases fatty degenerations occur. The connective-tissue, compressing the commencements of the bile-ducts, causes a retention of the secretion and a jaundiced condition of the liver; hence the deposit of pigment, which accumulates in the form of fine orange or sulphur-yellow granules, more rarely diffused through the cells. Another tint may occur from decomposition of the red matter of the blood, especially where branches of the hepatic veins are obliterated.

(*b.*) *The Condition of the Connective-tissue.*—Its increased amount presents numerous varieties as regards its distribution, to which are due the differences in the size of the granulations. The earliest increase is upon the finer subdivisions of the vessels in the interior of the liver, gradually involving lobules (*acini*). The bands of tissue sometimes surround single *acini;* at another time, three, five, or even seven *acini* may be inclosed. These bands increase at the expense of the *acini,* so that a little mass of brown pigment may be all that remains of them. In this way large masses of connective-tissue take the place of gland-substance. This new connective tissue varies as to elementary characters; at the circumference of the lobules it is fibrillated; in the substance of the lobules it is disposed to be amorphous; and in the thickened capsule it may be fibro-cartilaginous.

(*c.*) *The Condition of the Vascular System of the Liver* undergoes great and important changes. The smaller divisions of the portal vein are narrowed by the shrivelling of the connective-tissue, or by partial impermeability of the finest ramifications of the portal vein from inflammation, obliteration, or compression. The vessels lose their round form, and become angular and bulging. Thrombi ought to be looked for in the branches when destruction of capillaries is obviously in proportion to the disappearance of the granular substance of the liver. So long as the hepatic cells exist, the peculiar mesh-like capillary network also exists; but where the cells disappear, their place is supplied by a connective-tissue, and entirely new capillary channels make their appearance, forming elongated meshes, communicating as well with the veins as with the hepatic artery (FRERICHS).

The *trunk of the hepatic artery* becomes enlarged, and its capillary ramifications more extensive than in the healthy state, black pigment accumulating in its branches. A branched and tortuous network of vessels, of large calibre, appears in the connective-tissue, their peculiar and tortuous mode of distribution demonstrating their new formation. The *hepatic veins* are unchanged; but their obliteration may take place by inflammation propagated to the walls of the vessels from the capsule of the liver, when the capillaries of the hepatic vein are gradually destroyed, and their communication with the portal capillaries is interrupted.

(*d.*) *The Condition of the Bile-ducts.*—Their origin at the periphery of the lobule is destroyed by the pressure of the new connective-tissue; and there is apt to be catarrhal tumefaction of the mucous membrane of the larger branches.

These various changes give rise to a long series of functional derangements, which in practice constitute the *symptoms of cirrhosis.* These are mainly—(1.) Derangements of the chylo-poietic organs, from impediment to the passage of blood through the portal vein into the hepatic veins, and its stagnation in the radicles of the portal vein; (2.) impairment, passing to complete suspension of the functions of the liver.

Symptoms.—Derangements of the stomach, a loaded tongue, nausea, and occasionally vomiting and faint jaundice, are the earliest clinical phenomena, which usually come on very insidiously. They may even abate, but the degenerative process in the liver advances, and gradually undermines the constitution. Digestion continues feeble; distension and tenderness of the epigastrium, along with heartburn, flatulence, and constipation, are developed. The patients lose flesh and strength; their colour becomes pale, or dirty yellow, while the skin is dry and rough. With distension fluctuation may be found in the abdomen, while the liver is found reduced, and the spleen increased in size; and increasing ascites or tympanites induces more or less dyspnœa. Ascites is the most common and constant symptom of cirrhosis. Hæmorrhages from the stomach or intestines are apt to occur as the disease advances. Enlargement of the spleen, to the extent of two or three times its natural size, is common in the later stages (OPPOLZER, BAMBERGER, FRERICHS). Niemeyer considers this enlargement due to the same causes as the enlargement of the liver, and not a secondary lesion to the liver affection. The stomachal and intestinal hæmorrhages often relieve this splenic enlargement. Enlargement of the superficial veins of the belly is characteristic of cirrhosis, and demonstrates that the current of blood in the portal vein is greatly impeded. Slight febrile excitement only manifests itself towards the close, and in most cases diarrhœa terminates life by exhaustion. In other cases the fatal end is by pneumonia, acute pulmonary œdema, or peritonitis. Occasionally death occurs under symptoms of acholia; the patients become jaundiced, purpuric spots or ecchymoses are scattered over the skin; while delirium, convulsions, and deep coma close the scene.

Diagnosis.—Slight sallowness of complexion, a dull pain, or some degree of tenderness in the right hypochondrium, with occasional feverishness, in a person above the age of thirty, who has been long in the habit of drinking spirits to excess, are almost conclusive evidence of the existence of cirrhosis, even before there is any direct proof that the circulation through the liver is impeded (BUDD, *op. cit.*, p. 158). The urine is saturated with much abnormal colouring matter and urates, and when the spleen is also found enlarged, the diagnosis of cirrhosis may be certain.

Prognosis is always unfavourable, and the main question regarding

Treatment is the possibility of relieving the disorders of function which threaten life, and so to delay the fatal termination. Absolute abstinence from spirits is indispensable, and the diet should consist of mild, simple articles of nourishment, especially easily digested animal food. Coffee, spices, and articles irritant to the liver must be avoided. Swelling and tenderness indicate *leeches* and *fomentations.* Mild saline laxatives may be given; and when the tenderness ceases, the bowels must be kept open by *rhubarb* and *salines.* When nausea or vomiting occurs, *hydrocyanic acid, belladonna, morphia,* or *extract of nux vomica,* are particularly suitable. The gastric and intestinal catarrh require to be subdued by *alkaline carbonates.* They lessen the viscidity of the mucous secretion. When pain prevails, cupping or leeches are indicated over the liver, or leeches to the verge of the anus. Saline purgatives, such as *sulphate of soda* or *tartrate of potash* and *soda* (*Rochelle salts*), should also be administered, while iced drinks and low diet must be the rule of life.

What has been said under the subject of simple enlargement or congestion of the liver may be referred to here as indicating a line of treatment under the circumstances there provided for. The saline laxatives are best taken as mineral waters, especially at Karlsbad or Marienbad. Springs containing small quantities of iron are also of service, such as Eger, Franzensbrunnen, Kissengen, and Homburg (NIEMEYER).

FATTY LIVER.

LATIN EQ., *Jecur Adiposum;* FRENCH EQ., *Foie Gras;* GERMAN EQ., *Fettleber* —Syn., *Fettige Entartung der Leber;* ITALIAN EQ., *Fegato Grassoso.*

Definition.—(1.) *Infiltration or deposit of superfluous fat in the liver-cells, commencing at the circumference of the lobules, from the blood of the portal vein capillaries;* (2.) *Degeneration of the liver-cells, in which the nutritive process within the hepatic cells is disturbed, so that an abnormal increase of the fat naturally contained in the hepatic cells takes place, which remains there, so that they become engorged with oil.*

Pathology.—(*a.*) *Causation.*—Fatty liver is associated with fatty infiltration and degeneration. It contains an abnormal quantity of fat. In cases of great emaciation it may obtain its fat from reabsorption of subcutaneous fat. A free indulgence of rich and fatty foods and alcoholic or malted drinks, with very little exercise, are the conditions which lead to the *infiltration* of fat. The cod-liver oil, so largely supplied in doses, which in many cases literally flow through the intestines unconsumed, contributes largely to produce the fatty livers so frequently seen in pulmonary phthisis.

(*b.*) *Morbid Anatomy.*—The fat accumulates within the cells, first as minute particles surrounding the nucleus and clinging to it. These particles increase in size, coalesce, and form large drops of fat, distending the hepatic cells into large globules. The accumulation of fat and the degenerative process begin at the circumference of the lobules, and gradually advance inwards towards the region of the hepatic vein, so that the whole lobule becomes converted into a mass of fat. A slight amount of infiltration of fat does not alter the appearance nor the size of the liver. It can only be recognised microscopically. In extreme cases, which have passed on to degeneration, the liver is enlarged and flattened out, with thick and rounded edges. It is increased in absolute weight, but lessened in specific weight, and the colour varies from yellowish-red to a distinct yellow, according to the relative amount of blood and fat. Its consistence is diminished, and it feels soft, with a large development of fat in other parts of the body—the supply of nourishment being in excess of its consumption or combustion. In both conditions the nuclei of the hepatic cells are obliterated or obscured, and the *degeneration* thus takes place in a similar mode to the normal physiological infiltration; but the former tends to remain as a permanent lesion—the other is temporary, the deposit of fat being removed in the processes of nutrition, and conveyed into the general circulation; and the liver-cells remain clear, with their nuclei visible. When the fat remains to such an extent as permanently to obscure the nucleus, the liver is then doughy, like œdema, and pits on pressure with the finger, retaining also the impressions of the ribs after death. On section it leaves the blade of the knife covered with oil, and a little blood flows from the cut surface. Extremely fatty livers may contain forty-three to forty-five per cent. of fat (FRERICHS, VAUQUELIN) in the form of *olein* and *margarine*, in variable proportions, with traces of cholesterine. The fatty *infiltration*, except in extreme cases, does not seem to impair the functions of the organ, nor interfere with the circulation of blood in it.

Symptoms.—The existence of a fatty liver is only to be inferred by a study of the surroundings and antecedents of the case. There are no positive subjective symptoms. Impaired stomachal and duodenal digestion is common. The enlargement is painless, the surface is smooth, and its resistence so diminished that its edge is with great difficulty distinguished. Such conditions in fat persons, or in cases of *pulmonary phthisis*, are sufficient to cause fatty liver to be suspected only. Sometimes it seems to be a cause of diarrhœa in phthisical patients. There may be signs of fatty changes elsewhere.

Treatment.—Dr. Murchison recommends that large quantities of common salt be eaten with the food, while, if circumstances permit, the alkaline or saline mineral waters of Karlsbad, Marienbad, Kissengen, Ems, Vichy, Eger, Franzensbrunnen, or Homburg, are to be advised. The diet must be carefully prescribed, and regulated daily, as already indicated at p. 906.

PIGMENTARY DEPOSITION OR INFILTRATION OF THE LIVER.*

Definition.—*A granular pigment found in the blood, partly free and partly inclosed in cells, due to the influence of malaria, and which, entering all the*

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* Not recognised by the College of Physicians.

organs of the body by their capillaries, colours them more or less. Next to the spleen, the greatest amount of pigment is found in the liver and brain.

Pathology.—*Pigmentary Degeneration of the Liver*, the result of *melanæmia*, is only to be recognised at a *post-mortem* examination. It occurs in severe or pernicious remittent, intermittent, or malarious fevers. It is due to the accumulation of pigment-matter in the vascular apparatus of the gland, especially in the capillary network of the portal and hepatic veins; and the minute branches of the hepatic artery contain quantities of black colouring matter. Similar melanic matter is generally found in the spleen, kidneys, and brain; while the blood itself may contain dark granular masses, or nucleated pigment-cells, with black granules in their interior. The spleen seems to be the seat of formation of the melanotic matter (FRERICHS), when it passes into the portal vein. The effects of this degeneration are mainly due to the destruction of blood-corpuscles with which it is associated, tending to a condition like that of *chlorosis*. The bile is secreted in large quantity. There is extensive capillary stagnation, which gives rise to obstruction of the circulation of the blood in the roots of the portal veins. Exhausting hæmorrhages of an intermittent kind are apt to occur from the gastro-intestinal mucous membrane. Profuse diarrhœa, vomiting, and serous effusions are also common occurrences. In India the lesion tends to *suppurative hepatitis*.

Treatment of Degenerations.—The main indications consist in the removal of the causes, or the cure of the disease which has induced the degeneration. Easily digested and nutritious food must be given. Where chronic congestions prevail, a strict diet plays a powerful part in effecting a cure. Alcoholic drinks, sugar, and fat are to be avoided, as well as amylaceous substances. *Aloes, rhubarb, sulphate of soda*, are useful to remove the torpidity of the bowels. *Muriate of ammonia* is recommended by Dr. Budd, in doses of from *five* to *ten* grains three times a day. It probably relieves the liver, and does good by promoting the action of the skin and kidneys.

LARDACEOUS LIVER—*Syn.*, AMYLOID DISEASE OF THE LIVER; WAXY LIVER.

LATIN EQ., *Jecur Lardaceum*—Idem valent, *Morbus Jecinoris Amylodes, Jecur Cereum;* FRENCH EQ., *Fois Lardacé*—Syn., *Maladie Amyloïde du Foie;* GERMAN EQ., *Speck Leber*—Syn., *Speckige Oder Amyloide, Entartung der Leber;* ITALIAN EQ., *Fegato Lardaceo*—Syn., *Malattia Amiloidea del Fegato.*

Definition.—*The existence of a peculiar homogeneous, translucent, albuminoid material in the hepatic arteries, cells, and texture of the liver, resembling an infiltration, but the albuminoid material is not found as such in the blood.*

Pathology.—(*a.*) *Causation.*—The liver is one of the most frequent seats of lardaceous disease; and though frequently associated with fatty degeneration, and sometimes described as *scrofulous* liver, it has no necessary connection with either conditions, but is a substantive disease, affecting the blood-vessels and the glandular hepatic cells (see Vol. I., page 129, where its causes are indicated and its pathology described).

(*b.*) *Morbid Anatomy.*—The amyloid liver is usually pale or fawn-coloured, and sometimes so congested, that it has a mottled appearance. The limits of the acini are unusually well defined, and the section or cut surface appears smooth, dry, bloodless, and firm. Its peritoneal covering is smooth, tense, often opaque, and the liver substance may be as hard as a board. The tissue is extremely slow to decompose; its specific gravity is notably increased, in some instances above 1,080; but it may be reduced to the normal density by fatty co-existing degeneration. Microscopically the entire structure seems changed, and the peculiar structure of the liver has enabled the advance of the disease to be observed, and Virchow has traced its origin to the blood-vessels. His observations show that the minute branches of the hepatic artery (intralobular vessels) are affected, and from these it gradually extends to the hepatic cells immediately adjacent. When the disease has become established in an acinus, three zones, varying in colour and appearance, are observable,—(1.) The outermost, composed chiefly of portal vessels, is generally the seat of more or less fatty degeneration; (2.) the innermost zone, made up of the intralobular vessels (of which the vein is prominent in the centre when divided in section), surrounded by pigmentary deposit normal to the liver, or when it is the seat of pigmentary degeneration. (3.) The intermediate zone, between the external and the central parts, is the site of the albuminoid material, translucent and firm. When the hepatic cells become involved they acquire a homogeneous hyaline appearance, like particles of rough ice. They lose their nuclei. A cell-wall cannot be seen inclosing them; but they appear as an ill-defined, pellucid, glistening mass. As the cells become filled with the new material they gradually increase in size, and lose their characteristic, irregular, polygonal shape. Their cohesion, too, is increased; they cannot be separated from each other, so that several cells seem to have coalesced or grown together. The texture of the blood-vessels in which the disease commences becomes greatly thickened, but still remains translucid. When the disease exists in an extreme degree, the greater is the translucency of the tissue, and the pellucid appearance gradually extends over the entire area of a lobule; while the few blood-vessels it seems to possess emit only a little watery blood from their diminished calibre. Rarely it occurs in isolated patches; generally it is distributed throughout, but is more marked in some places. Towards the circumference of the liver, and shining through the clear peritoneal covering, the lobules or acini may be seen mapped out in a remarkably defined manner; in fact, in no disease of the liver is the appearance of lobules (which as a rule are not marked) so distinct as in this particular morbid condition (WILKS, *Guy's Hospital Report*, p. 123, 1856). This is due to the way in which the change begins and progresses. The fatty degeneration makes the margins of the acini still more clearly obvious; and the approximation of the fatty degeneration and albuminoid material gives an opaque white appearance, which encircles the lobules, and maps them out in a perfectly defined manner. A section well washed will show the dead-white opaque substance running in the course of the portal vessels, surrounding the more transparent substance of the acini, in the centre of which the hepatic vein is apparent.

By these characters the lesion must be distinguished from fatty degeneration. Up till the year 1854 these two forms of disease had often been confounded. The large and hypertrophic livers are for the most part due to lardaceous disease, either simple or combined with fatty degeneration; whereas the fatty degeneration is often present in the liver without much or any hypertrophy. In the purely fatty liver, also, the specific gravity is not raised, but reduced. It is recorded as low as 1,005. When the fatty co-exists with the lardaceous disease, the specific gravity of the organ may be normal. Fatty liver consists of precisely the same elements as the normal liver in a similar structural arrangement, but an essential element, namely—the fat—is morbidly increased in its cells. The aggregate of solids and water may be slightly increased or diminished, but the percentage on the whole is reduced. Oil may be deposited to an enormous extent in the gland-cells; and these growing or swelling out, the volume of the organ, as well as its weight, may be increased to a very great extent.

Characters of the Minute Tissue-elements in Lardaceous Liver.—(1.) *Gland cells.*—The finely granular contents gradually disappear, giving place to a homogeneous clear substance, which ultimately fills the cell. In a few hepatic cells the mark or trace of the nucleus may still be seen of a shining lustre; and, when completely altered, the cell resembles a brilliant pellucid homogeneous mass. The cells are excessively coherent; not easy to separate; and, indeed, so changed that a large solid aggregate mass is alone recognisable, in which neither cell, nor areolar matrix, nor blood-vessel is to be distinguished. (2.) *Blood-vessels.*—The walls of the more delicate vessels become thickened, rigid, homogeneous, and lustrous. Their channel is narrowed, and ultimately obliterated; while the still patent portions remain patulous on section. They appear as pellucid, transparently colourless cylinders, in which no trace of the delicate structure of blood-vessels can be detected. The minute ramifications of the hepatic artery are chiefly implicated in the first instance.

Solution of iodine, in the proportions already indicated (Vol. I., p. 131, *ante*), gives a deep red colour. The careful and slow addition of sulphuric acid changes the deep red to a dirty violet, and rarely to a blue tint. The tissue which remains exempt from the degeneration is distinguished by the greater amount of blood, and by greater softness and moisture.

Diagnosis.—The following are general and local grounds for suspecting the existence of lardaceous disease of the liver:—(1.) When there is general ill-health, expressed by *marasmus*, *anæmia*, or *dropsy*, constituting the primary symptoms in cases otherwise ambiguous, or which are associated with *diarrhœa, vomiting, cardiac systolic murmur*, and *ascites*—the fluid drawn off containing sugar (FRERICHS, WILKS). (2.) In cases where (after examining the blood) such symptoms as are mentioned cannot be traced to lesions of the organs to which we have hitherto been accustomed to refer these phenomena. (3.) In cases where the constitution is enfeebled, and health is impaired by *ulcerations of bones*, by *syphilis, malarious fever, tuberculosis, malaria.*

Symptoms.—The local indications of lardaceous disease of the liver are,—(1.) Uniform enlargement of the organ; (2.) increased consistence, indicated by firmness; (3.) association of these characters with tumid spleen and albuminous urine; (4.) association of these characters with any of the conditions enumerated in the general symptoms under (3). The prominent general symptoms of this fatal degeneration being "anæmia, prostration, exhaustion," the condition of the liver, spleen, and kidneys should be investigated in all such cases, and their condition especially recorded in cases of *syphilis, caries, necrosis,* and *intermittent fever.* Death usually occurs by exhaustion, unless a rapid end is brought about by *purulent peritonitis, dysentery, pneumonia, gangrene,* or *œdema of the lungs.* The gradual enlargement of lardaceous disease is not attended with pain. The dropsy is generally that of cachexia and hydræmia, and not from any obstruction to the portal circulation (BAMBERGER).

Treatment.—It is desirable to limit the suppuration of bones as much as possible by early surgical interference. *Iodine* and *iron* are the remedies indicated by the nature of the disease and the circumstances under which it occurs; but nothing is known as to the effects of remedies.

JAUNDICE—*Syn.*, ICTERUS.

LATIN EQ., *Morbus Regius;* FRENCH EQ., *Jaunisse;* GERMAN EQ., *Gelbsucht* —Syn., *Icterus;* ITALIAN EQ., *Itterizia.*

Definition.—*Certain morbid conditions in which many of the different tissues and fluids of the body are dyed yellow, or otherwise discoloured, more especially the conjunctiva and the connective-tissue, from the colouring matter of the bile.*

Pathology.—(*a.*) *Causation.*—Jaundice, though often a result of organic disease of the liver or duodenum, may yet occur when these organs are perfectly healthy or simply congested. On *post-mortem* examination, besides the yellowness of the cutis, the serum of the blood is generally found loaded with bile, and perfectly yellow. If jaundice has continued for some time, the fat is also yellow, as well as the bones and cartilages, the serous fluids, and even the milk expressed from the breast of the female. The disease may arise in two ways—(1.) By *mechanical obstruction* to the passage of the bile into the intestines, and the consequent reabsorption of the detained fluid into the blood. This may arise—(*a.*) from *impaction of gall-stones, inspissated or gritty bile, mucus, or parasites* in the hepatic duct or common bile-duct; (*b.*) *catarrh of the mucous membrane of the biliary passages or of the duodenum, about the orifice of the ductus communis choledochus, or stricture and closure of it;* (*c.*) *pressure upon the duct* from tumors about the head of the pancreas, growths from the liver, or enlarged glands in the portal fissure; also from fæcal accumulations in the colon; and ovarian, uterine, or renal tumors. (2.) The suppression of the biliary secretion, arising from some morbid condition of the liver itself, whereby biliary ingredients accumulate in the circulation (BUDD). Some of these ingredients or constituents of bile are generated in the liver itself —(*e.g.*, the bile acids); others exist pre-formed in the blood (*e.g.*, the green bile-pigment, or *biliverdine,* and the *cholesterine*). The mechanism of jaundice has therefore been regarded from two points of view—namely, (1.) Jaundice from suppression, retention, or non-elimination (BUDD, HARLEY); (2.) jaundice from reabsorption of bile (FRERICHS). The first of these forms is characterised by the rapid accumulation of green bile-pigment in the blood, until the serum, the tissues, and the urine are saturated with the pigment. Over this secretion mental states exercise a remarkable influence; so much so that mental emotion favouring congestion of the organ, also favours the stoppage of the secretion. Thus jaundice by suppression or non-elimination arises from—(1.) Innervation (HARLEY). Active and passive congestion similarly induce jaundice; hence jaundice by suppression or non-elimination arises also from (2.) disordered hepatic circulation (HARLEY). (3.) Another form of jaundice by suppression or non-elimination arises from loss or destruction of the secreting cells of the liver, as in *acute* and *chronic atrophy*, *cancer*, *tubercle*, *fatty degeneration*, and *lardaceous disease*. This form of jaundice also occurs in the course of yellow fever, malarious and relapsing fever; and when certain *poisons* are present in the blood—*pyæmia*, *snake-bites*, *phosphorous poisoning*, *mercury*, *copper* and *chloroform* (HARLEY). The second class of cases arises from the reabsorption of the secreted but retained bile. They are characterised by the accumulation of pigment in the blood; whence it stains the tissues, the urine, and the serum. The bile in these cases is absorbed from the distended ducts and gall-bladder; and the biliary products manufactured in the liver, equally with those formed in the blood, find their way back into the circulation, to be eliminated by the excretions. Hence the bile-acids (absent in the first class of cases of jaundice) are present in cases of jaundice from reabsorption, as well as the bile-pigment. These acids are said to possess the property of dissolving the red blood-corpuscles. Obstruction is chiefly due to the two following causes, namely:—(1.) Congenital deficiency (very rare); (2.) obstruction by disease of parts in the vicinity of the head of the pancreas, or of the *ductus communis choledochus*.

Symptoms.—Jaundice (from the different intensities of the colour of the skin) has been divided into the *yellow*, the *green*, and the *black* jaundice. It may come on suddenly, or it may be preceded for a few days by great depression of spirits, lassitude, and somnolescence, accompanied by slight pain in the region of the liver, but more commonly pain is not present. The first sign is a yellowness of the white of the eyes, then of the roots of the nails. It next appears upon the skin over the face and neck, and ultimately over the trunk and upper and lower extremities. The skin may present a variety of tints, from slight yellowness to a brownish or blackish green. The urine becomes of a deep red colour, stains linen yellow, and if nitric acid be gently added, a line of deep green is formed where the acid meets the urine. The bile is not always in the same state of combination in the urine, nor of the same quality. In some instances, where the colour of the patient is most marked, and the urine of its deepest hue, the addition of nitric acid effects no change. At the same time that the urine is thus discoloured, the stools, often copious, are white from the absence of bile in the alimentary canal. The pulse and heart's action is slow; it may fall as low as twenty in a minute. The patient complains of a bitter taste in the mouth, has much thirst, an absolute inaptitude for exertion, and suffers from a lowness of spirits amounting to hypochondriasis. In general the bowels are irritable and easily acted upon. In a few cases they are constipated; the stools are always unhealthy, and very offensive, from decomposition of intestinal contents in the absence of bile, and consequent flatulence. If the patient recovers, the first symptom of recovery is the appearance of bile in the stools, after which the yellowness fades away in the inverse order of the attack. The duration of jaundice is such that in some cases it terminates in about ten days, but generally it lasts from three to six weeks, or as many months.

Diagnosis.—Jaundice is to be distinguished from *chlorosis* and that sallow state which results from profuse uterine hæmorrhage, in which the white of the eye is clear, the urine limpid, and the stools healthy; so that the characteristics of jaundice are wanting. It is difficult, while it is of importance, to determine how far the jaundice is due either to obstruction or to non-elimination (suppressed secretion); and to Dr. Harley we are mainly indebted for methods of clinically distinguishing these two classes of cases by chemical examination of the excretions. His principle of diagnosis from this point of view assumes that in jaundice from suppression of bile, only those biliary products which exist pre-formed in the blood accumulate in the circulation; but that in jaundice from obstruction, the biliary products which are manufactured in the liver, equally with those pre-formed in the blood, find their way back to the blood, to be eliminated by the excretions. Cutaneous itchiness is often a distressing symptom, believed to be due to the bile acid in the blood. The absence of bile from the stools is indicated by their pipe-clay colour, offensive odour, and presence of fat. The amount of pigment in the urine is best judged of by the intensity of the colour of the uric acid crystals; but it is the presence of *biliary acids* in the urine which Dr. Harley considers characteristic of jaundice from reabsorption, as distinguished from jaundice arising from non-elimination or suppression. The readiest mode by which the biliary acids may be detected is the following:—

"To a couple of drachms of the suspected urine add a small fragment of loaf sugar, and afterwards pour slowly into the test-tube about a drachm of strong sulphuric acid. This should be done so as not to mix the two liquids. If biliary acids be present, there will be observed at the line of contact of the acid and urine—after standing for a few minutes —a deep purple hue. This result may be taken as a sure indication that the jaundice is due to obstructed bile-ducts. On the other hand, the absence of this phenomenon, and the occurrence of merely a *brown* instead of a *purple* tint, although in the earlier stages of jaundice equally indicative of suppression, is no indication of the cause of the suppression, which must be gleaned from other circumstances" (HARLEY, *On Jaundice*, p. 61).

Dr. Felix Hoppe's method is as follows:—

"(1.) Decompose the icteric urine to be examined with an excess of milk of lime; (2.) boil for about half an hour; (3.) filter; (4.) evaporate the filtered fluid nearly to dryness; (5.) decompose with a great excess of concentrated hydrochloric acid, and then keep the whole (before being again filtered) at the boiling-point for half an hour; (6.) to avoid spurting of the fluid it is necessary to renew the volatilised hydrochloric acid from time to time; (7.) leave the liquid to get completely cold, and then add six to eight times its volume of water; (8.) filter the dark-brown turbid solution thus obtained, wash out with water the residue on the filter, until the same runs through quite colourless; (9.) dissolve the brown resinous mass on the filter in 90 per cent. alcohol; (10.) decolourise by boiling with animal charcoal, filter, and evaporate to dryness in the water-bath; the residue is a yellow, resinous mass, which, if bile-acids be present, must consist for the most part of pure *choloidic acid.* In such a case it melts by warming, and emits the peculiar musk or soap odour. (11.) Lastly, dissolve in a very little caustic soda and some drops of warm water, add a very small piece of sugar, and allow three drops of concentrated SO_3 slowly to fall into it. At first the fluid becomes milky and troubled, and resinous flakes separate, which stick pertinaciously to the glass; but afterwards, by the addition of more SO_3, these again dissolve and produce a beautiful purple-red or dark-violet fluid" ("Abstract of Kühne's Paper on the Pathology of *Icterus*," by Dr. GEORGE SCOTT, of Southampton, in Beale's *Archives,* Vol. I., p. 343).

But these acids after a time gradually diminish and disappear from the urine in cases of obstruction and reabsorption when the secreting powers of the liver become impaired, when the hepatic gland-cells waste away; and then the abnormal products, *leucine* and *tyrosine,* appear in the urine. They are readily detected by the slow evaporation of an ounce of urine to the consistence of syrup, which is to be put aside to crystallise. *Tyrosine* is recognised by the fine stellate groups of needle-like crystals or spiculated cells, which appear like a rolled-up hedgehog with its bristles sticking out in all directions (HARLEY). It may be obtained in its pure state by—(1.) Adding a solution of *acetate of lead* to four ounces of urine, till a precipitate ceases to form; (2.) filtering; (3.) freeing the liquid from an excess of lead by a current of sulphuretted hydrogen; (4.) again filtering; and, (5.) evaporating the clear solution. The tyrosine is now colourless, and crystallises with the microscopic characters better marked. *Leucine* is known by its flat, circular, oily-like discs, without crystalline structure. It is soluble in water and boiling alcohol, but insoluble in ether. If sugar appears in the urine in cases of jaundice, with a diminution of bile-acids hitherto in quantity, it is generally the forerunner of a fatal termination (HARLEY).

Treatment.—As a general principle, the larger number of cases of jaundice from functional disorder (perhaps four out of five) get well spontaneously, aided by remedies judiciously selected according to the diagnosis already indicated. The first indication is to aim at removing the exciting cause; and in jaundice due to congestion of the liver, purgatives seem to act beneficially in the form of *blue pill* or *Plummer's pill,* with *aloes,* and *nux vomica* with *rhubarb pill mass.* In cases of acute jaundice—from suppression of the biliary secretion consequent on a powerful nervous shock or mental perturbation—Dr. Anstie has seen two or three doses of the *hydrochlorate of ammonia,* to the extent of grs. xx. every four hours, produce a restoration of the biliary secretion. He regards it as a most powerful restorative of the biliary functions. Acids and alkalies are alike contra-indicated in cases of jaundice resulting from active congestion of the liver. *Benzoin acid* has been recommended; and Dr. Harley has found it useful in cases of suppression of bile (by innervation, for example); but in cases of jaundice from obstruction it is injurious. *Podophyllin* is also of use in jaundice from suppressed secretion of bile. It ought to be combined with *hyoscyamus.* It is especially useful in cases of feeble liver, combined with vegetable tonics, such as *gentian* and *quinine;* but it ought not to be given in cases of jaundice from obstruction. The medicine of all others which has seemed most generally useful is *sulphate of magnesia,* in half drachm to drachm doses, combined with fifteen grains of *carbonate of magnesia,* and half a drachm of *aromatic spirits of ammonia,* given three times a day an hour before food. The *sulphate of magnesia* maintains a free action of the bowels; and the *carbonate of magnesia* neutralises any excess of acid in the stomach or bowels (BUDD, *op. cit.*, p. 289. See under *Hepatitis* and *Congestion*).

ACUTE ATROPHY OF THE LIVER.

LATIN EQ., *Atrophia Acuta;* FRENCH EQ., *Atrophie Aiguë;* GERMAN EQ., *Acute Atrophie;* ITALIAN EQ., *Atrofia Acuta.*

Definition.—*Simple jaundice, which gradually increases, with sensitiveness over the region of the liver, followed by violent constitutional disturbance, expressed by pyrexia, headache, delirium, hæmorrhages from various parts, and finally coma. The liver shrinks to one-half or one-third its normal size. A peculiar chemical decomposition takes place in the liver, whereby abnormal proximate principles are formed, which, being carried into the blood, may be discovered in various organs of the body, or, passing out by the kidneys, may be found in the urine.*

Pathology.—(*a.*) *Causation.*—The origin of acute atrophy of the liver is still unknown; but mental distress, violent mental emotions, dissolute habits, venereal excesses, syphilis, drunkenness, nervous depression, the influence of miasmata, the condition of pregnancy, typhus, pyæmia, scarlet fever, tropical malarious fevers, and poisoning from phosphorus, are the circumstances under which this form of inflammation has occurred. The destruction of the liver cell-tissue which ensues, is indicated during life by the symptoms of atrophy, and by those peculiar changes in the urine which, at the height of the disease, are almost pathognomonic. In the cases described by Frerichs the urine is described of a brown colour, and smelling of sulphurated hydrogen, having a feebly acid reaction, and containing a small quantity of bile-pigment, but no albumen. Looking to the occurrence of albumen in the urine of pregnant women, it may be that atrophy of the liver and the *nephritis* of pregnancy are both due to some condition of blood which in certain states of the constitution may be induced by pregnancy (WILKS). Atrophy of the liver has been most frequently observed in females; and more than half were attacked during pregnancy; and the period of life between twenty to thirty years of age seems most predisposed to the disease.

(*b.*) *Morbid Anatomy.*—The disease was first described by Bright and Graves, more recently and minutely by Frerichs in Germany, Wilks and

Murchison in this country. The liver undergoes a most remarkable atrophy. Its section presents a mottled red and yellow appearance. The microscope shows that complete destruction of its secreting cells occurs, so that in the most severe cases not a single cell has been left entire; dark masses of biliary pigment, hæmatine, and fat alone remain; and the kidneys are found gorged with new products. The redder portions of the liver are more destroyed than the yellow, which may show remains of cells containing bile. It seems the result of a form of hepatitis, of the nature of *parenchymatous inflammation,* already frequently referred to—*cloudy swelling* of the cell-elements, and their subsequent destruction by softening disintegration, so that the acini are not capable of recognition. The parenchyma is thus relaxed, shrivelled, and flabby, and the liver sinks against the posterior wall of the abdomen. The size of the organ is diminished in all directions, but especially in its thickness. The gland is flattened out, flabby, and wrinkled. Where the disease has advanced the farthest, the cut surface has an ochre-yellow or rhubarb-like colour; the blood-vessels are empty; and the traces of the outlines of lobules are obliterated. Where the process is less advanced, extravasations exist, or their remains, in the forms of crystals of *hæmatoidine.* The lobules are encircled by congested vessels; and a dirty yellow substance separates them from each other. Afterwards the capillary congestion recedes, the size of the lobules diminishes, their colour becomes yellower, whilst the intervening grey substance gradually increases, and extravasations exist upon mucous and serous membranes. Bright has recorded the weight of such livers as reduced to *two pounds,* or to *twenty-three ounces,* and even to *nineteen ounces.* Frerichs records the weight of one as low as *one pound thirteen ounces;* its relative weight to the entire body being as 1 to 68 or 54—*i.e.,* a reduction of more than one-half. Murchison records 28 ounces; or one-half the standard for the age of the patient.

The **Symptoms** set in like a bilious attack, with feebleness, rapidly followed by jaundice, febrile symptoms, and vomiting, delirium supervening by the third or fourth day. Convulsive fits soon pass into unconsciousness, associated with stertorous breathing, foaming at the mouth, clenched teeth, closed eyes, with pupils normal and susceptible. The pulse is at first abnormally slow, but at the outbreak of the nervous symptoms it gradually rises to 110 or 120, and presents remarkable variations as regards frequency and volume. Towards the close it increases in frequency, and becomes smaller and smaller till it can no longer be felt. The body-temperature rises very high. The symptoms generally resemble those of uræmic intoxication; collapse increases, perspiration becomes copious, and the patient usually dies comatose about the second day, more rarely about the fourth or fifth day after the first appearance of cerebral symptoms. The extent of hepatic dulness diminishes more and more as the disease advances, and not unfrequently the dull space disappears completely, without any tympanitic distension of the bowels to cause the dull sound over the liver to be obscured. The spleen is increased in volume, and splenic dulness is increased. The bowels are almost always confined. The stools are firm, dry, clay-like, deficient in bile, and at a later period not unfrequently dark-coloured and tarry, from the presence of blood. The urine, more or less saturated with a brown colouring matter, presents the reaction of bile-pigment, and deposits a light or greenish-yellow precipitate, in which the microscope detects the epithelium of the urinary passages and of the kidney coloured yellow; and the *needle-shaped crystals* (*tyrosine*) covered with colouring matter, either isolated or adhering in crystalline masses (FRERICHS). Sometimes the urine ceases to pass, and may be retained in the bladder as a clear bilious fluid, having a specific gravity of 1030 (WILKS); or ranging from 1012 to 1024, always of acid reaction, and occasionally slightly albuminous for short periods (FRERICHS). Its evaporation gives crystals of *leucine*, which are in round masses, and of *tyrosine*, which are needle-shaped. Sometimes pure tyrosine falls as a sediment. These crystals are also found in the substance of the liver, but only in the hepatic vein, not in the portal vein or hepatic artery (FRERICHS). In Murchison's case they appeared after the liver had been immersed in spirit (*Path. Trans.*, Vol. XIX., p. 248). The question as to the retention of urea or its lessening, as due to a want of complete metamorphosis of albuminous products, is not yet determined (PARKES, l. c., p. 286). The disease runs a more or less violent and rapid course. In severe cases it has terminated at the end of twelve or twenty-four hours (FRERICHS). In other cases, in from two to eight days, urgent symptoms ending the disease by extreme prostration or excessive vomiting (WILKS). It is scarcely ever prolonged to a week, except when it occurs after jaundice, when the whole duration of the disease may extend over four to five weeks. After the commencement of the characteristic symptoms, it almost invariably terminates fatally in five days.

The **Prognosis** is, therefore, in the highest degree unfavourable. Hæmorrhages are apt to occur (in one-half the cases) simultaneously from various parts of the body, usually from the stomach, bowels, or uterus. Pressure over the region of the liver gives rise to marked indications of pain, which are expressed even during coma.

Diagnosis.—It is apt to be mistaken for typhus fever, complicated with jaundice or with pyæmia. The range of temperature may help to indicate the disease, inasmuch as the temperature is said not to be increased in cases of acute yellow atrophy (see FRERICHS, Vol. I., p. 217).

Treatment.—English physicians recommend emetics and purgatives (CORRIGAN, GRIFFIN, HANLON). Frerichs recommends that the stronger purgatives should be used simply to remove congestion, such as *senna, aloes, colocynth;* and the doses should be such as to secure profuse evacuations. When atrophy has commenced, no benefit results from medicinal treatment. Severe pains indicate the use of *leeches, cupping,* and *cold cloths,* or *fomentations,* as recommended under "Enteric Fever" in Vol. I. The occurrence of hæmorrhage indicates the use of *mineral acids.*

SECTION XIV.—DISEASES OF THE HEPATIC DUCTS AND GALL-BLADDER.

INFLAMMATION OF THE HEPATIC DUCTS AND GALL-BLADDER.

LATIN EQ., *Inflammatio;* FRENCH EQ., *Inflammation;* GERMAN EQ., *Entzündung;* ITALIAN EQ., *Inflammazione.*

Definition.—*Inflammation, generally catarrhal, but occasionally croupous or diphtheritic, of the mucous membrane of the biliary passages.*

Pathology.—(*a.*) *Causation.*—Catarrhal inflammation is common; the other forms are rare. The catarrhal inflammation is generally an extension of gastric and duodenal catarrh, extending through the *ductus communis choledochus;* and the causes are those already mentioned which lead to congestion and enlargements of the liver.

(*b.*) *Morbid Anatomy.*—The *catarrhal* form is recognised by excessive secretion from the biliary passages; the *diphtheritic* form by a fibrinous exudation which may occur during *typhus, septicæmia,* or *cholera.*

Symptoms.—There is usually excessive hyperæmia of the liver, causing simple enlargement, with symptoms of obstruction to the flow of bile and of jaundice. The tongue is coated, and there is a bad taste in the mouth, eructations from the stomach, and dyspepsia; and there may be signs of gastro-duodenal catarrh. The fæces are free of colour, and of a whitish, dirty clayey aspect.

Treatment is similar to that of catarrh of the intestines, and especially in the use of *nitro-muriatic acid* internally and as baths.

GALL-STONES; GALL-STONE OR BILIARY COLIC.

LATIN EQ., *Calculi Fellei;* FRENCH EQ., *Calculs Biliaires;* GERMAN EQ., *Gallensteine;* ITALIAN EQ., *Calcoli Biliari.*

Definition.—*Concretions of certain biliary constituents in the biliary ducts, or accumulating generally in the gall bladder, as gall-stones, the passage of which through the duct into the duodenum gives rise to symptoms known as "gall-stone colic."*

Pathology.—(*a.*) *Causation.*—The predisposing causes of gall-stones are a too full animal diet, combined with a sedentary life, or those states of indigestion which react on the functions of the liver. Gall-stones appear to be peculiar to adults; and generally occur after twenty, but commonly between forty and sixty. They affect women more frequently than men, and persons of sedentary rather than those of active habits of life. They are particularly frequent in patients with carcinoma of the stomach and liver (NIEMEYER).

(*b.*) *Morbid Anatomy.*—Bile in its healthy state is of a deep yellow colour, extremely bitter, followed by a sweetish after-taste. It is a little heavier than water (sp. gr. 1,018), and miscible in that fluid in every proportion. In the gall-bladder bile is usually of the consistency of thin molasses, having a specific gravity of 1,026 to 1,032—cystic bile. It contains a little free soda, and is feebly alkaline or neutral. The daily amount secreted by a man is about 56 oz. (Bidder and Schmidt). The proportion of solid matter it contains is from 9 to 17 per cent.; the cystic bile, about 10 per cent.; hepatic bile, 3 to 5 per cent., composed of substances peculiar to bile; among which may be distinguished two resinous acids,—namely, the *Glycocholic* or *cholic acid* in small quantity; and the *Tauro cholic* or *choleic acid* (*bilin*), in which 25 per cent. of *sulphur* of the bile exists. They are united in the bile with soda as a base.

The bile may be green or yellow, and those colours may be pale or intense; or it may be as fluid as water, or as viscid as tar. Its taste also varies, sometimes bland, or so acrid as to excoriate the lip. These different states are found in the most oppositely diseased conditions of the liver. Under certain circumstances the diseased bile assumes a concrete form—that of gall-stones. Although their principal seat is the gall-bladder, they have been found "*in transitu*" in the *cystic duct* and in the *ductus communis.* Occasionally they have been found in the *hepatic ducts,* where they are apt to excite *suppurative hepatitis* (Cruveilhier). They are sometimes found in the intestinal canal, after having passed from the gall-bladder into that cavity. They may be found filling the gall-bladder, the structure of the liver and gall-bladder being perfectly healthy, even when the gall-bladder contains numerous calculi with sharp angles and edges. In the ducts of the liver they may be branched or coral-like.

In some instances the *ductus cysticus* is obliterated, so that *hydrops vesicæ felleæ* may result with biliary congestion. Or the gall-bladder is thickened by chronic inflammation, or ulcerated, particularly at the fundus, where considerable congestion of blood and œdema of the mucous membrane may exist, short of ulceration. When ulceration commences it may lead to perforation of the gall-bladder, opening directly into the peritoneum or into some part of the intestinal canal, usually the stomach or duodenum, limited by adhesive inflammation. The walls of the bladder become contracted, its cavity diminishes, and its contents dry up into a chalky mass, in which the previously existing biliary calculi are imbedded as in a cyst. If the calculi are imprisoned in the ducts of the liver, hepatic abscess may result. Perforation may occur through the abdominal wall. In rare instances any of the bile conduits may be obliterated from inflammation, in consequence of the constant irritation. Fistulous communications may thus be established in various directions from the gall-bladder, and large biliary calculi may be found imbedded in various places, sometimes causing intestinal obstruction.

Chemically, gall-stones are composed principally of *cholesterine* and *colouring matter,* in various proportions, with some *animal matter,* the usual *bile salts,* and perhaps a trace of *iron. Bile pigment,* with *choloidinic acid* and its calcareous base, occasionally accumulate in solid masses. *Cholesterine* (which may exist in the large proportion of 88 to 94 per cent. of the whole calculus) is soluble in *boiling alcohol, ether,* and in *nitric acid.* It is tasteless, inodorous, and burns by the flame of a lamp till it is altogether consumed. It is also lighter than water, and insoluble in that fluid. The colouring matter (generally combined with cholesterine, often forming of itself a large portion of the gall-stone) is inodorous, insipid, and heavier than water. It is likewise insoluble in that fluid, in alcohol, or in acids; but is soluble in alkalies, whence it is precipitated, of a brownish-green colour, on the addition of water. The calculi found in the human gall-bladder have been divided into *crystallised, deposited, amorphous,* and *porcupine-like* calculi (Dr. POWEL). The *crystallised* concretions, when fractured, look like spermaceti; and the crystals, like those of that substance, are easily broken into a greasy powder. They are semi-transparent, but seldom retain their purity throughout, being, near their circumference, coated or mixed with a brown colouring matter. At the central point of these colourless crystals, to which radii converge, there is mostly a small particle of coloured matter, resembling dried bile, which has served as the nucleus of crystallisation. This crystallised part having reached the size of a pea, becomes itself a centre around which many deposits are made of variously confused and irregular strata, the surface of which may in turn become the starting-point of a fresh crystallisation. The *deposited* gall-stone consists of biliary matter in laminæ, like the arrangement of an onion or of some urinary calculi. The *porcupine-like* calculi are small and round, having a number of projecting points. The *amorphous concretions* are such as bear no mark of crystallisation, or of any regular structure; but as they dry they break into layers, showing a laminated mode of formation.

Biliary calculi vary considerably in their specific gravity. This probably depends on the greater or less quantity of animal matter they may contain. They vary in number. From one to 1,000 have been found in the gall-bladder. When single they are usually of a round or oval figure. In size they vary from a pin's head to that of a nutmeg or a walnut, or they may be even as large as a hen's egg. When extremely numerous, they are small, of a dark brown colour, and occasionally slightly agglutinated with viscid bile. When the number is small (from two or three to eight) the size may be considerable. When the accumulation in the gall-bladder is made up of several, they are loosely adapted or fitted to each other by facets, owing to their mutual friction.

As to the formation of gall-stones, differences of opinion exist. The *cholesterine* of healthy human bile is only in a very small proportion, and is produced in excess in a fluid state. From this, if a nucleus of any kind (*epithelium* or *mucus*) be present, crystals may immediately form upon it. Thus a person apparently in good health may have a large gall-stone formed in his gall-bladder. Deposited gall-stones are formed by an excess of colouring matter, or by some morbid state of the bile, in which that principle is readily separable when any nucleus is present. Inspissation or mere concentration of the bile; deficiency of certain ingredients, such as the alkaline constituents, with excess of the acids; decomposition, and other chemical changes resulting in precipitation of cholesterine and pigment, all favour the process of gall-stone formation.

Symptoms.—The formation of gall-stones is unattended with pain, and, once formed, may lie latent for a considerable time in the gall-bladder without causing any trouble to the patient. At length, some cause (exertion in a stooping posture) may force a stone into the *cystic duct,* when a series of formidable symptoms arise, which continue till the calculus has passed into the duodenum. Occasionally calculi of small size may pass through the *ductus cysticus* and *communis choledochus* without exciting pain or any other symptom. The experiences at such watering-places as Karlsbad, where the stools are carefully examined for gall-stones, afford numerous examples of this (NIEMEYER). The painful symptoms which attend the passage of gall-stones into the duodenum have received the name of *gall-stone* or *biliary colic.* The attack is sudden. The patient is seized with shivering, accompanied by violent and acute piercing, griping, insupportable pain at the pit of the stomach, or at the point corresponding to the opening of the duct into the duodenum. From this point it spreads over the whole abdomen to the right side of the thorax and right shoulder, and darts through the back. It occurs in paroxysms, varying from a few minutes to a few hours, till the gall-stone has passed into the intestine. The pain is so great that patients most tolerant of pain moan and double themselves up, or roll about on the bed or floor. Nausea or vomiting may be so severe that everything is rejected, and the matters thrown up may contain bile and small biliary calculi or biliary sediment. Excessive pain and vomiting are the leading features of the passage of a gall-stone. It is impossible for those who have witnessed a case of this kind not to be struck with the resemblance many of its symptoms bear to those of parturition—a comparison women frequently make when describing their sufferings. There is this difference, however, that when the pain intermits there is a deep-seated soreness and fulness of the right hypochondrium and epigastric regions. Like parturition one attack of pain succeeds another, till at length this more urgent symptom ceases, and the calculus may be inferred to have passed into the intestine. After that has taken place, the soreness and uneasiness gradually cease, and the patient is relieved. In some cases, at an early period of the attack, jaundice makes its appearance, and may continue after the calculus has passed. *The pulse* during the paroxysm is for the most part natural, unless the patient is exhausted by long continuance of pain, when it becomes small, the skin cold, and the face pale and distorted. The patient may faint; and prolonged faintness is a rare cause of death from gall-stone colic. The heat of the body also is not increased. The dejections, according as the obstruction is more or less complete, are clay-coloured or natural; and, on a close examination from time to time, they may be found to contain the offending calculus, the surface of which should be carefully examined for *facets* or marks of the presence of others remaining behind. The duration of the attack may be only a few hours or a few days, or several weeks may elapse before the gall-stone is expelled. Gall-stone colic is particularly liable to occur during digestion.

Diagnosis.—The passage of a gall-stone is to be distinguished from hepatitis by the pains being in general of greater intensity, and paroxysmal; and also by the pulse continuing natural.

Prognosis.—The prognosis is always favourable, unless the calculus be of such magnitude as to render its passage almost impossible, or unless it be connected with organic disease of the liver.

Treatment.—When the symptoms of gall-stone passing the duct are present, the curative indications are to facilitate its passage into the intestine, to relieve the intense pain which accompanies it, and to prevent that inflammation which the presence of an extraneous body of any magnitude is calculated to produce in the duct. The first thing to be done is to calm the sufferings of the patient. Half a grain of solid *opium*, or a quarter of a grain of *morphine*, or twelve drops of *laudanum*, or the *mistura camphoræ*, ℥xj., conf. opii ʒss. to ℈ij., c. sp. æth. nitr. ℥j., should be given every hour, or every two hours, till some relief is obtained, or till there is slight narcotism. Similar doses may be repeated every four or six hours, till the pain has ceased. If the vomiting be severe, and the remedies be rejected, the opiate may be given *hypodermically*, commencing with not more than one-fourth of a grain of the *muriate of morphia* in solution; or opium may be administered by enemata of *laudanum*. Careful *inhalation of chloroform*, till anæsthesia is produced, also gives great relief, and is a valuable remedy when paroxysms are most violent. It not only calms pain, but may lead to the cessation of the spasmodic contraction of the biliary passages, and thus favour the expulsion of the calculus (MURCHISON). *Chloroform* also, when given internally, appears to exercise some influence as a solvent of biliary concretions. Dr. J. Barclay relates an instance in which ♏ ii. to ♏ iii. of *chloroform*, given three or four times a day, afforded great and immediate relief (*Brit. Med. Journal*, 1870). Dr. Murchison has seen marked benefit from the *extract of belladonna*, given in half grain doses every two or three hours. The *hydrate of chloral*, as it produces extreme muscular relaxation, is also a likely remedy. Small pieces of ice in the mouth may relieve vomiting. Emetics or laxatives are not to be given during the attack. A warm bath should be immediately prepared, with a temperature of 100° to 110°, or as hot as the patient can bear it, and the immersion should continue till he is in some degree exhausted. The intention of the bath is to relax by means of heat the muscular fibre of the ducts, and thus relieve the pain and facilitate the passage of the gall-stone. The effect is generally so agreeable to the feelings of the patient that, on the recurrence of the pain, he may ask for a repetition of it, and his wishes should be complied with. If a warm bath cannot be procured, fomentations, or a large linseed poultice, should be applied over the abdomen. Dry heat is always at hand, and hot flannels, hot sand, or hot chamomile flowers afford some relief. Bleeding is not called for. The calculus having passed, and the patient being relieved, the secretions of the liver should be rendered more healthy. A course of neutral salts, or of the Cheltenham or Leamington waters, ought to be taken in this country. Under the use of the Karlsbad waters immense quantities of gall-stones are evacuated with proportionately little difficulty (NIEMEYER). The same is true of other *alkaline* mineral waters, such as Vichy, Marienbad, and Kissingen.

The radical treatment of biliary calculi ought to be attempted during the intervals between the attacks of gall-stone colic, by—(1.) *Solvent believed to act on the calculi.* These are,—(*a.*) *Alkaline solvents*, which ar to be preferred above all others. Under their influence the calculi seem to be really dissolved or broken up, so as to disappear without leavin any trace; or they are expelled entire in abundant bilious evacuations This alkaline treatment comprises the *fixed alkalies, soap-lye, salts of soda carbonate of ammonia;* but especially the waters of Vichy, Vals, Karlsbad and Ems. These are used as drinks and as baths; and they must b employed perseveringly, at different periods, during several years in suc cession. (*b.*) *Durande's remedy,* which consists in the administration of half a drachm to a drachm every day, in the morning, of a mixture composed of *fifteen grammes* (*i.e.,* about ʒiii.) of *sulphuric ether,* and *ten grammes* (*i.e.,* about ʒii.) of *oil of turpentine,* has enjoyed a reputation since 1790, as a solvent of biliary calculi. It has been extensively used and recommended by many Continental physicians. In this country Copland speaks favourably of it; but whatever virtue the remedy may possess as a solvent, it is probably due more to the *ether* than the *turpentine,* the former being a ready solvent of cholesterine (Waring). The dose is to be gradually increased till about a pound of this mixture has been taken. Its mode of action is not known; but where it has succeeded, the calculus has been expelled without being dissolved. It seems in some way efficient in promoting the expulsion of calculi, probably by its anti-spasmodic action, rather than acting as a solvent. Recently various substitutes have been proposed in place of Durande's remedy, and also variations from the original dose (Niemeyer). One is a popular remedy, a mixture of *oil of turpentine,* ℈ii., with ʒi. of spirits of *sulphuric ether,* which has been prescribed in *drop doses* by Rademacher. (*c.*) *Diet* has a great influence upon the quality of the bile. Fresh laxative vegetable food, grapes, fruits, and whey ought to constitute the staple articles of a regulated diet. All fat should be as much as possible excluded from food, and the plainest diet, in the form of roasted meats or boiled meats, with vegetables and farinaceous food, should be rigidly proportioned to the wants of the system. The drinks should be water or lemonade, and an avoidance of alcoholic or malted stimulants. (2.) *Treatment which seeks to assuage pain.*—With this end in view, Lutten prescribes *opium* in larger doses than those already mentioned—namely, two and one-third to three grains of solid opium at once; but he considers the hypodermic injection of *solution of morphia* a preferable (*Nouveau Dictionnaire de Médicine et de Chirurgie Practiques;* also, *Society Med. de Paris,* 1866; and *Sydenham Society's Biennial Retrospect* for 1865-66). The intense agony is more effectually relieved by large doses of opium than by any other remedy, particularly if it be combined with the use of the hot bath (Dr. Waring). Two grains of *solid opium,* or ♏ xl. of the *tincture of opium* may be given, either by mouth or in the form of enema, to be repeated in half an hour if the pain is not relieved. It may also be advantageously combined with a full dose of *ether* or *chloroform,* care being taken in case of over-dosing with the opiate.

Large draughts of hot water, containing *carbonate of soda* in solution (ʒi. to ʒii. to twenty ounces of water) give more immediate relief than any other means (Dr. Prout). The first dose or two may be rejected; but the addition of a few drops of laudanum and steady perseverance in the remedy will eventually ensure its success (Murchison). No doubt can exist as to the efficacy of *alkaline* waters in removing the symptoms of *gall-stones.* It is certain that the salts of Vichy and of Karlsbad waters relieve the symptoms of this affection, provided they are administered in hot water, and so as to represent nearly the strength of the natural springs.

CHAPTER XX.

DISEASES OF THE URINARY SYSTEM.

SECTION I.—THE KIDNEY AND ITS SECRETION IN RELATION TO DISEASES OF THE URINARY SYSTEM.

A CAREFUL study of the structure and functions of the kidney is of importance in relation to diseases of the urinary organs. The determination of the quality and quantity of urine is one of the best methods of determining not only morbid conditions of the kidney, but of appreciating many changes which go on in the body during disease. In every case of disease (in wasting, febrile, and constitutional diseases especially), much may be learned regarding their natural history by a careful examination of the urinary excretions, and of the microscopic characters of the sediment, especially when studied in connection with the ranges of temperature of the body, the number of respirations, and the state of the pulse, during daily periods of twenty-four hours. A formula has been already suggested at pages 259 to 275, Vol. I., as a method to be followed in daily records; and it is now clearly understood that the *absolute amount excreted in a given time, and referring that amount to body-weight*, are the only conditions which will yield anything approaching to accurate scientific results (PARKES). "In a medical point of view it is a mere waste of time to estimate the constituents in a certain quantity of urine, passed at any particular hour of the day or night, without ascertaining the relation which that quantity, with its constituents, bears to the whole quantity passed during the *twenty-four* hours; and this is the more necessary as the amount of fluid secreted varies considerably in healthy persons (SUTTON). The volumetric methods of determining the urinary constituents have greatly facilitated such investigations; and these methods are clearly expounded in the works of Hoppe, Neubauer, and Vogel, Thudichum, Beale, Hassall, and Sutton; and, above all, the profession is indebted to the late Dr. Parkes for his philosophical exposition of the value of our knowledge regarding the state of the urine in health and disease, as set forth in his admirable work on that subject, already frequently referred to. From these works the following sections are compiled.

SECTION II.—ON DETERMINING THE COMPOSITION OF THE URINE IN DISEASE.

It is examined for two purposes,—(1.) To discover the condition of the urinary organs; (2.) to determine the course of the abnormal metamorphoses of tissue in the body which lead to alterations in the composition of the several excreta. The normal urinary constituents have probably a fixed physiological range concurrent with age and weight of the body, so that a person ought not to continue passing regularly an amount of any constituent greatly above or below the limits of the range proper to him, otherwise some morbid condition must be sought for to explain the nature of the occurrence. The averages of the excreta for at least ten days should be taken in health, in order to arrive at an accurate standard. But as this is not possible in cases of disease, certain empirical formula have been laid down for calculating the urinary excretion in a sick person whose normal excretion is unknown. The following are the details of the facts to be determined and recorded:—(1.) Record the age and weight of the patient in pounds avoirdupois.* (2.) Collect all the urine passed in twenty-four hours; measure it in cubic centimetres, and record its absolute amount. (3.) Observe and record as to the following general properties, viz.:—(*a.*) Specific gravity; (*b.*) Uræmatine, as determined by Vogel's colour table;† (*c.*) Clearness or turbidity on emission or after rest; (*d.*) determine the absolute weight, by multiplying the quantity passed, expressed in cc., by the figures expressing the specific gravity,—the result is the weight in grammes. (4.) Set aside the following quantities for the volumetric determination of urea, 40 cc.; uric acid, 300 to 500 cc.; phosphoric acid, 40 cc.;‡ chloride of sodium, 40 cc.; sulphuric acid, 100 cc.; degree of free acidity—sugar, 20 cc.; albumen—solids, 20 grammes. (5.) Collect and examine the sediment. (6.) Determine the amount of excretion normal to the individual by the following empirical formula (PARKES):—Multiply the following figures by the weight of the person in pounds avoirdupois; the result is the excretion in grains, in twenty-four hours, of the several ingredients of the urine. It is necessary to make corrections:—(1.) If the person be between forty and fifty, calculate according to columns 1 or 2, and then deduct 10 per cent.; for ages between fifty and sixty deduct 20 per cent.; for ages between sixty and seventy deduct 30 per cent.; for ages upwards of seventy deduct 50 per cent. (2.) If the person has been starving for two or more days (as in some fevers), deduct one-third from the calculation made according to the table; if the diet be meagre, deduct one-eighth or one-sixth; if pretty plentiful, yet still below that of health, deduct one-tenth. (3.) If there be total inactivity, deduct one-tenth; if there be merely quietude, deduct one-twentieth.

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* Beneke recommended, in 1854, the very useful "*Patent Decimal Waagen*" *of Schoenemann* (patent decimal balances), for the determination of the body-weight of patients, and also for physiological and clinical investigations. These balances are manufactured by Messrs. Kuhtz & Co., at Brandenburg, on the Havel. They are made either entirely of iron or partly of wood. In the latter case the absolute weight of the wooden seat may change slightly with the atmospheric moisture, although perfectly seasoned dry wood is used in the construction. The balance turns with 1 gramme in 130 pounds. A small ivory scale, opposite the end of the lever, enables the appreciation of even still smaller differences of weight. The price of such a balance, constructed in wood (with weights, large and small, in a box), is 45 thalers. (See *Berlin Klin. Wochenschrift*, viii., 18, 1871; also Schmidt's *Jahrbeucher*, 1871, 8.)

† Vogel's colour table is made by taking certain colours as starting points, and representing the following groups for comparison:—

First group—Yellowish urines, represented by gamboge mixed with a greater or less amount of water, giving three grades, the first of which is almost colourless, as follow:—(1.) *Pale yellow* (gamboge with much water); (2.) *Light yellow* (gamboge with less water); (3.) *Yellow* (gamboge with very little water).

Second group—Reddish urines, represented by the above yellow grades mixed with a greater or less amount of red—*i.e.*, gamboge with crimson lake in three grades, as follow:—(4.) *Reddish-yellow* (gamboge with a little crimson lake); (5.) *Yellowish-red* (gamboge with more crimson lake); (6.) *Red* (crimson lake with little gamboge).

Third group—Brown or dark urines, represented by gamboge, crimson lake, and more or less Prussian blue, in three grades, as follow:—(7.) *Brownish-red* (red with an admixture of a little brown); (8.) *Reddish-brown* (more of the brown than in the last); (9.) *Brownish-black* (almost black, with a touch of the reddish-brown). (See THUDICHUM, *On the Pathology of the Urine*, p. 134.)

‡ The precipitate from the urea estimation is sufficient.

	In Men between 20 and 40.	In Women between 20 and 40.	In Children between		In Young Men and Women between 16 and 20.
			3 and 8.	8 and 16.	
Urea, ...	3·53	2·96	6·83	5·20	4·39
Chlorine, ...	0·875	0·817	1·44	1·097	0·926
Sulphuric Acid, . .	0·214	0·25	0·414	0·315	0·266
Phosphoric Acid, . .	0·336	0·336	0·65	0·495	0·418

SECTION III.—VOLUMETRIC ESTIMATION OF THE MORE IMPORTANT CONSTITUENTS OF THE URINE, AND THEIR PATHOLOGICAL RELATIONS.

The Volumetric Method of Analysis is one which enables ordinarily skilful operators, or medical men who cannot devote much time to practical chemistry, to determine with sufficient accuracy the amount of the most important constituents of the urine, such as *urea, uric acid, chloride of sodium, phosphates, sulphates*, and the *free acidity* of this excretion. But there are many volatile substances excreted by the kidneys which are not easily estimated. For example, *turpentine, copaiba, myrrh, iodine, rhubarb*, the *odoriferous particles of asparagus*, or of other substances. Certain conditions are necessary for success, namely,—(1.) *Solutions* of the reagents or tests, the composition and strength, or chemical power, of which are accurately known. (2.) *Burettes*, graduated tubes or vessels from which portions of the test-solutions may be accurately delivered. (3.) *The power of determining by the eye* when the decomposition produced by the test-solution with the urine has ceased, so that the quantity of test-solution used (*i. e.*, with which certain components of the urine have combined) may be accurately determined. It is necessary to exercise the greatest care in the graduation of the measuring instruments, and in the strength and purity of the standard solutions; because a very slight error in the process is greatly magnified—in proportion, in fact, as it is multiplied to represent the amount in large quantities when the actual observation has been made upon a small amount.

1. **Estimation of Chlorides.**—They are calculated as *chloride of sodium*, the test-solutions required being—(1.) The *standard solution of the nitrate of mercury;* and, (2.) the "*baryta-solution*," as it is commonly called. The method was devised by Liebig, and its principle is as follows:—"If the

solution of *nitrate of mercury,* free from any excess of acid, is added to a solution of urea, a *white gelatinous precipitate* is produced, containing urea and *oxide of mercury* in the proportion of 1 eq. of the former to 4 eqs. of the latter. But when *chloride of sodium* is present in the solution, *the precipitate does not occur until all the chloride of sodium is converted into chloride of mercury* (*sublimate*) *and nitrate of soda,* the solution *remaining clear.* If the exact point be overstepped, the excess of mercury immediately produces the precipitate above described, so that the *urea* present acts as an indicator of the end of the process. It is therefore easy to ascertain the proportion of chlorides in any given sample of urine by this method, if the strength of the mercurial solution is known; since 1 eq. of *oxide of mercury* converts 1 eq. of *chloride of sodium* into 1 eq. each of *corrosive sublimate* and *nitrate of soda*" (SUTTON). The steps of the process are as follow:—

(1.) Take 40 cc. of urine; (2.) mix with 20 cc. of the baryta-solution; (3.) pour the thick mixture upon a small dry filter; and when sufficient clear liquid has passed through, (4.) take 15 cc. of it (= 10 cc. of urine), and just neutralise it, or render it acid by a drop or two of nitric acid; (5.) bring this urine fluid under the burette which contains the test-solution of the nitrate of mercury, which is to be allowed to drop gradually, drop by drop, into the beaker containing the urine, which is to be constantly stirred with a glass rod; (6.) as soon as the precipitate does not disappear by stirring, the operation is finished, but a permanent precipitate is produced (= urea and oxide of mercury); (7.) the volume of the test-solution used is to be read off the burette, and the amount of *chloride of sodium* calculated therefrom; (8.) the chlorine may be estimated by the following formula:—As 58·8 eqs. of chloride of sodium contain 35·5 eqs. of chlorine, the chlorine in the urine is obtained by the equation,—

58·8 : 35·5 : : amount of chloride of sodium in the urine : *x* (the chlorine it contains).

The average amount of chlorine excreted in twenty-four hours is about 8·21 grammes, or 126·76 grains; 13·6 grammes, or 210 grains of chloride of sodium, were the chloride always united with that substance. Vogel and Parkes consider the mean to be 7 grammes, or 108 grains = 11·5 grammes, or 177 grains of chloride of sodium in twenty-four hours; the range above and below the mean being from 30 to 60 per cent.

In relation to body-weight, the averages of chlorine are as represented in the second line of the table at p. 936, paragraph (6).

Pathological Relations.—The chlorine contained in the urine is wholly derived from the food; and a part, chiefly united with *sodium,* passes out of the system without having entered into the composition of the tissues; another portion, uniting with the tissues, is only set free on the disintegration of these tissues; and therefore whatever exceeds the amount taken in by the food is derived from and represents change of tissue. In *ague* its elimination is increased during the cold and hot stages. In *acute pneumonia* its elimination is greatly lessened, often entirely absent, but re-appearing in the urine eight or ten days after resolution has set in and excess of urea has passed away. It evidently accumulates in the inflamed portion of the lungs. The amount is also reduced in *typhus and typhoid fever, febricula, scarlatina, erysipelas, puerperal fever, pleurisy, acute capillary bronchitis, acute pulmonary phthisis, acute rheumatism, cholera, acute and chronic Bright's disease.*

2. **The Estimation of Urea** in urine is based on the combination which forms between *urea* and *oxide of mercury in neutral or alkaline solutions.* The method was devised by Liebig. The precipitate is insoluble in water, or in weak alkaline solutions. The standard test-solutions are the same as in the preceding estimate; and the indicator which shows when all the urea has entered into combination with the mercury, and when the latter predominates, is a solution of *carbonate of soda.*

(1.) Take one volume of the baryta-solution (20 cc.), and mix with two volumes of urine (40 cc.);* (2.) after filtration, take 15 cc. of the fluid (= 10 cc. of urine) for each analysis, in small beakers; (3.) bring the beaker under the burette containing the mercurial solution, which is to be added in small quantities so long as a distinct precipitate is seen to form, the mixture being stirred constantly; (4.) a plate of glass or porcelain is now to be sprinkled with a few drops of solution of carbonate of soda, and a drop of the mixture brought from time to time in contact with the drops of soda-solution by means of a glass rod. So long as the mixture of the two drops thus brought in contact remains white, free urea is still present in the mixture, and more of the test-solution must be added to the urine, till the contact of the drops with the soda-solution produces a yellow colour, which is distinctly apparent. (5.) Record the quantity of mercurial test-solution used, and so calculate for the amount of urea contained in the 10 cc. of urine, and hence in the total discharge for twenty-four hours; (6.) repeat the analysis at least twice.

Another method has been recommended by Dr. Edmund W. Davy (*Phil. Mag.*, Vol. VII., fourth series, p. 385). The process is based on the fact that *urea* is readily decomposed by *hypochlorite of soda,* when the *nitrogen* being evolved as a gas, the amount of urea is estimated from the amount of nitrogen gas produced by the decomposition. A strong glass tube is required, about fourteen inches long, closed at one end, and its open extremity ground smooth. The bore of the tube must not be larger than the thumb can conveniently cover—*i.e.*, half an inch in diameter. It ought to be graduated into cubic inches, commencing from the closed end, and each cubic inch again sub-divided into 10ths and 100ths. The following are the details of the process :—

(1.) Fill the tube more than one-third full of fluid mercury; (2.) pour in carefully half a fluid drachm to one drachm of urine; (3.) holding the tube in one hand near its open extremity, and having the thumb in readiness to cover the aperture, quickly fill it completely full, with a solution of hypochlorite of soda (taking care not to overflow the tube), and then instantly cover the opening tight with the thumb; (4.) rapidly invert the tube once or twice to mix its contents; and, (5.) finally open the tube under mercury contained in a strong cup or small mortar. (6.)

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* The precipitate may be reserved for determining the phosphoric acid.

The tube is left in the upright position till the evolution of gas ceases, which it generally does in from three to four hours. (7.) Record the amount of gas found, and estimate the urea by the following data:—1·549 cubic inch of gas represents one grain of urea. The hypochlorite of soda used should always be five or six times the volume of the urine operated upon; and the *liquor sodæ chlorinatæ* should be perfectly pure, prepared according to the process of the Dublin Pharmacopœia, as the *liquor* of commerce always gives erroneous and exaggerated results.

A third method for determining the urea consists in the use of the following table of specific gravities from 1,003 to 1,028 (already printed in Vol. I., and repeated at pages 940 and 941 for convenience of reference), which is founded on many observations of urine, both of health and disease. The Rev. Samuel Haughton devised this table, and its results are approximations to the daily excretion of urea in all cases in which sugar is absent, and albumen either absent or only present in small quantities (*Medical Times*, Oct. 22, 1864). It is a table of double entry, to be used by finding the daily excretion of urine in fluid ounces, and its specific gravity, by means of a carefully graduated urinometer. When these data are found, at the intersection of the corresponding columns the excretion of urea is given in grains.*

The mean amount of urea excreted by adult males between twenty and forty years of age during the twenty-four hours is 33·18 grammes, = 512·4 grains. If the mode of life be equable, the amount remains pretty constant; but the maximum and minimum amounts passed on any one day by an individual are usually about *one-fifth* above or below the mean amount. In relation to body-weight, the average excretion of *urea* is as stated in the first line of the table at p. 936, paragraph (6).

Pathological Relations.—All the nitrogenous tissues contribute, by their disintegration, to the formation of urea; and the urea excreted may be taken as a measure of the extent of tissue-change or waste. In all *pyrexial states* its formation is increased; but its elimination may be increased or diminished (see chapter on "Fever," Vol. I.) When freely eliminated by

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* My friend, Dr. De Chaumont, Professor of Hygiene in the Army Medical School, has compared the trustworthiness of these processes in twenty-six cases (mostly healthy, and none either albuminous or diabetic), with the following results:—"By Liebig's process the actual average gave 25·78 parts in 1,000. The average of nineteen of these taken from Haughton's table (the remaining seven being of specific gravity above 1,028, the limit of Haughton's table), gave 28·17 parts in 1,000. The average of the whole twenty-six, deduced from Haughton's second formula (viz., *excess of sp. gr. over* 1,000 × 10 = grs. in a pint) gave 32·81 parts in 1,000.

The average obtained by the formula A:—*Excess of sp. gr. over* 1,000 × 10 = parts in 1,000, gave 28·71 parts in 1,000.

The average obtained by the formula B:—*Excess of sp. gr. over* 1,000, *after elimination of the sp. gr. due to chlorides*, ÷ ·5 (= × 2) = parts in 1,000, gave 26·200 per 1,000.

The average obtained by the formula C:—*Excess of sp. gr. over* 1,000, *after elimination of sp. gr. due to chlorides*, ÷ ·52 = parts in 1,000, gave 25·65 parts in 1,000.

Tabulating these we have:—

True Average,	= 25·78	*error.*
Haughton's Table,	= 28·17	+ 9·2 *per cent.*
Haughton's Second Formula,	= 32·81	+ 23·4 ,,
Dr. De Chaumont's Formula A,	= 28·71	+ 11·3 ,,
Dr. De Chaumont's Formula B,	= 26·203	+ 1·6 ,,
Dr. De Chaumont's Formula C,	= 25·650	− ·5 ,,

Fluid Ounces.	SPECIFIC GRAVITY.																									
	1003	1004	1005	1006	1007	1008	1009	1010	1011	1012	1013	1014	1015	1016	1017	1018	1019	1020	1021	1022	1023	1024	1025	1026	1027	1028
20	35	36	43	57	71	85	100	103	106	119	130	136	142	151	160	196	233	241	249	257	265	274	276	278	279	280
21	37	38	45	59	74	89	105	108	111	124	136	142	149	158	168	205	245	253	261	269	278	288	290	292	292	294
22	38	40	47	62	78	95	110	113	116	130	143	149	156	166	176	215	257	265	274	282	292	301	303	305	306	308
23	40	41	49	65	81	97	115	118	121	136	149	156	163	173	184	225	268	277	286	295	305	315	317	319	320	322
24	42	43	51	68	85	101	120	123	127	142	156	163	170	181	192	235	280	289	299	308	319	329	331	333	334	336
25	43	45	53	71	88	106	125	129	132	147	162	170	177	188	200	245	291	301	311	321	332	342	345	347	348	350
26	45	47	55	73	92	110	130	134	137	153	169	176	184	196	208	254	303	313	324	334	346	356	359	360	362	364
27	47	49	67	76	95	114	135	139	143	159	175	183	191	213	216	264	314	325	336	347	359	369	372	374	376	378
28	48	50	59	79	99	118	140	144	148	165	182	190	198	221	224	274	326	337	349	360	372	383	386	388	390	392
29	50	52	61	82	103	122	145	149	153	171	188	197	205	228	232	284	337	349	361	373	386	397	400	402	404	406
30	52	54	64	85	106	127	150	155	159	177	195	204	213	226	240	294	349	361	374	386	399	411	414	416	418	420
31	54	55	66	87	109	131	155	160	164	182	201	210	220	233	248	303	361	373	386	398	412	425	428	429	432	434
32	55	57	68	90	113	135	160	165	169	188	208	217	227	241	256	313	373	385	398	411	425	438	442	443	446	448
33	57	59	70	93	116	140	165	170	175	194	214	224	234	249	264	323	384	397	411	424	438	452	455	457	460	462
34	58	61	72	96	120	144	170	175	180	200	221	231	241	256	272	333	396	409	423	437	451	466	469	471	474	476
35	60	63	74	99	124	148	175	180	185	206	227	238	248	264	280	343	407	421	436	450	464	479	483	485	488	490
36	61	64	76	102	127	153	180	185	191	212	234	244	255	271	288	352	419	433	448	462	477	493	497	499	502	504
37	63	66	78	105	130	157	185	190	196	218	240	251	262	279	296	362	430	445	461	475	490	507	510	513	516	518
38	65	68	80	108	134	161	190	195	201	224	247	258	269	286	304	372	442	457	473	488	503	520	524	527	530	532
39	67	70	82	111	138	166	195	200	206	230	253	265	276	294	312	382	453	469	486	501	516	534	538	541	544	546
40	69	72	85	114	142	170	200	206	212	236	260	272	284	302	320	392	465	482	498	514	530	548	552	555	558	560
41	71	73	87	116	145	174	205	211	217	241	266	278	291	309	328	401	477	494	510	527	543	562	566	568	571	574
42	74	75	89	119	148	178	210	216	222	247	273	285	298	317	336	411	489	506	523	540	557	575	580	582	585	588
43	75	77	91	122	152	182	215	221	228	253	279	292	305	324	344	421	500	518	535	553	570	589	593	596	599	602
44	76	79	93	125	156	186	220	226	233	259	286	299	312	332	352	431	512	530	548	566	584	603	607	610	613	616
45	78	81	95	128	160	191	225	231	238	265	292	306	319	339	360	441	523	542	561	579	597	616	621	624	627	630
46	80	82	97	130	163	195	230	236	243	271	299	312	326	347	368	450	535	554	573	592	611	630	635	638	641	644
47	82	84	99	133	166	199	235	241	249	277	305	319	333	355	376	460	546	566	586	605	624	644	648	652	655	658
48	84	86	101	136	170	203	240	246	254	283	312	326	340	362	384	470	558	578	598	618	637	657	662	666	669	672

49	85	83	103	139	174	207	245	251	259	289	318	333	347	370	392	480	569	590	611	631	651	671	676	680	683	686
50	87	90	106	142	178	212	250	257	265	295	325	340	355	377	400	490	581	602	623	644	665	685	690	694	697	700
51	88	92	108	144	181	216	255	262	270	301	331	346	362	385	408	499	593	614	635	656	678	699	704	708	710	714
52	90	94	110	147	185	220	260	267	276	307	338	353	369	393	416	509	605	626	648	669	692	712	718	721	724	728
53	92	96	112	150	188	225	265	272	281	313	344	360	376	400	424	519	616	638	660	682	705	726	731	735	738	742
54	94	98	114	153	192	229	270	277	286	319	351	367	383	408	432	529	628	650	673	695	718	740	745	749	752	756
55	95	99	117	156	195	233	275	283	292	325	358	374	390	415	440	539	639	662	685	708	732	753	759	763	766	770
56	96	100	119	159	199	238	280	288	297	331	364	380	397	423	448	548	651	674	698	720	745	767	772	776	780	784
57	98	102	121	162	202	242	285	293	303	337	371	387	404	430	456	558	662	686	710	733	758	781	776	790	794	798
58	100	104	123	165	206	246	290	298	308	343	377	394	411	438	464	568	674	698	723	746	772	794	800	804	808	812
59	102	106	125	168	209	251	295	303	314	349	384	401	418	445	472	578	685	710	735	759	785	808	814	818	822	836
60	104	108	128	171	213	255	300	309	319	355	391	408	426	453	480	588	697	722	748	772	798	822	828	832	836	840
61	106	109	130	173	216	259	305	314	324	360	397	414	433	460	488	597	708	734	760	784	811	836	842	845	850	854
62	108	110	132	176	220	263	310	319	329	366	404	421	440	468	496	607	719	746	772	797	824	849	856	859	864	868
63	109	112	134	179	223	267	315	324	335	372	410	428	447	475	504	617	730	758	785	810	838	863	869	873	878	882
64	110	114	136	182	227	271	320	329	340	378	417	435	454	483	512	627	742	770	797	823	851	877	883	887	892	896
65	112	116	138	185	230	276	325	335	345	384	423	442	461	490	520	637	754	782	810	836	864	890	897	901	906	910
66	114	118	140	187	234	280	330	340	351	390	431	448	468	498	528	646	766	794	822	849	877	904	911	915	920	924
67	115	120	142	190	237	284	335	345	356	396	437	455	475	505	536	656	778	806	835	862	891	918	925	929	934	938
68	116	122	144	193	240	288	340	350	361	402	443	462	482	513	544	666	790	818	847	875	904	931	939	943	948	952
69	118	124	146	196	244	292	345	355	367	408	449	469	489	520	552	676	802	830	860	888	917	945	953	957	962	966
70	120	126	149	199	248	297	350	361	372	414	456	476	497	528	560	686	814	843	872	901	930	959	966	971	976	980
71	121	127	151	201	251	301	355	366	377	419	462	482	504	536	568	695	826	855	884	913	943	973	980	984	990	994
72	122	128	153	204	255	305	360	371	382	425	469	489	511	544	576	705	838	867	896	926	956	986	994	998	1004	1008
73	124	130	155	207	258	310	365	376	388	431	475	496	518	551	584	715	849	879	909	939	969	1000	1007	1012	1018	1022
74	126	132	157	210	262	314	370	381	393	437	482	503	525	558	592	725	861	891	921	951	982	1014	1021	1026	1032	1036
75	128	134	159	213	266	318	375	386	398	443	488	510	532	566	600	735	872	903	934	964	995	1027	1035	1040	1046	1050
76	130	136	161	216	269	323	380	391	404	449	495	516	539	573	608	745	884	915	946	977	1008	1041	1049	1054	1060	1064
77	132	138	163	219	273	327	385	396	409	455	501	523	546	581	616	755	895	927	959	989	1021	1055	1062	1068	1074	1078
78	134	140	165	222	276	331	390	401	414	461	508	530	553	588	624	765	907	939	971	1002	1034	1068	1076	1082	1088	1092
79	136	142	167	225	280	336	395	406	420	467	514	537	560	596	632	775	918	941	984	1015	1047	1082	1090	1096	1102	1106
80	139	144	170	228	284	340	400	412	425	473	521	544	568	604	640	785	930	964	996	1028	1060	1096	1104	1110	1116	1120
	1003	1004	1005	1006	1007	1008	1009	1010	1011	1012	1013	1014	1015	1016	1017	1018	1019	1020	1021	1022	1023	1024	1025	1026	1027	1028

the urine, its amount stands in close relationship with the exaltation of temperature and intensity of the fever. Retention of urea is a very unfavourable circumstance. No retention occurs in *acute yellow atrophy of the liver*, in *epilepsy*, or in *jaundice*. There are some cases, which stand alone, in which the excretion and elimination of urea are augmented, which were originally noticed by Prout, and which Willis subsequently described as cases of "*azoturia*." They have recently been noticed in an excellent paper by Dr. Sieveking in the pages of the *British Medical Journal* for May, 1865. They are characterised by a great excess of urea in the urine, without increase in the quantity of the urine, and apparently without febrile reaction, but with languor, weakness, and nervousness, associated sometimes with dyspepsia, intemperance, mental anxiety, or sexual excess. Such cases are apt to pass into *diabetes mellitus* (PROUT).

3. **Estimation of Sulphuric Acid** requires the *chloride of barium* test-solution and Beale's filter. The details of the process are—

(1.) Measure off 100 cc. of urine into a Florence flask; (2.) add a little hydrochloric acid (twenty or thirty drops); (3.) apply heat through a sand-bath till the acidulated urine boils; (4.) allow the chloride of barium test to flow very gradually into the urine; (5.) remove the heat, and allow the precipitate to subside after each addition; (6.) continue adding the test till the precipitation is complete; (7.) use Beale's filter for ascertaining the end of the precipitation. When no precipitate is formed either by chloride of barium, or by sulphate of potassa or soda, the analysis is complete.

The mean of *sulphuric acid* excreted in twenty-four hours is 2·012 grammes = 31·11 grains, with a range of 45 per cent. above or below. In relation to body-weight, the average excretion of sulphuric acid is as stated in the third line of the table at p. 936, paragraph (6).

Pathological Relations.—Sulphuric acid originates in the urine from various articles of food and drink, in which it exists ready formed: from the sulphur of the food, by oxidation in its passage through the system; from oxidation of the sulphur of the tissues; or from the oxidation of sulphur contained in substances such as *taurine* and *cystine*. Its elimination is increased in *febricula, typhoid fever, typhus, variola, pyæmia, milk fever, delirium tremens, acute pneumonia, rheumatic fever, chorea.*

4. **Estimation of Phosphoric Acid** is based on the fact, that when nitrate or acetate of uranium is added to a solution of tribasic phosphoric acid, containing acetate of ammonia and free acetic acid, the whole of the phosphoric acid is thrown down as double phosphate of uranium and ammonia, having a light-brown colour, and a composition represented by the formula $2(Ur_2O_3), NH_4O, PO_5 + Aq$. This volumetric method for the estimation of phosphoric acid was devised by Mr. Francis Sutton, of Norwich, independently of Neubauer and Pincus, who, independently of each other, also arrived at the same process; but Mr. Sutton states that Neubauer was the earliest discoverer of the method. The standard test-solution required is the nitrate of uranium, of which 1 cc. represents 0·1 grain of phosphoric acid. A solution of ferrocyanide of potassium (yellow prussiate of potash), in the proportion of 1 to 20, is to be used as

an indicator to stop the process, so soon as the reaction is complete. The details of the process are—

(1.) Take the precipitate produced by the baryta-solution in 40 cc. of urine, as mentioned at page 936, paragraph (6), having been set aside after the fluid is filtered from it. (2.) Wash the precipitate once with cold water. (3.) Treat it, while still on the filter, with warm acetic acid, to dissolve all the phosphate of baryta, which passes through the filter, leaving the sulphate behind. (4.) Wash the filter with a small quantity of boiling water, so as to remove the last traces of phosphate. (5.) Add sufficient ammonia to the solution to neutralise the acetic acid, unless the quantity of the latter be large, when somewhat less than enough to neutralise may be added. Under any circumstances the liquid must be fully acidified with acetic acid before being tested as to its strength (*titrated*), and must contain a tolerable quantity of acetate of ammonia. (6.) Take a measured quantity of the urinary phosphate solution (say 20 cc.) in a beaker, and gently warm it, and bring it under the burette containing the uranium-solution. (7.) Portions of the uranium-solution are to be delivered in and constantly stirred, until, when a drop is taken out with a thin glass rod, and placed in the middle of a large drop of the solution of yellow prussiate of potash on a white plate, a faint but distinct chocolate-brown colour is produced at the point of contact. A slight excess of uranium-solution is required to produce the brown colour, which indicates that all the phosphoric acid contained in the amount of urine in the beaker has been thrown down. (8.) Record the amount of uranium-solution used, and estimate accordingly—1 cc. of the solution precipitating 0·1 grain of phosphoric acid.* *The mean amount of phosphoric acid* excreted in twenty-four hours is 3·164 grammes = 48·80 grains, with a range in the same person of from 35 to 50 per cent. The amount in relation to body-weight is given in the fourth line of the table at page 936, paragraph (6).

Pathological Relations.—Its elimination in excess represents tissue-change or waste beyond what the food or drink accounts for. In rickets and softening of the bones the phosphates are increased. In other diseases our information respecting phosphoric acid is very uncertain. In alkaline urine the *earthy* phosphates are deposited as a white sediment. *Mineral acids, anodynes, alkaline bicarbonates, opium,* and preparations of *iron, zinc,* and *strychnia* are of service, variously combined, according to the nature of the case.

5. **The Estimation of Uric Acid.**—An improved method of estimating the *uric acid* by *iodine* has been devised by my friend Professor De Chaumont, and published by him in the *Medical Report of the Army Medical Department* for 1862 (published in 1864). The determination of *uric acid* by *iodine* is founded on the fact that solution of iodine is lecolorised by uric acid, in a definite proportion—which appears to be our equivalents to one of uric acid. Professor De Chaumont has

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* Collect all the precipitates in a large bottle, and when sufficient has been obtained, ecover the uranium by igniting the dry precipitate in a porcelain crucible, with the arbonaceous residue produced by burning tartrate of soda and potash in a covered rucible. The uranium is thus reduced to protoxide, while the phosphoric acid unites ith potash and soda, and can be entirely extracted with boiling water. The prooxide of uranium left may be dissolved in nitric acid, and evaporated to dryness in a ater-bath (SUTTON, *Volumetric Analysis*, p. 208).

kindly given me the following account of his method:—*The Solutions* required are—(1.) A standard solution of iodine. The alcoholic tincture being liable to change, it is best to dissolve the iodine in water with the aid of iodide of potassium; thus, 6·35 grammes (= $\frac{1}{20}$ of an equivalent) of iodine, with 12 grammes of iodide of potassium, are dissolved in a litre of distilled water. Of this solution, 1 cc. contains ·00635 of iodine, and will decompose ·0021 of uric acid. If the materials be accurately weighed and measured, it will be unnecessary to graduate the solution; but should this be desired, then the following solutions must be prepared:—(2.) Solution of uric acid, ·168 gramme is to be dissolved in a litre of water. This is best made by dissolving the uric acid in a small quantity of *liquor potassæ,* and neutralising the excess with dilute acetic acid. Of this solution, 12·5 cc. will completely decolorise 1 cc. of the solution of iodine. (3.) A solution of starch, carefully filtered and free from suspended grains.

The Process.—(1.) Filter the urine from mucus. (2.) Acidify the urine, should it be alkaline, with acetic acid. (3.) Put 10 cc. into a beaker glass, and dilute it to 50 cc. with distilled water. (4.) Add 5-10 cc. of starch liquor. (5.) Drop in the solution of iodine from a burette graduated to $\frac{1}{10}$ cc. (6.) Stir each time, and wait till blue colour disappears. (7.) Do not add more than $\frac{1}{10}$ cc. at a time. (8.) When the blue colour has remained permanent for *an hour,* read off the number of cc. of iodine used, and multiply by 21, which will give the quantity per litre.

Example.—10 cc. of urine took 3·5 cc. of iodine,
3·5 × ·21 = ·735 gramme per litre.

For a more accurate analysis, it is necessary to wait for twenty-four hours until all the uric acid is decomposed, taking care to add fresh portions of starch from time to time as it becomes converted into dextrine. A correction is then applied as follows:—(1.) Precipitate the salts with Liebig's baryta-solution (see process for urea). (2.) Filter and acidify with acetic acid. (3.) Test with iodine as before. (4.) Deduct th number of cc. used after the baryta from the gross amount first used and from the remainder calculate the uric acid as before.

Example.—10 cc. of urine took 3·5 cc. of iodine.
10 cc. so precipitated, filtered, and re-acidified, took ·45 cc. of iodine
Then 3·5 − ·45 = 3·05 cc. of iodine.
3·05 × ·21 = ·6405 gramme in a litre—the net result.

The mean amount of uric acid excreted in twenty-four hours appear to be ·555 gramme = 8·569 grains. The observations to determin the average in any individual must extend over at least *five days.* Th range between the maximum and minimum amount is as great as fron 20 to 30 per cent. (Parkes).

Pathological Relations.—The elimination of *uric acid* indicates meta morphoses of tissue. It increases after full, heavy, indigestible meals and exists not only in the spleen in health, but in still larger quantity i most of the affections, including *ague,* in which the spleen is mor especially implicated. It is increased in most of the active febrile an inflammatory affections. It is increased during the paroxysm of *intermittent* fevers, also in *typhoid* and *typhus* fevers. In *small-pox* and *scarlatina* it is increased; and in *pneumonia* the increase is very large. The sediments of the *urates* and of *uric acid* are most abundant from the seventh to the thirteenth day. It is increased in most *cardiac affections*, in *hepatic cirrhosis* and *leucocythæmia;* and considerably increased in *rheumatic fever.* During attacks of *gout* it is lessened or absent in the urine, and present in the blood; but as soon as fever and paroxysms abate, precipitates of *urates* and of *uric acid* become abundant. Its free elimination is a favourable symptom. In the majority of chronic affections the *uric acid* is lessened in elimination. When the excess in the urine is due to indulgence in animal food, its mal-assimilation and defective cutaneous excretion, the amount of animal food must be reduced, the diet regulated carefully, and the functions of the digestive organs improved and strengthened. When *lactic*, *acetic*, or *butyric* acids occur as products of faulty digestion, all food known to produce such a result must be avoided. The vegetable bitters—*e.g.*, *cinchona*, *gentian*, *calumba*, *serpentaria*, with or without *rhubarb* and *soda*, and followed by such remedies as keep the colour free, such as *compound galbanum pill*, with *extract of colocynth* and *croton oil*, will be found of great service. The action of the skin must be promoted by warm clothing. Exercise in the open air must be indulged in as much as possible; while cold baths in the morning, and friction with horse-hair gloves, aid the action of the skin. The best solvent is obtained by drinking freely of soft or distilled water; but alkaline remedies, like the carbonates and bicarbonates, often give relief, as well as the salts of the vegetable acids, the *acetates*, *citrates*, and *bitartrates*, given in sufficient doses. The occurrence of *uric acid* in excess in the diseases mentioned must be managed according to the nature of the treatment of each disease.

6. **Estimation of Diabetic Sugar.**—The principle of the process is thus explained by Mr. Sutton. It is based on the fact that although a mixture of pure sulphate of copper, tartrate of potash, and caustic soda, mixed in proper proportions, may be boiled without undergoing change; yet, if only a trace of sugar be added, a very slight warming is enough to precipitate a portion of the copper as a protoxide (Cu_2O). It is found that one atom of pure sugar = 180, is capable of reducing exactly 10 atoms, = 307 of oxide of copper (CuO), to the state of protoxide. Therefore, if the quantity of copper reduced by a given solution of sugar is known, it is easy to find the quantity of sugar present (*Volumetric Analysis*, p. 210). A *standard solution of pure sulphate of copper*, with *tartrate of potash* and *caustic soda*, is required. It is to be prepared as follows:—

(1.) 34·64 grammes = 346·4 grains, of pure sulphate of copper, previously powdered and pressed between blotting-paper, are weighed and dissolved in 200 cc. of distilled water; (2.) in another vessel 173 grammes = 1,730 grains, of pure crystallised tartrate of soda and potash (Rochelle salt) are dissolved in 480 cc. of solution of pure caustic soda (specific gravity, 1·14); (3.) the two solutions are then to be mixed, and the deep clear blue solution diluted with distilled water till the whole measures 1,000 cc. *One* cc. of the solution so prepared represents 0·05 grain of grape or diabetic sugar. It must be preserved for use in a dark place, and in well-stoppered bottles kept full. It should bear heating when diluted with about four or five times its quantity of distilled water, without any precipitate taking place, and should always be submitted to this test before being used. If any precipitate does occur, it probably arises from the alkali having absorbed carbonic acid; and in this case the addition of a little fresh caustic soda-solution remedies the evil (SUTTON, l. c., pp. 211 and 233).

The following are the details of the process for analysis:—

(1.) 10 cc. of clear urine are diluted by means of a measuring flask to 200 cc. with water, and a large burette filled with the fluid; (2.) 10 cc. of the copper solution ($= \frac{1}{2}$ grain of sugar) are then measured into a flask, or white porcelain capsule, and 40 cc. of distilled water added; (3.) the vessel is to be arranged over a spirit-lamp under the burette, and brought to boiling; (4.) the diluted urine is then delivered in cautiously from the burette until the last traces of blue colour are removed from the copper solution, and the precipitate is of a distinct red colour; (5.) it must be remembered that the urine has been diluted twenty times; so that the quantity used, divided by twenty, will represent the amount of the original urine used; and the estimate is to be made accordingly.*

7. **Estimation of Free Acid** is measured by a solution of carbonate of soda containing 530 grains in the 10,000 grain measure = 53 grammes in the litre; and is represented by determining how many grains of crystallised oxalic acid a certain quantity of the soda-solution will neutralise. The details of the process are as follow:—

(1.) Take 50 or 100 cc. of perfectly fresh urine; (2.) add from a burette a standard solution of soda, in small portions at a time (say 5 cc., or drop by drop); (3.) after every addition test the fluid by moistening a thin glass rod or feather with the mixture, and streak it across some well-prepared violet litmus paper; when the streaks cease to become red, the analysis is complete; (4.) estimate how much of the standard solution has been used, and express the acidity as equal to so many grains of crystallised oxalic acid.

8. **Estimation of the Total Solid Matter.**—(1.) Measure 5 cc. into a shallow platinum or porcelain capsule; (2.) place it beside a vessel of strong sulphuric acid under the receiver of a powerful air-pump, and keep it *in vacuo* till all moisture is removed.

9. **Estimation of Total Saline Matter.**—(1.) Measure 10 cc. into a small porcelain crucible; (2.) evaporate to dryness; (3.) add about ten drops of nitric acid; (4.) heat the crucible to dull redness; (5.) suffer it to cool, and add ten more drops of nitric acid; (6.) heat it up again gradually to a moderately strong heat, until all the carbon is destroyed, and the residue is white; (7.) let it cool, and weigh.

10. **The Specific Gravity of Urine** is best taken by measuring 100 cc. into a beaker or flask whose weight is accurately known. The increase of weight in grains will be the specific gravity, water being 1,000. Instead of 100 cc., 50 or 25 cc. may be taken, when the weight, multi-

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* Haughton recommends the following formula:—"Multiply the excess of specific gravity over 1,000 by 20,—the result is grains of sugar in a pint of urine." The average of 234 determinations by Dr. De Chaumont gave 61·06 parts in 1,000; the average by Haughton's formula gave 77·65,—error, + 27· per cent. Dr. De Chaumont proposes to divide excess of specific gravity over 1,000 by ·54—result, = parts in 1,000. Haughton's factor, = ÷ ·4348, or × 2·3. Dr. De Chaumont's factor, = ÷ ·54 or × 1·89.

plied by 2 or 4, will be the specific gravity (SUTTON). The urinometer gives the least accurate of all results.

For further details regarding the processes of volumetric analysis the reader is referred to the excellent treatise of Mr. Francis Sutton.

SECTION IV.—THE MICROSCOPIC EXAMINATION OF THE URINE, AND THE PATHOLOGICAL RELATIONS OF THE DEPOSITS.

In perfectly healthy urine there ought to be no sediment whatever, unless it be the merest haze of mucus, or the slightest precipitate of urates caused by a low temperature. Even these may be abnormal (PARKES). *Urinary sediments*, being precipitated in an amorphous state are termed *sand*; in a crystallised state, *gravel*; and when concreted into masses, *stone* or *calculus*. Besides this excess of the natural constituents, there are some other precipitable substances occasionally found in the urine, which are entirely new or morbid formations, as the *oxalate of lime*, and the *xanthic* and *cystic oxides*, substances, although soluble, perhaps, in certain proportions in healthy urine, yet become deposited, and form urinary sediments, which concrete into calculi. The *urinary sediments* have been broadly arranged by Dr. Parkes into the following three classes (l. c.) :—

CLASS I.—SUBSTANCES SUSPENDED IN THE URINE WHICH HAVE NEVER BEEN DISSOLVED.

The most important sediments belong to this class, and consist chiefly of organic bodies derived from the structures composing the urinary organs, or of the *productive* effects of disease upon the kidney, such as *inflammation, tubercle, cancer.* They commence to precipitate as soon as the urine is passed. They often afford the only signs of kidney disease, or of the implication of the kidney in some general affection. They are made up of the following substances :—

1. *Mucus and Epithelium from the Urinary Passages.*—In many diseases the quantity of epithelium from the *bladder* is increased, indicating catarrhal inflammation of its lining membrane. The epithelial cells are of various sizes and stages of formation (Fig. 59); and frequently free nuclei are seen. In catarrh of the bladder the mucus, from its cohesion, is apt

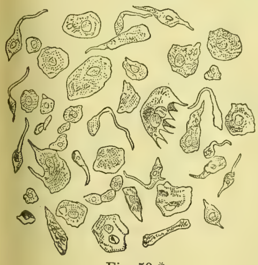
Fig. 59.*

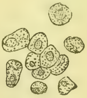
Fig 60. †

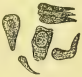
Fig 61. ‡

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* Epithelium from the bladder. Many of the large cells lie upon the summit of the columnar and caudate cells, and depressions are seen on their under surface. One is seen near the centre of the figure (after Dr. BEALE, *On the Urine*, p. 259).

† Epithelium from the pelvis of the kidney (after Dr. BEALE, l. c., p. 188).

‡ Epithelium from the ureter (after Dr. BEALE, l. c.).

to form thin transparent flakes or cylinders, resembling casts from the prostate or kidney. The epithelium-cells from the *pelvis of the kidney* are often triangular or caudate, with well-defined nuclei. They generally adhere together in groups of three to ten, when they appear to have an imbricated arrangement, and perhaps are more closely connected than natural by adhesive mucus (Fig. 60). They are never found in healthy urine, but are present very commonly in catarrhal and calculous cystitis. Tailed or caudate cells are also sometimes present with pelvic epithelium. The epithelium from the *ureter* is columnar in its character (Fig. 61), and not unlike that found in the male urethra, which is mostly columnar, and is more flattened than that of the bladder, and less regular than that of the pelvis. Mixed with it there is a good deal of scaly epithelium, especially towards the orifice of the urethra. Large cells of scaly epithelium are also often met with in the urine of females. They are derived from the *vagina;* and vary in size and form, sometimes very irregular in shape, and with uneven ragged edges (Fig. 62).

Renal epithelium is only found in disease. It consists of round or slightly compressed cells, or masses of material, with well-defined central portions of nuclei, which are not cleft like the pus nucleus under the action of acetic acid, but become at first more defined, and afterwards paler and smaller. In the urine they are less polygonal and more rounded than they are in the rena canals (Fig. 63). Their presence indicates desquamation from the tubes; while their morbid condition and admixture with other products may indicate still greater disease. Thus, it is sometime fatty; the whole space between the nucleus and the cell-wall being filled with fatty globules—changes and their value which will be fully considered under "Chroni Bright's Disease."

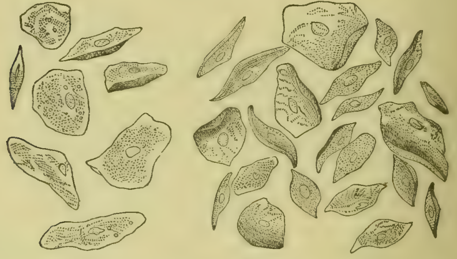

Fig. 62.*

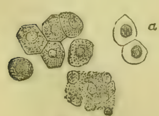

Fig. 63.†

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* Vaginal epithelium from urine (after Dr. BEALE, l. c., p. 260).

† Epithelium from convoluted portion of uriniferous tube.—(*a.*) Treated with aceti acid × 215 (after Dr. BEALE, l. c., p. 187).

2. *Other Cell-Forms occur in the Urine, the productive results of inflammatory diseases* of the mucous membrane of the urinary passages. They are chiefly granules, singly or in clusters, and pus. The pus is known by its nucleus becoming cleft into two, three, or five divisions, under the action of acetic acid. The amount of pus is generally far greater in *cystitis* than in *pyelitis* or *nephritis;* and wherever there is a markedly large quantity of pus it is generally from the bladder, unless there be a prostatic or other abscess communicating with the urethra.

3. *Cancer-Cells* occur in cancerous disease of the bladder, kidney, or urethra.

4. *Tubercle-Masses* may occur in tuberculous disease of the bladder or kidney.

5. *Cylinders* occur in the urine, and have various modes of origin:—(*a.*) From the *bladder,* as long, flat, membraniform, twisted, or folded bodies. (*b.*) From the *prostate,* as coagula, two or three times as broad as renal cylinders. They are soluble in acetic acid. Amylaceous corpuscles may exist in them. (*c.*) From the *ureter* and *pelvis* of the kidney the coagula are cylindrical, pyriform, or globular. (*d.*) From the kidney-tubes cylinders or renal casts are formed in various diseases. They vary in breadth from $\frac{1}{500}$ to $\frac{1}{1000}$ of an inch—*i.e.*, from about the breadth of the straight renal tubes to half or a third of that size. Their length varies from $\frac{1}{200}$ to $\frac{1}{50}$ of an inch. The terms used by Dr. George Johnson (to whose researches on diseases of the kidney we owe much) express very well the special character of the several varieties of casts—namely, (1.) Epithelial casts; (2.) large waxy casts; (3.) small waxy casts—the hyaline casts of Vogel and Basham; (4.) granular casts; (5.) oily casts; (6.) bloody casts; (7.) purulent casts. *Solution of iodine* is the best reagent for making the waxy hyaline or transparent casts visible. They are probably albuminoid.

6. *Kidney-Structures* may occur in the urine if the structure of the kidney has commenced to break up from any cause.

7. *Blood-corpuscles* indicate rupture of vessels somewhere in the urinary passages. Copious hæmorrhage is usually from the bladder, carcinoma of the kidney, or calculus of the pelvis. The corpuscles are often much modified in form.

8. *Fibrine* is present in many cases of bloody urine.

9. *Corpora amylacea* may occur from the prostate gland.

10. Fat. 11. A fatty substance, to which the name of urostealith has been given. 12. *Spermatozoa.* 13. *Sarcinæ.* 14. Hair. 15. *Various entozoa* have been occasionally found in the urine. (See under "Entozoa," Vol. I.)

CLASS II.—SEDIMENTS FORMING IN THE URINE AFTER SECRETION, BUT WHICH MAY DEPOSIT IN THE RENAL PASSAGES OR AFTER EMISSION, EITHER IN CONSEQUENCE OF CHEMICAL CHANGES OR FROM CHANGE OF TEMPERATURE.

(1.) Uric Acid, its Forms and Pathological Relations.—(*a.*) *Uric Acid* occurs in various combinations with bases, such as *soda, potash, lime,* or *ammonia,* and more or less coloured with urine-pigment. It may be associated with phosphate or oxalate of lime. The uric acid sediments in combination with bases are generally associated with increased acidity of the urine, either by normal acids, such as uric, sulphuric, phosphoric, hippuric, or with the formation of acids developed after emission. They are known as "urates," and commonly spoken of as yellow lateritious "brick-dust," or "fever sediments." They dissolve when the urine is heated to 130° Fahr. They are also soluble in potash and liquor ammonia. Acids decompose them after warmth, and liberate uric acid. Three forms are distinguishable—(1.) Irregularly formed or amorphous particles; (2.) round globules of various sizes; (3.) fine acicular prismatic crystals. These forms may deposit at *two* different periods after emission —(1.) As soon as the urine has cooled down to the temperature of the atmosphere. In such cases the water is diminished—the urine being that known as febrile urine; or it occurs when there is an absolute increase of uric acid. (2.) They deposit some hours after the urine has been passed, and long after the urine has been of equal temperature with the air. Under these circumstances the deposit betokens increased acidity from changes in the pigment or extractives, or uroxanthine. The acids so formed may be lactic, acetic, or butyric; and a drop of acid added to such urine will anticipate the deposit.

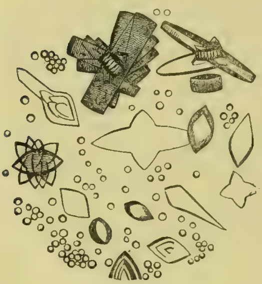
Fig. 64.*

(*b.*) *Uric Acid Sediments* in their pure state occur in the form of rhombic prisms, or rhombic plates, or of thin hexagonal plates like cystine; but the most usual forms are referable to some variety of the rhomb (Fig. 64). Such deposits often present the aspect of a granular sand of a golden lustre, sometimes mingled with blood-discs; and generally the deeper the colour of the urine the darker is the uric acid sediment. Its appearance does not necessarily indicate that an excess of uric acid is forming in the body. The urine is generally yellow and transparent, and the acid is deposited slowly without admixture of urates. Liquor potassæ, and also nitric acid in excess, dissolve uric acid.

Pathological Relations.—Lithuria, or *Lithic*, or *Uric Acid Diathesis*, are names which have been given to that general constitutional state in which the urates are secreted in such excess as to be deposited in inordinate quantities in the chamber-vessel on the urine cooling; or, when in still greater abundance, deposited in an amorphous or crystallised state, either in the cavities of the kidneys or bladder. The specific gravity of such urine varies from 1,015 to 1,035; it always gives an acid reaction, and is of a deep copper or red colour. *Uric acid deposits* not only occur when the patient is in his best health; but in many persons who have died while labouring under this constitutional state the kidney has been found healthy. On the other hand, the kidney has been found occasionally studded all over with crystals of uric acid in the tubes, especially in children. Uric acid diathesis, however, may coexist with most forms of disease of the kidneys, ureters, or bladder. It is by no means uncommon to find a calculus of this substance, the nucleus of a mulberry, or other calculus, formed in the tubuli or pelvis of the kidney, and sometimes in the bladder. The colour of *uric acid crystals* and *sediments of the urates* varies greatly. The pure *lithate of ammonia* is white; but, owing to the presence of the colouring matter in the urine, it is usually deposited of a yellow or wood colour. The *purpurates*, owing either to the nature of the colouring matter of the urine, or to other circumstances not yet determined, are of a pink, light red, or brick colour. These different salts may be deposited in a crystallised or in an amorphous state, or as small calculi. The *lithic acid* which exists in healthy urine in such a state, and in such proportions as to be held in solution at ordinary temperatures, in certain conditions of the system may be precipitated from that secretion in a *crystallised* and nearly pure state.

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* The most usual forms of uric acid sediment, with blood-corpuscles intermixed (after Dr. Otto Funke).

The *amorphous* and *impalpable lithic acid sediments* consist in general of *lithic acid* in combination with *ammonia,* and only in a very few instances with *soda.* The sedimentary deposits of the *lithates of ammonia* may be white, yellow, pink, or red; while the sedimentary deposits of the *lithates of soda* are white. The crystallised salts form gravel. The yellow amorphous sediments may concrete in the bladder or kidney, and form calculi. In general the *lithates* and *purpurates,* whether in a crystalline form or as an amorphous sediment, even when in great excess, are held in solution at the temperature of the body; but in some instances their superabundance is so great that they are deposited, even at this temperature, either within the bladder or kidney, so that the last portions of urine are so loaded with them as to resemble a stream of blood. If the excess be yet greater, or if a nucleus be present, a concretion may form either in the kidney or bladder, but more commonly in the former. The nucleus may be either a piece of hardened mucus, or a portion of fibrine or other substance, or it may be a crystal of lithic acid. The *lithates,* however, are frequently only the nucleus of a calculus of different formation, as the *phosphates* or *oxalates.* Indeed, we not unfrequently see the lithates, the phosphates, and the oxalates deposited in alternate layers in the same calculus, thus affording absolute demonstration of three or four different conditions of the constitution having prevailed during the formation of the same calculus. Persons labouring under *idiopathic uric acid diathesis* are, in other respects, generally healthy, and the remote causes are errors in diet and sedentary habits. A too full animal diet, as rich old black meats and game, are among the most frequent. When the predisposition, however, to the diathesis is great, every substance, even the most opposite, *that causes indigestion* will produce it, as a heavy dumpling or new bread, the richer sorts of fish or salted meats, acid fruits, or saccharine matters. Wines which are so-called "heavy-bodied"—*i.e.,* full of sugar and solids not yet decomposed by the vinous fermentation, and malt liquors, are still worse, from the rapidity with which they ferment and turn acid. Besides the result of errors in diet, a deposit of the *lithates* is incident to many diseases, as *gout* and *rheumatism.* The appearance of *urates* may be a critical termination and first faint indication of recovery from fever, or severe forms of inflammation. It also results from morbid states of the liver. It may result from an irritable state of the bladder, or from stone in the kidney or bladder. The effects of diet are so marked in children, that we can hardly feel surprised that any error of diet, as overfeeding them, should be followed by lithic acid deposits. Urinary calculi are consequently common in children, chiefly among those of the poorer class, in whom those errors are likely to be most considerable. When stone forms in the bladder of children the ages most affected are between four and nine years. Of 506 children operated on at the Norwich Hospital, 223 were under 12 years of age, while 271 were between 14 and 15. Two-thirds of all the cases of stone result from uric acid diathesis. After these periods the ages of 40 and upwards furnish the greater number of cases of gravel. In many instances *lithiasis* appears to be hereditary, and those attacked are usually of sthenic constitution.

Symptoms.—The fact of *lithic acid* being in excess is palpable, from the yellow, red, or pink deposit in the chamber-vessel as the urine cools. When this is moderate in quantity the patient suffers neither local nor general inconvenience. Many persons are never better than when they are passing an excess of the *lithates.* But when it is deposited as an amorphous sediment in the bladder, the last portions of urine are so loaded with it that the patient apprehends he is passing blood. He is then troubled with itching and pain in the urethra in making water; but if the disease becomes chronic the bladder becomes irritable, the urine loaded with mucus, the healthy sympathy between the bladder and prostate is destroyed, so that the urine is only passed after great forcing, and in trifling quantity, and with painful and severe suffering. The secretion of a great excess of the lithates is seldom a purely local disease, but is accompanied by more general affections, as asthma, palpitation of the heart, rheumatism, or gout; and during its continuance these diseases often, in a great measure, subside. Although the passage of an amorphous sediment (unless it be in such quantity as absolutely to obstruct the passage), is seldom productive of much local inconvenience, unless it be of long continuance, yet when the lithic acid crystallises so as to form gravel, or a still larger concretion, the expulsion of this foreign body is always attended with much pain, and gives rise to what has been termed nephritic colic (*colica nephritica*), similar to the biliary or hepatic colic which attends the passage of gall-stones. The passage of a calculus from the kidney into the bladder may be preceded by dull pains in the back, and some sickness. More commonly the attack is sudden, and the patient, perhaps, in his best health, and engaged in the ordinary transactions of life, is on the instant seized with excruciating agony in the loins, with retractation of the testicle, irritation of the bladder, and often with nausea and vomiting; but in all this suffering the pulse retains its healthy frequency, and the heat of the body is natural. At length the pain intermits, and the patient has a short interval of ease. The paroxysm however, returns more or less frequently, till the patient is relieved as by a charm, the calculus having passed into the bladder. Again, after an uncertain interval, the mass of gravel or calculus becomes impacted in the neck of the bladder. The same phenomena present themselves, till at last, after an effort to pass water, the noise of a stone falling into the chamber-vessel is heard as the sound of most pleasant music, and the gravel or calculus is found. The duration of this fit may last from one hour to many, and sometimes continues for many days. Occasionally, however, the calculus has acquired so great a magnitude that it becomes impacted in the ureters, and death has ensued from this cause. In general, calculi pass from one kidney only at a time, but sometimes they may pass simultaneously from both kidneys; and should they be large, or the passage long, an entire suppression of urine has been the consequence, and the patient may die from that cause. Sometimes the calculus so rapidly increases by sedimentary deposits, that it is detained altogether in the kidney, when it not only takes the form of the pelvis of the kidney, but branches out in every direction like a piece of ginger. In a few instances, a calculus thus formed in the kidney may lie latent and cause little inconvenience to the patient. More commonly, however, it acts as a foreign body, and the kidney becomes the seat of abscess or other disorganisation, and the patient suffers immensely with pains in the back, irritability of the bladder, aggravated by the frequent discharge of pus, of blood, or mucus. Existence under these circumstances becomes a burthen; and death at length terminates the patient's ceaseless sufferings. A calculus having passed into the bladder sometimes increases so fast that it acquires a magnitude too great to escape by the urethra; a stone in the bladder is thus formed. This condition necessarily requires an operation, which will be found treated of in the several text-books on surgery.

Diagnosis.—The red lithates can only be confounded with blood, from which, when intense in colour, they may with difficulty be distinguished. On cooling, however, the subsidence of an impalpable or gritty deposit, the presence of much mucus, the absence of fibrine, and of albumen, when the urine is treated by nitric acid, enable us readily to distinguish them. The *white* or *light-coloured lithates* are distinguished from the *phosphates* by the urine being acid, by the absence of the abundant mucous discharge which always accompanies a large deposition of the phosphates, and from the urine not becoming alkaline or fetid if kept for a few hours. The precipitated lithates also are readily dissolvable by heat, which the phosphates are not.

Prognosis.—While the deposits are sedimentary, the prognosis is always favourable, however large the quantity discharged. Even when gravel or small calculi are formed, the chances are many that they will be discharged before attaining any considerable size. Also, when retained in the bladder, nothing but a surgical operation can remove it; and consequently the chances resolve themselves into the proportionate numbers which recover or die after the operations of lithotrity or lithotomy at the age and under the circumstances of the patient.

Treatment.—The medical treatment of the lithic acid diathesis is by *alkalies* or *neutral salts, turpentine*, and saccharine matters.

The celebrated Morgagni suffered greatly from lithic acid gravel, and his remedy was half a drachm of *carbonate of potash* night and morning, gradually increasing the dose till he took three drachms during the day. "The acid of his urine soon became saturated, the pain in his loins diminished, his urine became less loaded, and potash was at length found in that fluid in excess." He repeated this remedy as often as he was threatened with an attack, and always with success. Much benefit may be derived from the *citrate of potash*, or the *common effervescing draught.* When the patient's bowels require a more active agent, the *sulphate of magnesia*, the *sulphate of soda*, or the *iodide* or *bromide of potash* may be substituted. The *pure alkalies*, from the smaller doses in which they can only be administered, are less beneficial than the *neutral salts. Magnesia*, in Mr. Brande's experiments, produced much less marked effects on the urine than either the *subcarbonate of potash* or *soda*, while *lime-water* produced no very sensible effect whatever. *Phosphate of ammonia*, in doses of ʒi. daily, largely diluted, is of service; and Dr. Garrod reports favourably of it in chronic conditions of the gouty habit. *Colchicum wine* (♏xv.) twice or thrice a day, followed by a saline aperient, such as Pullna water, or *hunjadi janos*, and from forty to fifty minims now and then in the morning, is a mode of treatment attended with advantage. Dr. Garrod also calls attention to the value of the *salts of lithia* in uric acid formation associated with gout, and to the value of the Baden-Baden and Bath mineral waters, both of which contain *lithium.* The *citrate of lithia* may be given in doses of five to ten grains, and the *carbonate* in three to six grains. Frequent use of the Turkish bath is of great service.

Besides *alkalies*, *turpentine* has some repute in the cure of the lithic acid diathesis. The celebrated Dutch drops are supposed to be principally composed of *sp. terebinthinæ* and of *tinct. opii*, coloured by some colouring matter. Dr. Henry gives two cases of the beneficial effects of this remedy. One of them, a lady, who, when threatened with an attack, always had recourse to it; and the uniform effect was the discharge of a sandy substance in such quantities that often four ounces of gravel were discharged in two or three days. *Opium* always gives relief.

The treatment of nephritic colic is similar to that for the passage of a gall-stone, as the *warm bath*, *mild purgatives*, and *opiates.* If the calculus, having escaped from the kidney, is retained in the bladder, an operation for its removal is necessary, and the case then becomes purely surgical.

Dietetic Treatment is of the greatest importance in the cure of the lithic acid diathesis. The experiments of Wollaston and Vauquelin have shown that in proportion as animals are fed on animal diet or on azoted substances, their urine becomes more and more loaded with lithic acid. While Majendie has shown by a counterproof, that if a dog be fed on non-azoted substances, as sugar, every trace of lithic acid disappears from the urine. A lady at Paris, suffering from gravel, having heard of Majendie's experiments, made trial of sugar on herself, eating more than a pound daily. She persevered in this regimen for six weeks, when her gravel disappeared. She then returned to her old regimen, and at the end of three months her fits of gravel returned. Of all diets the quantity of animal food should be reduced. It is necessary also that port as wel as French wines should be abandoned, and all those things which, according to the idiosyncrasy of the patient, are likely to produce indigestion o acidity of the stomach. The patient also should be warmly clad, ris early, and take a considerable amount of exercise.

(2.) SEDIMENTS OF HIPPURIC ACID.—Owing to the solubility of th acid its sediments are rare. When it does occur, it is in the shape of long four-sided, acuminated prisms, or acicular needles fixed on uric acid crystals, with which they are sometimes confounded, as well as with phosphates. They are distinguished from phosphates by being insoluble in acids; and from uric acid they may be separated by boiling with strong alcohol (PARKES).

(3.) PHOSPHORIC ACID, ITS FORMS AND PATHOLOGICAL RELATIONS.—(*a.*) *Sediments containing phosphoric acid* are formed of the *ammoniaco-magnesian phosphate*, the *phosphate of lime*, and the *phosphate of magnesia.* The ammoniaco-magnesian phosphate occurs in the form of beautiful transparent prisms, or in foliaceous, penniform, or stellar prisms or crystals. (*b.*) *The phosphate of lime* and the *phosphate of magnesia* form a white amorphous powder, or they occur as round small globules, or as prismatic crystals with oblique summits (HASSALL). Acids dissolve all those sediments; heat has no effect upon them. (*c.*) *The ammoniaco-magnesian phosphate* is almost always deposited as a result of the decomposition of urea, and the deposit usually commences on the surface of the fluid, where the urea is most exposed to the air. The *phosphate of lime* and the *phosphate of ammonia* are thrown down if the urine becomes alkaline from fixed alkali, as after vegetable food or the carbonates of the fixed alkali, or after the salts which form them have been taken. Decomposition of the urea in these cases is generally rapid, and *ammoniaco-magnesian* phosphate is produced. Such also is the case with urine of chronic diseases, or during convalescence from acute diseases, when the urine is feebly acid. The phosphates and urates occur together under the following conditions:—(1.) When urates having been deposited, the urea decomposes to a slight extent—enough to form the ammoniaco-phosphate, and yet not enough to dissolve the urate. (2.) When crystallised uric acid has formed and been acted upon by the ammonia formed from the urea, the crystals of uric acid disappear, and are replaced by round globules of *urate of ammonia,* mixed with the precipitate of phosphates. These deposits are always white, and unchanged by heat; soluble in dilute hydrochloric acid, but insoluble in ammonia and in liquor potassæ. Mucus, pus, or blood may mask the chemical reactions. A small quantity of solution of sesquicarbonate of ammonia, added to a large quantity of healthy urine, will yield prisms of the triple phosphate.

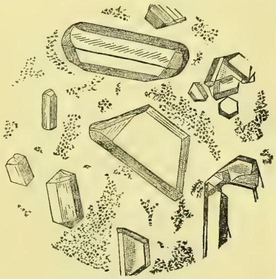
Fig. 65.*

Pathological Relations.—Ceramuria or Phosphatic Diathesis are names which have been given to that general state of the body in which phosphates tend to appear in the urine. The phosphates are secreted in a state of health in the proportion of one part in 1,000 of urine. When this proportion is abnormally increased, so that they are largely deposited either in the kidney or bladder, or even in the chamber-vessel, the condition has been termed the *phosphatic diathesis.* The remote causes of this constitutional state are, exposure to cold and wet, poor diet, blows on the back which injure the spinal marrow, and more especially diseases of the bladder, as *vesical catarrh*, *stone in the bladder*, *the introduction of a bougie*, or *other irritating causes.* This form of disease sometimes occurs in children, but more commonly in adults between thirty and forty years of age. It affects both sexes, but more commonly the male than the female. Those affected are usually of an asthenic, pale, leucophlegmatic temperament, and in some instances are believed to have inherited it. Though often resulting from disease of the bladder or kidney, yet occasionally it exists when no such disease is present. It is the prevalence of this diathesis that causes those large calculi sometimes found in the kidney or bladder. The phosphatic salts, like the super-lithates, have the property of reddening vegetable blues, and they show an acid reaction. When the earthy bases of *lime* or *magnesia* are from any cause secreted in greater abundance than natural, they combine with the phosphates, which are thrown down in the form of insoluble phosphates, and may be deposited either as a sediment on the urine cooling, or in the bladder or kidney before being passed, or, being retained in those cavities, may concrete into a stone or calculus.

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* Ammoniaco-magnesian phosphate in prisms, mixed with amorphous granules, phosphate of lime, and granular urates (after Dr. THUDICHUM).

Symptoms.—When this diathesis is a primary affection, the patient is usually of a sallow complexion, stout, but effeminate, and of great irritability of nerve. He suffers from indigestion, flatulence, constipated or disordered bowels, his stools being either black or clay-coloured. His bladder is highly irritable; he has pain in his back and loins; his urine is abundant and loaded with mucus, with a copious white sediment, so that the latter portions of urine pass like so much milk. The duration of this condition may be long, on account of the diseased state of the bladder with which it is connected. In cases, however, in which that viscus is healthy, it readily yields to medical treatment; but in some instances, when all appears to be proceeding favourably, the lungs become affected, and the patient dies of phthisis. The urine is pale, increased in quantity, often turbid, and covered with an iridescent pellicle or film consisting of a solution of the *triple phosphate of ammonia and magnesia* much *mucus* is deposited, with a copious precipitate of the phosphates, s that the urine may appear like so much chalk and water. It is generall of low specific gravity, 1,001, 1,002, or 1,003. As the phosphates hav little tendency to crystallise, a nucleus is necessary before the sedimen can concrete into calculi; and it is in this form of disease that we fin such singular substances in their centres, as a clot of blood, a piece o hardened mucus, broken ends of sounds or bougies, bits of straw, bodkin pins, plum-stones, beans, nut-shells, and bullets. *The calculi* which for on these nuclei are of three descriptions, composed of the following ingr dients:—Out of 102 calculi examined there were of phosphate of lim nearly pure, 8; of triple phosphate, or phosphate of ammonia an magnesia, 3; mixed or fusible calculi, being a mixture of the two preceding, 91.

They are distinguished from all other calculi in being soluble in an excess of *phosphoric acid.* They are distinguished from one another by the *phosphate of lime calculus* being nearly infusible, by the *mixed* being readily fusible, while the *triple phosphate* is known by the minute crystals which form between the interstices of the laminæ. Every other form of calculus, whether the lithate or oxalate, from the irritation it occasions, constantly produces a deposit of a soft coating of the phosphates. When the phosphatic deposition is once well established, it is seldom followed by the deposition of strata of any other description. Thus, of 823 calculi examined by Dr. Prout, he found only three specimens in which the phosphates had been followed or surrounded by other calculous deposits. The physical characters of the *phosphatic calculi* are, that they are white, soft, easily broken down, and deposited in concentric laminæ like the lithic acid concretions.

Diagnosis.—The *phosphatic sediments* may be distinguished from the *lithic* by the urine, though at first acid, becoming putrescent, and giving an alkaline reaction after standing a few hours. *Ammonia* also added to the urine throws down a white cloud, which consists of the phosphate of lime with some of the ammoniaco-magnesian phosphates, a test which would render the lithates soluble. The best test, however, is the addition of *phosphoric acid*, which redissolves the precipitate.

Prognosis.—When this diathesis is unaccompanied by disease of the bladder the prognosis is always favourable. When, however, it results from a morbid state of the bladder or diseased structure of the kidney, it is always of long duration.

The **Treatment** of this affection is by some mineral acid combined with an opiate. The acid may be the *nitric*, *muriatic*, the *phosphatic*, or the *sulphuric;* but the *dilute sulphuric acid* is generally preferred, as being most pleasant to the taste. A most usual prescription is the *infusion of roses*, with an addition of ♏ v. to ♏ x. of *dilute sulphuric acid*, together with *tinct. opii*, ♏ iij. to ♏ v. every six hours. This combination is generally efficient in checking the formation of phosphates, and the *sulphate of magnesia* may be added to it if the bowels should require to be regulated. *Benzoic acid* has been found of great use (gr. x. to xv.) in a mucilaginous draught. It may be given in still larger doses—twenty to forty-four grains a day (Garrod). When the phosphatic diathesis depends on vesical catarrh, or other diseased state of the bladder, *salicine* gr. x. thrice daily has occasionally been found successful. Others prefer the *inf. diosmæ*, and others *uva ursi.* The pure alkalies in these cases should not be used.

The diet should be as nourishing as the state of the diseased viscus will allow; and acid wines and ripe fruits greatly assist in effecting the cure.

(4.) Oxalate of Lime, its Forms and Pathological Relations.—Opinion is very undecided as to the significance of deposits or *sediments composed of oxalate of lime.* It occurs in one out of every three cases, taking the cases indifferently as they occur in an hospital. It appears (Fig. 66) in four forms:—(1.) As octahedra (*a*); (2.) as hour-glass contracted, or dumb-bell-like bodies (*b*); and (3.) compound octahedra (*c*) may also be seen; (4.) as small, flattened, bright discs, very readily mistaken for blood-discs. *Oxalate of lime*, although found in the blood, is probably a result of chemical changes in the renal passages. The octahedra grow or increase in urine after it is passed; but the dumb-bell crystals are said not to do so. The dumb-bell crystals of oxalate of lime are mainly formed in casts in the kidney tubes (BEALE). The oxalic acid of urine most probably results from uric acid (PARKES). It may be a substitution for the excretion of carbonic acid of the lungs (SCHMIDT). In order that oxalic acid shall form in the urine, there must either be irritation from a calculus, or from some other cause; or there must be fermentive changes from mucus. If there be no evidence of any of these conditions, the oxalic acid of fresh urine may be presumed to come from the blood (PARKES). Crystals of oxalate of lime are insoluble in water—are unaltered by boiling either in *acetic acid* or in *liquor potassæ;* and are soluble in nitric acid without effervescence.

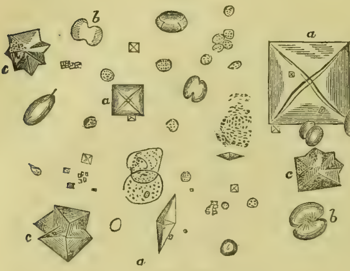

Fig. 66.*

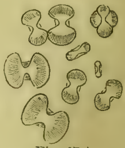

Fig. 67.†

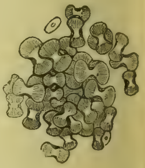

Fig. 68.‡

Pathological Relations.—Many articles of diet contain oxalic acid in abundance; but there are certain articles of food and drink the use of which, when digestion is deranged, is followed by the formation of oxalate of lime and its presence in the urine. Sugar in excess, the use of frothy sparkling beer or wine, turnips, parsnips, carrots, cauliflower, asparagus, may all cause a "temporary oxaluria" (PROUT, ROBIN, VERDEIL, ROSE, BIRD, and HASSALL). When the respiratory functions are impeded, oxalates appear in the urine, and during convalescence from some severe diseases, as *typhus fever*. *Oxaluria* is the name given to that general state of the

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[*] (*a.*) Octahedra; (*b.*) dumb-bell; (*c.*) compound octahedra; circular and oval crystals of oxalate of lime (atfer Dr. BEALE, l. c.).

[†] Dumb-bell crystals of oxalate of lime, of very regular form, from the urine of a child two years of age, suffering from jaundice (after Dr. BEALE, l. c., p. 300).

[‡] Collection of dumb-bells, such as often forms the nucleus of a calculus (after Dr. BEALE, l. c., p. 300).

constitution in which oxalate of lime is secreted, as first determined by Dr. Wollaston. The remote cause of this constitutional condition is not determined, for persons who seem in the best health may void oxalate of lime in the urine. It is supposed, however, to be most frequent among those who eat largely of common sorrel (*rumex acetosa*), or of tomata (*solanum lycopersicum*), and of the leaf-stalks of the rhubarb plant, all of which many persons are fond, and all of which contain oxalic acid. *Oxaluria* may exist before puberty, and from that period till sixty. It is most usual between forty and fifty; is common in both sexes, and is not incompatible with gout.

Oxalate of lime very rarely appears under the form of an amorphous sediment; still it has occurred mixed with the lithic amorphous sediments; but this is uncommon. Its appearance is still more rare under the form of crystallised gravel, so much so that Dr. Prout mentions only two instances. Renal calculi of this formation are not very uncommon. When detained in the bladder they may acquire a considerable size, are rugged, dark-coloured, and tuberculated, and from these appearances have been termed the "mulberry calculi." Oxalate of lime enters as a constituent part into about one-fourth of all calculi in the following order of abundance:—(1.) *Oxalate of lime;* (2.) *lithic and mulberry;* (3.) *mulberry and lithic;* (4.) *mulberry and phosphates;* (5.) *fusible and mulberry.*

When heated before the blowpipe the *oxalic acid* is decomposed, and pure lime remains, which gives a strong brown stain to moistened turmeric paper. This calculus is insoluble in the alkalies, but by digestion in carbonate of potash it is decomposed, and the insoluble carbonate of lime is left. When reduced to powder and digested in nitric or muriatic acids a perfect solution is effected. It is not dissolved by acetic acid—a circumstance which distinguishes it from the *ammoniaco-magnesian phosphate.* It is distinguished from the *phosphate of lime* by being insoluble in phosphoric acid.

Symptoms.—*Oxaluria* is attended with no prominent feature. The urine is acid, of a good colour, remarkably pure, and free from all sorts of sediment as well as gravel. The patient is therefore hardly sensible of any inconvenience till he is attacked by a fit of nephritic colic, caused by the passage of the calculus from the kidney or bladder, or till he is troubled, supposing it to be retained, by symptoms of stone in the bladder or kidney.

Treatment.—Respecting the medical treatment of *oxaluria*, Dr. Prout recommends that we should try to induce a *lithic acid diathesis;* but it must be questionable whether the disease substituted is not as dangerous as the one under which the patient originally laboured. He speaks, however, of having seen much advantage derived from *mineral* acids and the *sulphates of iron* or of *quinia.* The mineral acids are useful in correcting the dyspepsia associated with the elimination of oxalates. *Henbane* is considered by Dr. Harley as a most valuable remedy. Water containing lime should be avoided, and distilled water used instead; and in many cases it will be necessary to empty the intestines by suitable purgatives.

Dietetic Treatment.—The patient should carefully avoid eating substances containing oxalic acid.

(5.) Leucine, its Forms and Pathological Relations.—*Sediments of Leucine* are precipitated from the urine in round corpuscles, sometimes with a concentric form, and look like heaps of fat; but when crystallised from pure solutions, it appears as fine, dark-coloured needle-like crystals. It must be separated by careful sublimation (Parkes). The suspected leucine is to be placed on platinum, carefully moistened, and then dried with nitric acid. The almost imperceptible flake which is left is to be moistened with caustic soda, and evaporated carefully over a spirit-lamp. If leucine is present, it forms an oily-looking drop (Scherer).

Pathological Relations.—It is believed to be the result of the disintegration of certain of the nitrogenous tissues of the principal glands, and occurs particularly in *acute yellow atrophy* of the liver, where it is found in the substance of the liver itself.

(6.) Tyrosine, its Forms and Pathological Relations.—*Sediments of Tyrosine* are of a greenish-yellow colour, composed of heaps of fine needles, which can be obtained on evaporation. It ought to be treated with nitric acid, like *leucine*, and then a little *liquor soda* used. The nitric acid gives a deep orange-yellow colour, which becomes deep yellow on evaporation. The soda gives the yellow flake a red tinge; and on heat and evaporation, a black-brown residue is left (Scherer, quoted by Parkes).

Pathological Relations.—It is found wherever leucine is met with.

(7.) Cystine, its Forms and Pathological Relations.—*Sediments of Cystine* form a white or light fawn-colour, amorphous, rather bulky precipitate, or they appear at once as six-sided plates (Fig. 69). In both cases ammonia dissolves it,—so do fixed alkalies and their carbonates; and from this solution it crystallises on spontaneous evaporation. It does not disappear when the urine is gently warmed; and it is insoluble in carbonate of ammonia, in dilute hydrochloric acid, and in acetic acid.

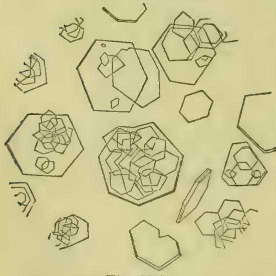
Fig. 69.*

Pathological Relations.—It is associated with the excessive elimination of *sulphur.* Its persistence in the urine may be hereditary, and is generally associated with derangement of the functions, or organic disease of the liver. It occurs also in cases of chlorosis. *Cystinuria* is the name which has been given to the general state of the system in which cystine is eliminated by the urine in abnormal amount. The cystic oxide was described by its discoverer, Dr. Wollaston, in the *Philosophical Transactions* for 1810; and from the similarity which this substance bears to certain oxides in uniting both with alkalies and acids, Dr. Wollaston termed it an oxide, and gave it the name of *cystic oxide*, on the supposition of its being peculiar to the bladder. Dr. Marcet, however, has found it in the kidney. The first calculus examined by Dr. Wollaston of this description was taken from a boy five years old. It has been found also in the adult; and Professor Stromeyer found it in two instances in one family. Its formation as a concretion results from a general constitutional state. It has been discovered in the urine in a state of solution, of mechanical suspension, and also in the solid form of a calculus, either pure or encrusted with the phosphates or lithates. The concretion, when pure, is not laminated, but appears as one uniform mass confusedly crystallised through its whole substance, having somewhat of the appearance of the ammoniaco-magnesian phosphate, though more compact. Before the blowpipe it emits a peculiarly fetid smell, quite distinct from that of *uric acid*, and is consumed. It is characterised by the great variety of reagents in which it is soluble. It is dissolved abundantly by the *muriatic*, *nitric*, *sulphuric*, and *oxalic acids;* by *potash*, *soda*, and *ammonia*, and even by the neutral carbonates of *soda* and *potash*. It is insoluble in *water*, *alcohol*, *bicarbonate of ammonia*, and in the *tartaric*, *citric*, and *acetic acids*. The urine in which this substance has been found was copious, of a yellowish-green, of a strong peculiar smell, and of a low specific gravity; it was entirely free from uric acid, and the urea deficient in quantity. This diathesis is of unfavourable prognosis, and its mode of treatment not yet determined. Dr. Prout has seen most benefit from the *nitro-muriatic acid*. The peculiar smell of the urine abates under its use; but the complaint shows a great disposition to return when the medicine is left off.

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* Cystine precipitated by acetic acid from its ammoniacal solution (after Thudichum).

CLASS III.—SEDIMENTS COMPOSED OF SUBSTANCES FOREIGN TO THE URINE, AND WHICH ACCUMULATE IN THE URINE ALWAYS AFTER EXPOSURE TO THE ATMOSPHERE.

(1.) *Fungi* in acid urine—*e.g.*, the *Penicillum glaucum*, its *spores*, *thallus*, and *fructification*. (2.) The *Torrulæ cerevisæ* in saccharine urine begins to form in two or three hours after emission. It forms a gelatinous mass, composed of *sporules*, which subsequently develop into *beaded threads*, and in a few days aërial fructification appears. (3.) *Vibriones* and *Monads* occur in urine containing much mucus, and which is feebly acid or alkaline.

All urine should be collected in glass vessels, which are scrupulously clean; and *the quantity operated upon should embrace the whole urine passed during the twenty-four hours.* All sediments should be examined—(1.) Within a few hours after excretion; (2.) after twenty-four hours.

Of the substances dissolved in the urine, or becoming obvious only under special conditions, there are some which require more special notice, namely :—

1. *Hæmatine*, or *Blood-pigment*, occurs either as blood-corpuscles or separated from them. The corpuscles fall to the bottom of the tube or glass, and form a sediment. If the hæmatine is separate from the cor-

puscles, it constitutes a pigment completely dissolved in the urine, and to which it gives a brown or black colour (see scale of colour). It is always accompanied by albumen. Urine of a brown or black colour, due to blood-pigment dissolved in the urine, is to be determined by the elimination process in part—bile and vegetable pigments being absent. Urine containing *blood* in obvious quantity permits it generally to coagulate into blackish masses like pieces of black currant jelly; and when it partly coagulates in the bladder, linear masses of clot, of nearly the shape of leeches, are passed from the urethra. The urine has a port-wine hue, and abundance of entire red discs of blood-corpuscles may be detected with the microscope. These sink with readiness to the bottom of the vessel. Their non-granular surface, uniform size, and yellow colour, are characters sufficient to identify them. *Blood-pigment*, or *hæmatine*, dissolved in the urine, does not necessarily indicate local disease or rupture of vessels in any part of the urinary organs. It must be regarded as indicating rather a specially morbid condition of the blood, as is associated with septic poisons, or with profound cachectic diseases (PARKES). It may be observed in *typhus fever*, *malignant variola*, *remittent fever*, *yellow fever*, *scurvy*, and *Bright's disease*. Albumen is said always to co-exist.

2. *Albumen.*—Its presence must be regarded as an indication of disease, although it has been observed to occur in small quantity, and temporarily, in the urine of some persons supposed to be at the time in a healthy state; although it is more than probable that slight disease is present, or that such grave affections as Bright's disease may be commencing, to terminate fatally perhaps two years later (CHRISTISON, *Monthly Journal*, Jan., 1851; PARKES, p. 184). Very slight disease—not more than what appears to be dyspepsia—may have albuminuria associated with it, of a temporary nature. It has been observed in *intermittent fever*, *typhoid fever*, *rubeola*, *variola*, *pneumonia*, *pleuro-pneumonia*, *scarlatina*, *bronchitis*, *pleurisy*, *peritonitis*, *intestinal catarrh*, *follicular enteritis*, *acute rheumatism*, *chlorosis*, *cardiac diseases*, *phthisis*, and *malarious cachexia* (SOLON, FINGER, PARKES).

Hæmaturia has been found associated with albuminuria in *scarlatina*, *rheumatism*, *arthritis*, *typhus* (during the second week), *nephritis* (*calculous?*), *pneumonia*, *erysipelas*, *torpor*, *hepatitis*, *peritonitis*, *phthisis*, *chlorosis*, *intense oxaluria*, *phosphatic diathesis*, *last month of pregnancy*, *uterine disease*, *bladder-cancer*, *internal use of turpentine* (HEYWOOD THOMSON, *Lancet*, July, 1857; PARKES).

The late Dr. Parkes made a strong distinction between "temporary" and "permanent" albuminuria. The former ought to imply that the albuminuria, after lasting for some days or even weeks, finally disappears entirely. The latter implies that albuminuria does not disappear. He found temporary albuminuria, with the quantity of albumen large, in *acute lobar pneumonic cases* and *acute renal cases*, such as Bright's disease. He found albumen considerable in amount in *typhoid*, *variolus*, and *scarlatinal cases*, and small in amount in *paraplegia* (*spinal*), *hemiplegia*, *chroni phthisis*, *acute pleurisy*, *acute bronchitis*, *in hypertrophy*, *dilatation*, *and valvula affections of the heart*, *in acute and subacute rheumatism*, *in purpura hæmorrhagica*, *in typhoid and typhus fever*, *and in erysipelas* (PARKES, l. c., p. 188)

"Permanent albuminuria" he found associated with all forms of *Bright's disease,* with *encephaloid and cystic disease of the kidney,* with *leucocythœmia and presumed lardaceous disease of the kidney, in chronic phthisis and in pleurisy, where casts and kidney-structures were seen in the urine, in hypertrophy and valvular affections of the heart, in cerebral softening of hemiplegia, in pancreatic disease, in purpura hœmorrhagica, and in typhoid fever.* The result is, that if heart diseases be excluded "permanent albuminuria" indicates kidney disease invariably, and the distinction between "temporary" and "permanent" albuminuria is apt to be drawn with insufficient care. In all cases where it occurs there is either congestion and increased lateral pressure on the vessels of the kidney, or there is absolute structural disease of their walls.

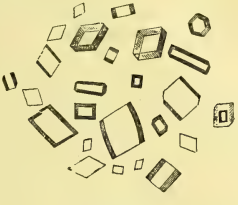


Fig. 70. *

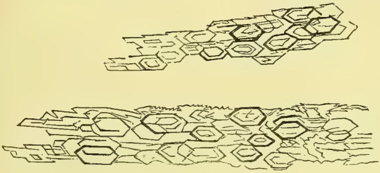
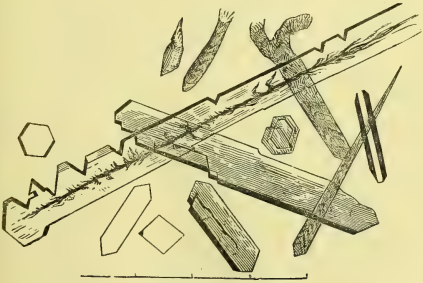


Fig. 71. †



100th of an inch × 42.

Fig. 72.‡

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* Oxalate of urea—perfect crystals (after BEALE, "Urinary Deposits," No. II., Plate IV.)

† Oxalate of urea (after BEALE, "Urinary Deposits," No. II., Plate IV.)

‡ Oxalate of urea from urine—extraction by alcohol, and an oxalate formed by the addition of oxalic acid.

In the Mauritius, and in Bourbon, a singular hæmaturia sometimes occurs, in which a varicose condition of the lymphatics has been supposed to cause the albumen (PARKES, p. 190).

3. *Urea.*—It may be of importance to determine the presence of urea in a small amount of fluid, as that of the ventricles of the brain. The process has been already explained. If in place of nitric acid, oxalic acid is used, we obtain crystals of *oxalate of urea* (Figs 70, 71). The alcoholic extract of urea leaves, on spontaneous evaporation, acicular crystals of the form shown in Fig. 72.

SECTION V.—DISEASES OF THE KIDNEY.

BRIGHT'S DISEASE—Syn., ALBUMINURIA.

LATIN EQ., *Morbus Brightii*—Idem valet, *Albuminuria;* FRENCH EQ., *Maladie de Bright*—Syn., *Albuminurie;* GERMAN EQ., *Bright'sche Krankheit*—Syn., *Albuminurie;* ITALIAN EQ., *Malattia di Bright, Albuminuria.*

Definition.—*A generic term, including several forms of acute and chronic disease of the kidney, associated with albumen in the urine, frequently with dropsy, and with various secondary or intercurrent affections, resulting from deterioration of the blood.*

Pathology.—(*a.*) *History.*—The disease has been named after Dr. Bright, an eminent physician of Guy's Hospital, who, in 1837, first drew the attention of the profession to the connection which he observed to subsist between certain forms of anasarca and lesions in the kidneys. It has of late years been most extensively investigated by Christison, Frerichs, Ringer, Walshe, Parkes, Gairdner, Wilks, Goodfellow, Basham, and others. The questions which the subject opens up in pathology are of extreme importance. Dr. Bright (who first discovered the relation subsisting between the morbid kidneys and certain cases of anasarca) described the disease, which now bears his name, as an affection of the kidney passing through *three* stages. Martin Solon describes *five* stages, and Rayer no fewer than *six*, and Rokitansky *nine*. More recent observers, such as Frerichs, Reinhardt, and Chambers, recognise three stages. Those who contend for the difference of stages affirm that in the first stage the kidneys are unusually large, flabby, loaded with dark venous blood, and hardly in any respect different from what is observed in diffuse inflammation, except that externally the kidney has a granular appearance caused by the deposition of a dark, reddish-yellow matter, the result o inflammatory products. In opposition to the views which hold tha *Bright's disease* is always one morbid state passing through successiv stages, the observations of Dr. George Johnson, of King's College; of Dr. Wilks, of Guy's Hospital; and of Dr. Goodfellow, of the Middlesex, lea to the belief that there are several lesions of the kidney developed independently of each other, and associated with the phenomena of Bright disease. In previous editions of this text-book I submitted a large amount of evidence, sufficient to justify our regarding Bright's disease a a constitutional affection culminating in a variety of structural lesions of the kidneys, each of which is accompanied by the persistent separation of more or less albuminous serum from the blood, and by its presence in the urine, the connective tissue, and serous cavities of the body. According to this view it ought to be classed with *gout* and *rheumatism.* The urine frequently contains blood, renal structures, exudation material, and desquamative renal products. The kidneys may be either large or small, atrophic or not, with fatty, or albuminous, or lardaceous degeneration, and altered in various ways as regards the vessels, the tubes, and the epithelium. In Bright's disease, as with many others, an undue share of attention has been bestowed upon the prominent local lesions in which the existence of the constitutional state has finally culminated. For example, the anatomical characters of *tubercle* in *phthisis;* of *cancer growths* in the *cachexia* of *carcinona;* of the *supra-renal bodies* in *morbus Addisonii;* of the *serous effusions* into the spinal and other visceral cavities in *beri-beri;* of the *kidney* in *Bright's disease* and *mellituria;* of the sediments in the urine in *oxaluria, lithiasis,* and the like—all these have been so prominently dwelt upon that a very narrow view is apt to be taken of their important antecedents—namely, the constitutional states under which they have respectively become developed, and therefore, also, of the principles which ought to guide the treatment of these respective diseases, and of the means by which their development may be prevented. A vast amount of evidence has been accumulating since Drs. Wilson Philip, Tweedy Todd, Sir James Clark, Bennett, and Ancell, demonstrated how prominently the general health was impaired, and how forms of dyspepsia prevailed long before *scrofula* became fully expressed, with or without the deposition or growth of *tubercle.* This kind of evidence tends to show that not only are constitutional phenomena characteristic of scrofula long antecedent to the expression of the disease by local lesions, but that other diseases, having similar constitutional antecedents, ought to command a more extended study from this point of view than they have hitherto obtained. *Bright's disease* and *diabetes mellitus* evidently belong to a group of general or constitutional diseases. The cause of the disease is to be sought for in some anomalous constitution of the blood—to something which gives rise to a change in the blood, rather than to any specific disease of the kidney (ROKITANSKY, 1841 to 1846). It is this anomalous condition of the blood which must be regarded as the primary affection, which, from some peculiar relation to the kidney, is followed by the secondary disorganisation of the renal tissue. The general or constitutional origin of the disease is also shown in the fact that both kidneys express the local lesion about the same time, or with a brief interval. In 1849, Dr. Walshe (Emeritus Professor of Medicine in University College) published a commentary on a clinical lecture, in which he taught that Bright's disease was not essentially a renal, but a blood disease (*Lancet,* July 14, 1849). There seems to be now an abundance of facts and arguments to establish—(1.) That from the very commencement Bright's disease is a general disease of the blood; (2.) that the renal and other textural changes are not the cause of the phenomena comprehended under the name of Bright's disease; (3.) that the local lesions which occur in the kidney do not even furnish any measure of the intensity of the characteristic phenomena of Bright's disease; (4.) that the textural changes in the kidney and blood-vessels generally are in reality the local expression of the constitutional state, just as tubercle is of tuberculosis; (5.) that the primary constitutional and blood changes are probably due to a morbid condition or direction of the primary and secondary digestion processes; and, (6.) that Bright's disease is thus recognised as one of the typical *diathetic* or *constitutional diseases.* The more eminent pathologists and discriminating physicians of the day have taken a similar view of the essential nature of Bright's disease—that it "depends on some humoral derangement of the entire system, and (the subacute inflammation of the kidney) commences as functional excitement, manifested in an act of over-secretion. The *materies morbi* seeks to effect its discharge by means of an increased activity in the secreting functions of the kidney. It stimulates the kidney, the result of which is not so much an increase of the watery secretion as it is an augmented cell-growth in the tubules of the gland. This acceleration of function is incompatible with the maturity of the secreted products; the epithelial cells undergo various arrests or modifications of development, and become more or less palpably imbued with evidences of inflammation" (Mr. JOHN SIMON). Dr. George Johnson teaches a similar doctrine. He shows that the morbid states of the kidney found in Bright's disease "are often mere local manifestations of a more general constitutional disorder;" and Sir Thomas Watson concurs with him in the belief that "it is the quality of the blood sent to the kidney, and not its quantity, that determines the renal disease." The experiments made by the late Dr. Parkes (*Med. Times and Gazette*, 1854, p. 394) regarding various phenomena in Bright's disease, tend to strengthen this view of its constitutional development; especially when studied in connection with the observations and experiments of Lehmann, Bernard, and Pavy in relation to the excretion of a substance by the liver, and the formation of sugar in the blood when that substance comes in contact with albumen. The condition of the urine before and after food is a point which has been in a great measure neglected till Parkes showed its importance in cases of this affection. In many cases of persistent albuminuria with renal casts and anasarca (not dependent on heart disease) the albumen in the urine is augmented after food. But apart from the albumen excreted, Parkes found that the solids excreted were increased. A weak, thin, quiescent man gave off daily 981 grains of non-albuminous solids, whose average in health did not exceed 650 to 750 grains. It has usually been considered that in chronic Bright's disease the non-albuminous solids are diminished (CHRISTISON, FRERICHS, JOHNSON); but this rule has numerous exceptions (PARKES). To detect the increase of the solids, ALL the urine passed during the twenty-four hours must be collected and examined. It is then found that the urine is of low specific gravity, but so copious that the total amount of solids in it is very great. Such cases are found to be constantly feverish —not so much so as to be detected by the hand; but the thermometer under the axilla will show that the temperature is uniformly more than a degree and a half above the healthy standard of 98° Fahr. Thus the thermometer will delicately indicate how an increased metamorphosis of tissue is going on in such cases; and observing such an increase of urinary non-albuminous excreta, it may be concluded that they are partly due to increased disintegration of tissue. "The morbid material which thus stimulates the kidney in its struggle for elimination will sometimes consist of products of faulty digestion—*e.g.*, lithates, oxalates; sometimes of matters cast upon the kidney in consequence of suppressed function in other organs—*e.g.*, the skin, the liver; sometimes the mysterious ferment of a fever poison—*e.g.*, typhus, scarlet fever, enteric fever" (Mr. JOHN SIMON). This view of the subject and method of investigating cases of Bright's disease, as well as of other wasting diseases, is not sufficiently practised and studied by the student of medicine. It will be found, as Parkes has shown, that many chronic cases of Bright's disease are often really slow febrile cases, just as tuberculous cases are. In Bright's disease, and constitutional diseases generally, much may be learned regarding the nature of the individual case by examinations of the urinary excretion, and microscopic characters of the sediment, in connection with the temperature of the body, the number of respirations, and the state of the pulse. The following formula may be suggested as a method for a daily record of the chemical composition of the whole urine passed during twenty-four hours; and if chemical and microscopical examinations of the urine are made from day to day, and carried on continuously, the particular morbid state of the kidney associated with Bright's disease may be probably or approximately ascertained:—

DATE.	Quantity in cubic Centimetres.	Specific Gravity.	Solids (in grains).	Urea.	Albumen.	Sulphuric Acid.	Phosphoric Acid.	Extractives.	Remarks.

The albuminous character of the blood after meals was long ago pointed out by Dr. Andrew Buchanan, late Professor of the Institutes of Medicine in Glasgow (*Trans. Phil. Society of Glasgow*, Vol. II.) He found a peculiar state of the blood, amounting to an appearance of leuchæmia, which immediately succeeds digestion in healthy persons, which seems to contain numerous molecular and corpuscular elements, resembling chyle, or lymph, and consists of fat emulsionised with albumen. To this substance, which is still further elaborated in the blood, in the glands, and in the lungs, he gave the name of *pabulin*. The earliest notice of milky blood is that in the *Philosophical Transactions*, No. 6, p. 100. During the processes of digestion in the stomach, albuminous substances are known to undergo extraordinary changes; and during the transit of the blood through the liver, the fibrine and albumen undergo still further modification, so that 30 per cent. of the albumen entering the liver in the portal vein disappears in that organ, and is not to be found in the blood of the hepatic vein

(LEHMANN). Thus the liver exerts an overwhelming influence on every digested aliment which enters it, and the influence which it exercises over albuminous aliments is not the least important (PARKES). In the interesting and suggestive work published by Dr. Basham, there are cases detailed which illustrate, upon these principles, the constitutional origin of *Bright's disease;* so that, connecting all these observations together—namely, those of Andrew Buchanan, Walshe, Lehmann, Bernard, Parkes, Pavy, Carpenter, and Basham—may it not be suggested as a topic for further inquiry (and at all events as a direction which inquiry should take), whether or not substances usually elaborated by the liver are not arrested in Bright's disease, or that the liver is insufficient to use up the albumen carried into the blood, which therefore comes to be eliminated by the kidney? "May it not be possible that, by some failure in preparation, either by the stomach or liver, albumen enters the blood of the right side of the heart in a still crude state, and in a condition similar to that in which it would have been had it been introduced through the jugular vein? and thus, being unassimilatable, is it not excreted by that ready outlet, the kidney? In support of such a supposition, we have the facts, that many cases of kidney disease seem to be most probably of blood origin, and that among the common antecedents of Bright's disease are circumstances of diet or mode of living which would be likely to impair the processes which should go on in the stomach or liver. In how many cases of Bright's disease have dyspeptic symptoms been present for a long time before renal signs have shown themselves? In how many other cases are signs of liver diseases coincident with the renal signs? In how many *post-mortem* examinations, although no liver disease has been suspected, do we find evidence that for a length of time the structure of this organ has been seriously diseased? In the history of Bright's disease there are many reasons for believing that the nutrition of tissues is early and deeply affected" (PARKES). The renal engorgement is certainly not the first in the order of morbid change: it is secondary to more remote morbific action, pervading the system throughout. An altered, defective, or contaminated state of the blood is the source, in all probability, of the morbid phenomena which follow. A careful inquiry into the antecedents of patients suffering from Bright's disease will exhibit the predisposing causes of their failing health; and these will ever be such as exercise a marked influence over the quality of the blood, rather than such as have any special reference to the kidneys. The most frequent and patent of these predisposing causes are habits of intemperance, inducing an alcoholised state of the blood; the various febrile poisons, particularly the scarlatinal; a scrofulous habit of body, or the tuberculous constitution (BASHAM). We see here a wide field for future exploration, especially in discriminating the forms of dyspepsia or of ill-health which precede these cases, which are frequently to be seen among the wandering uncertain crowds who frequent the outdoor or dispensary practice of our large city hospitals. In the morbid anatomy of Bright's disease, and diseases generally of constitutional origin, the glandular structure of the stomach and intestines, as investigated by Handfield Jones, Schäpfer, Habershon, and Fox, promises to yield important results in connection with the early history of such cases.

(*b.*) *Causation.*—The predisposing causes of Bright's disease may be enumerated under the following heads—namely, hereditary tendency or constitution of body, age, sex, climate, occupation, habit of life, intemperance, especially as regards alcohol, deficiency of food, with fatigue and mental anxiety, exposure to cold and moisture, influence of disease-poisons, such as *scarlet fever, cholera, measles,* and *erysipelas.*

Intemperance and *gout* are the circumstances which seem to bring about the disease most frequently in advanced life. In *youth* and *childhood* the hereditary constitution of body, exposure to cold, or the effects of general diseases, such as *scarlatina, erysipelas, measles, variola, syphilis, gout, rheumatism, tuberculosis;* the indiscriminate and excessive indulgence in fermented liquors, such as *ale* and *stout;* the ingestion of *turpentine, ether, naphtha, chloroform, alcohol, lead,* and the like, are the agents which tend to that morbid state of the constitution, and of the digestive functions, which lead to Bright's disease. Excessive devotion to mental work ought also to be regarded as a cause, especially if it leads to dyspepsia. A cold, humid, and variable climate is another powerful predisposing cause, although the disease is by no means infrequent in tropical climates. These are causes which act directly upon the blood and nervous system, through the digestive and secretory functions, which tend to interfere especially with the circulation and secretion of the kidney. There may also be such inherited weakness of constitution, that impaired digestion may lead to the development of Bright's disease, rather than to any other constitutional ailment. There are also causes of Bright's disease which may be regarded as mechanical sources of irritation, but which so affect the constitution that Bright's disease rather than any other is the result—*e.g.*, the *irritation of blows,* of *cantharides,* or other irritants; the presence of calculi in the kidney, the practice of *onanism,* or excessive *venery,* and the existence of *amenorrhœa* or *uterine irritation.* The influence of spirits and of beer tends each to induce a distinct variety of morbid kidney (Dr. GOODFELLOW). From the action of spirits the kidneys do not enlarge, nor vary much from the normal weight; but they become extensively granular, and full of small cysts. The cortical portion is greatly reduced in thickness, and there is much fatty matter in them. A tendency to fibroid growth is engendered—a tendency which the kidney shares with other organs, such as the *liver* and *lungs.* The granular form is also associated with gout as a primary morbid condition. It is a form of kidney lesion also produced by lead poisoning. A man with indications of lead poisoning, and who at the same time has albuminuria, is almost certain to become the subject of granular kidney. It is therefore common amongst painters, plumbers, compositors, tin-workers, and gasfitters. In persons who become subjects of Bright's disease from the excessive consumption of beer, the lesions of the kidney are of a mixed nature, something between the large white kidney seen after scarlatina and the true granular kidney, with more or less fatty deposit both in the tubes and in the interstitial tissue. But while intemperance in drinking is a recognised cause of Bright's disease, it is not unknown among children and other persons whose living has been strictly sober. Sir Thomas Watson mentions the case of a young girl, fifteen years old, who had never menstruated, and who became affected with Bright's disease; and he remarks that the disorder has been known in many instances to follow a sudden check or suppression of the catamenia.

(*c.*) *Morbid Anatomy.—The acute form of Bright's disease* seems to bear a similar relation to chronic Bright's disease that cases of acute phthisis bear to scrofula. It is described under the name of "*acute desquamative nephritis*" by Dr. George Johnson (whose investigations have especially elucidated this form of kidney disease); and it corresponds with the acute inflammatory dropsy of many writers. Elements closely allied to pus form in the kidney-tubes. In the rapidly fatal forms the products in the urine occur at first in the form of casts—a catarrhal process. These casts may accumulate and block up the tubes. The kidney is then enlarged, of a white colour (the large white kidney of Bright). Acute dropsy is constant, often ascribed to cold, or as a result of scarlatina. Urine may be for a time suppressed, and the little which passes is of a red-brown colour, generally from blood. The sediment is abundant, and deeply tinged with blood. It contains albumen and blood-casts, and is "composed of coagulated fibrine, blood-corpuscles, cells having for the most part the character of renal epithelium, and occasionally crystals of uric acid. Some of the fibrine is coagulated in irregular masses, having no definite form; this is always the case when the hæmorrhage has been abundant and rapid, so that much of the blood has escaped from the kidneys before it has had time to coagulate; but with these masses there will be seen numerous cylindrical bodies composed of fibrine, which, having exuded from the Malpighian bodies, have coagulated in the tubes, and, escaping thence, present solid cylindrical moulds of the interior of the tubes, in which are entangled blood-corpuscles and epithelial cells, which have been shed by a process of desquamation from the surface of the tubes" (JOHNSON, *On the Kidney*, p. 89). To such casts, characterised by the presence of recently formed and entire epithelial cells, Dr. Johnson proposed the name of "*epithelial casts*" —their average diameter being about $\frac{1}{700}$ of an inch. Death, sometimes after only a fortnight's illness, discloses a large soft kidney, with swollen cortical substance of a dark colour, on removal of the capsules, and the surface much injected. On section it exudes drops of blood. To the microscope the tubes are opaque, being filled with lymph-corpuscles and granular matter. Some have lost their epithelium, and are filled with coagulated fibrine, which, if expelled, would form a fibrinous cast. At a later period the pyramidal portions of the kidney are of a dark colour; but afterwards the cortical part becomes paler and softer, the surface not smooth when cut, and giving out a turbid fluid when squeezed. The tubes are seen to be denuded of epithelium, and filled with masses of secretion, which take on the forms of the tubes. Scanty and bloody urine are conditions associated with engorgement of the Malpighian tufts. These are visible as red specks to the naked eye, and the tubes may be found filled with blood, or containing hæmatine. The morbid process continuing, secretion collects in the tubes, and fills them to an extreme degree, and the more it accumulates, the larger and whiter the kidney becomes. The material can be seen with the naked eye in the cortical part. The surface is pale and smooth, with stellate or arborescent venation, surrounding numbers of opaque white spots. A section shows similar appearances, but the white material is now seen disposed in lines. If a congeries of tubes are swollen between the blood-vessels, granulations are formed. The new material consists of large masses of dark, granular, and fatty matter, which distend the tubes. A section shows, microscopically, large portions of tubes quite opaque; their natural lining is gone, and contiguous tubes become united or massed together by the new material which fills them. It is often difficult to determine whether the partition-membranes of the tubes exist or not (WILKS). Granular matter may also be seen in some parts of the straight tubes. It is exceptional to find it in the capsule of the Malpighian tufts.

There are several different forms of lesion in the kidney associated with the phenomena of *chronic Bright's disease.* The several forms are distinguished by certain anatomical characters, and by more or less characteristic symptoms throughout the disease. Two at least of these forms may be considered as typical and distinct; and other forms may be recognised as a commingling of these two—modifications of them—or a mixture of these typical states together, but not to be considered as gradations from one form into another. The two forms are essentially different, and never pass by any pathological process from the one to the other (WILKS, GOODFELLOW). Other forms of morbid kidney in this disease have been regarded as degenerations, namely,—(1.) the fatty; and, (2.) the amyloid or lardaceous kidney.

(1.) *Granular Kidney*—Syn., *Contracted Granular Kidney, Chronic Desquamative Nephritis, Gouty Kidney.*—In this form of lesion the kidney is smaller than the normal kidney, sometimes only half, or even less than half, the natural size. It is hard, contracted, red, and granular. The cortical part is much narrowed; it is wasted, and almost allows the cones to touch each other. The capsule, generally thickened and opaque, comes very near the margin of the pyramidal portion, and is in general separated with great difficulty from the surface, so that more or less of the substance of the kidney is torn away with it. The surface of the organ is granular; its substance is tough, coarse, and fibrous. The distribution of the veins on the surface of the kidney appears to determine the true shape or limits of the granulations and depressions seen on the surface. The granulations are composed of bundles of tubes lying between the meshes of these veins. It is this form of kidney which shows so well the fibrous matrix between the tubules when examined by the microscope—an appearance which some attribute to the shrunken state of the kidney, while others consider this matrix to be entirely new structure. There can be no doubt of a matrix or skeleton of connective-tissue existing in the kidney, and holding its tubules together, although it may in some cases present almost little beyond a granular appearance between tubes and blood-vessels. It is extremely delicate and difficult to be seen, unless the light is well managed; but it becomes intensely hypertrophied in the small contracted kidney of chronic Bright's disease. A microscopic section shows the connective-tissue greatly increased, and having a coarse appearance, passing inwards from the depressions on the surface, the tubes lying side by side, and closely packed together, shrivelled up and imbedded in the new connective-tissue. They are by no means uniform in appearance. Some are almost, if not quite solid, forming fibrous cords. Some are of very irregular shape, from contractions and dilatations, giving a sacculated or varicose appearance to the section. Many of these sacculated expansions being cut off from the rest of the tubule, form independent cavities or cysts, both in the cortex and in the pyramidal parts. They may be only capable of recognition by the microscope, although sometimes obvious to the naked eye. In the pyramids such cysts are sometimes arranged in a row, of an oval shape, lying end to end. Their walls are generally very thin; but nuclei and epithelium, as in neighbouring tubes, may still be visible. They are formed by portions of tubes, obstructed so as to inclose a space which forms the cyst, or by dropsical distension of the capsules of the Malpighian glomeruli. The large cystic kidney may be an exaggeration of the process (CLYMER). These tubules, cysts, or cavities are devoid of epithelium; and if any remain, it is imperfect, shrivelled, and granular. The walls of the tubes are puckered, and present an irregular outline (WILKS, GOODFELLOW). The process which leads to this form of kidney generally extends through many years, and is seldom found without, at the same time, a similar process having taken place in the *liver*, or the *spleen*, or the *lungs;* and it is more than probable that these have been affected contemporaneously from the influence of the constitutional cause which affects the body generally. The increase and alteration of the fibrous element tend to induce the atrophy and destruction of the tubes and glomeruli, and at the same time there are a degeneration and actual wasting of the whole tissues of the kidney. In the most extreme or advanced stage of granular kidney, the size is greatly reduced. I have weighed such kidneys as low as twelve drachms. The capsule is generally thickened, opaque, and not easily separated. On removal, the granulations are seen of a lighter colour than other parts—about one-eighth to one-sixteenth of an inch in diameter—marked out by irregular, livid, vascular depressions, contrasting with the pale, bloodless, granular elevations. The minute changes in advanced granular degeneration, as seen by the microscope, show that the essence of this affection consists in a slow irritation of the connective-tissue, especially that which immediately surrounds the Malpighian bodies and blood-vessels, followed by an increased germination of that tissue, so that it gradually usurps the place of the tubules, which it contracts and flattens till they become mere threads. The most conspicuous microscopic appearances and alterations are at the surface, immediately beneath the capsule, at the points of superficial depression, where little streamlets of fibrous tissue pass into the organ, imbedding the Malpighian bodies and compressing the tubes. As the fibroid material extends inwards, it becomes diffused, and spreads over and between the tubes in its neighbourhood. Or its existence may be chiefly indicated by the contraction it has caused, so that an angular space under the capsule is seen filled by the shrivelled remains of the tubes. In such cases the Malpighian bodies are aggregated, owing to the contraction of the parts between them, while they resist the compressing agency. The new fibrous tissue may be seen in isolated patches of some extent in the deeper parts of the cortex. In some portions the cortical tubes have been reduced to impermeable microscopic threads, whilst in others they are irregularly dilated. The effusion which leads to increase of fibrous tissue does not take place uniformly throughout, but at points a little removed from each other. The tubes in the track of the effusion may be involved, whilst others escape, and, in the earlier stages of the disease particularly, many tubes may be found perfectly natural. Those which are altered are in one or two conditions, both of which are commonly found in the same kidney: (*a.*) They are packed with epithelium, or distended with dark granular matter, probably the result of the breaking up of the cells subsequent to their detachment from the wall of the tube; (*b.*) a transparent fibrinous material may take the place of the epithelial lining, and occupy the tubes. This material may be quite uniform and glossy, or studded with oil-globules. Sometimes it is broken up into very fine fragments, and passes out with the urine as dark, coarse, granular casts, which often look opaque and granular till touched with acetic acid, when they become clear, and show the broken pieces of fibrine of which they chiefly consist. The condition of the tubes which form the cones is precisely the same.

The granular kidney has, on the other hand, been regarded by some as a result of original productive changes or growth of new material, which first shows itself in the large white kidney seen in certain cases of Bright's disease. In this form the kidney varies in size to nearly double, and weighs from six to twelve ounces, or more. Its external cortical secreting part is increased at the expense of the internal medullary or purely excreting part. Between the base of the pyramids of a cut section and the investing capsule, an extreme case may measure from half an inch to an inch. The general appearance of the kidney is a whitish or yellowish-white colour. It is anæmic, partly from the obliteration of the vessels, partly from more or less abundant serosity, and partly from the diminution in the amount of red blood-discs. In some parts the veins may be enlarged and turgid; in the interior of the kidney such veins are tortuous, on the surface they have an arborescent form. The medullary portion may present various degrees of engorgement, or it may be as exsanguine as the cortical part; but such extreme bloodlessness is rare. Such a kidney is flaccid. Its morbid development may be traced through two stages; and if the patient lives through these two, it may be recognised even in a third. If death ensues early, the kidney is found to be of tolerably firm consistence; its surface smooth, and of a more or less deeply suffused redness, very numerously interspersed with minute points of a deeper red. The capsule, a little more vascular than natural, may be easily detached. On section, the increase in size is in the cortical substance, which is minutely injected with blood, and presents an uniform redness, with numerous dots of a deeper red—engorged Malpighian bodies. Other red spots may be visible, due to ecchymoses. Sometimes there may be portions of a lighter shade of colour, from the growth of new material. With this general state of engorgement there may be actual extravasation into the Malpighian capsules, into the tubules, and even into the intertubular substance. The pyramids are redder than natural, and the mucous membrane of the calyces and pelvis is injected. The tubes are filled with epithelium, mixed with fibrinous matter and blood, and sometimes holding together minute crystals of oxalate of lime or lithic acid. The entire parenchyma, but especially the cortical substance, is peculiarly pulpy and friable; and the surface, on section or on fracture, yields a reddish-brown slightly viscid fluid, more or less bloody, and delicately flocculent or opaque. The *materies morbi*, seeking to effect its discharge, stimulates the cell-growth in the tubules of the gland—"an acceleration of growth and of function which is incompatible with maturity of the secreted products. The epithelial cells, therefore, undergo various arrests or modifications of development. The ultimate tubules are found gorged with an uneliminable excess of crude and vitiated secretion. Blood, amorphous matter, and an infinite range of cell-growth, from pus-globules to the healthy germination of the gland-cells, present themselves in various combinations. By products such as these the tubes are plugged and irregularly distended; and they not unfrequently burst and are annihilated. So close is the compaction of material, even in many of those tubes that have no shaped inflammatory products within them, that they are plainly impervious; and it is only by artificial means—by further tearing of the fragment, or by the use of chemical agents (under the microscope)—that we can satisfy ourselves that the dense plug in question consists of but agglomerated gland-cells" (SIMON). A second or more advanced stage in the morbid development of this large white kidney may be recognised in the still greater enlargement of the organ; but the blood engorgement has subsided, the cortical substance is now paler than natural—of a whitish or yellowish-white colour. It is still more thickened or enlarged relatively to the pyramidal portion, is somewhat soft and friable, and on pressure yields a turbid milky-looking fluid. The granular material is to be found almost entirely in the cortical part, both in the convoluted tubes and between them, and may be found dipping down between the tubuli of the pyramids, and separating them. The tubes of the pyramids at the base thus present a radiated, striated, and frayed or unravelled appearance; and in the cortical substance numerous whitish lines are traceable from the surface inwards, some of which are composed of numerous isolated whitish *puncta*, while others are uninterrupted. The pyramids are of a reddish colour, of different shades in different cases. The vessels of the cortex are extensively obliterated, and the new material in this stage has undergone more or less of fatty metamorphosis. These whitish *puncta* are more particularly abundant in the superficial layer of the cortical part. They are composed of tense granules of a yellowish-white colour (Bright's and Christison's granulations), of the size of a poppy seed or a pin's head. In the cortical substance these granulations are chiefly due to the lesions in the Malpighian capsules. Occasionally it happens that a patient may survive through these two stages of the kidney lesion, when a third stage of development may be recognised, which this large white kidney will pass through. The fatty degeneration progresses, and becoming absorbed, a wasted, shrivelled, flaccid, but tough kidney results. Microscopically, the appearances of this large white kidney vary somewhat with its stage of development. The Malpighian bodies are irregular in size, some being excessively enlarged, others being smaller than natural. Their capsules are more or less distended with granular matter, and so also are the tubes. The tufts of capillaries composing the glomeruli seem wasted, and only a few large distended ones are visible. Fat-globules may also be detected, and granules of fat in extreme minuteness. In some places the tubes are empty, and compressed by intertubular new material; but in general they are greatly distended, with altered epithelium, mixed with granular matter more or less fine. Here and there a tolerably healthy capsule and tube may be distinguished close to one that has undergone the changes already noticed. The altered epithelium which abounds contains varying quantities of oil. Some of the cells are large, others are shrivelled up and otherwise imperfect in form, and all of them are more or less granular; and lines of the same granular matter may be observed between the straight tubes. *The next stage* is marked by the granular matter penetrating still deeper into the cortical substance, and gradually increasing till it invades the whole of the medullary substance of the kidney. This granular substance is of a greyish-red or greyish-yellow colour, with something of a *cheese*-like appearance. The kidney may be larger than natural, sometimes of the natural size, and sometimes, though rarely, diminished. Its consistency also varies; if enlarged, it is softer than the healthy kidney; but, if diminished, it is firmer. Its colour, viewed externally, may be a pale tint of the natural hue; more commonly it is of a greyish-yellow or yellowish-red colour, and mottled. Its surface is strongly granulated, and even rough. In this state, if the kidney be injected, the matter of the injection does not penetrate the cortical portion (Dr. BRIGHT). This is the stage in which a complete and general metamorphosis of the inflammatory products into fat takes place. *The last stage* is marked by the granular deposit invading the tubular portions of the kidney, so that the tubuli are obliterated to a very considerable extent, perhaps entirely converted into one homogeneous degeneration. The kidneys are then generally contracted, and are smaller than usual. Their surface is lobulated, pale, and granular, resembling the roe of a salmon or the vitelarium of a bird. Their consistency is sometimes softer and sometimes harder than natural; and Dr. Bright speaks of some instances in which they cut like cartilage. This is the stage which leads to final atrophy and wasting of the kidney.

The changes in the circulatory apparatus of the granular or contracted kidney has been the subject of much controversy in recent years, without conclusive results. Under the name of *arterio-capillary fibrosis*, Drs. Gull and Sutton have described certain changes, not only in the kidneys, but throughout the capillaries and blood-vessels of the whole body, of the nature of a *hyaline* thickening of their walls with loss of elasticity. The granular kidney is then but a part of a general morbid state commencing in the capillary vessels of the whole body. Cardiac hypertrophy is a result of this change. On the other hand, Dr. George Johnson describes the walls of the small arteries, not only in the kidneys, but in various parts throughout the body, as greatly thickened; and that this thickening is due to hypertrophy of the muscular coats of those vessels, in consequence of the arteries opposing the entrance of the unhealthy blood into the tissues associated with Bright's disease. Hence the cardiac walls also become hypertrophied to overcome the resistance. In 1877, Dr. Richard Thoma, of Heidelberg, made a series of observations to determine the relative permeability under constant pressure of the renal vessels in this disease, as estimated from the outflow from the renal vein, comparing this with the same in healthy organs, the comparison being made with equal conditions as to age. The result is to show that not only is the actual quantity which can be passed through the organ in a given time reduced very largely, but *the proportion of the amount passing out by the renal vein to that introduced by the renal artery is much less than in healthy organs.* He has also made various calculations as to the size and measurements of the normal kidney, and of several sectional areas of its blood-vessels; and by injections alike in the normal and granular kidney he has proved that the capillary network in granular kidney is very scanty, and that, in many cases, the *vasa afferentia* anastomosed directly with the *efferent* vessels, being cut off from the *glomerulus* by the growth of fibrous tissue. As a rule, in spite of the fibroid growth of the intima, the lumina of the vessels are not smaller than normal; only exceptionally, when the endo-arteritis has reached a high grade, does this take place. These changes are connected with those endo-arterial processes—described by Köster, Friedländer, Trompetter, and others—as taking place in almost all new formations of connective tissue; they bear a distinct relation to the amount of interstitial growth. These facts are especially opposed to Gull and Sutton's theory, that the narrowing of the lumina of the vessels by the new growth causes the obstruction to the circulation; they agree with independent observations that, as a rule, the vessels in granular kidneys are certainly not of less calibre than normal; they prove that the anatomical condition of the organ itself, independent of any vital changes, such as arterial contraction, causes a great increase in the resistance to the circulation through it; they explain the clinical phenomena of *polyuria* and *albuminuria* by the increased permeability of the vascular walls. General capillary anæmia is a marked feature of even the early stages of this disease, and it is at least possible that the same cause, acting generally, may produce, although to a less extent, an universal reduction of the capillary network (*Brit. Med. Journ.*, November 24, 1877, p. 735).

(2.) *The Fatty Kidney*, as a degenerative change, may not always be associated with the phenomena of Bright's disease. The presence of fat in the kidneys in cases of Bright's disease is altogether a secondary process, and due to a degeneration of the inflammatory products and normal structures. The renal secreting cells are found to contain distinct globules of oil or fat granules, which obliterate all appearance of a nucleus. Little, however, is positively known regarding this degeneration as an independent form of Bright's disease. It is a degeneration which supervenes to a great extent upon the "large white kidney," and is found more or less associated with it in every instance. The fatty kidney is large and flaccid, full of yellowish or whitish striæ and marks, combined or not with the characters of the parenchymatous inflammation just described. The calibre of the urinary canals are diminished, and are separated by fatty masses.

(3.) *Lardaceous Kidney*—Syn., *Amyloid Disease, Waxy Kidney.*—The lardaceous kidney is usually associated with the phenomena of Bright's disease. It constitutes the eighth form of kidney lesion which Rokitansky describes in connection with that disease. It is the form most commonly found in patients who have suffered from constitutional syphilis or mercurialism; also in scrofulous or rickety persons, or who have suffered from prolonged suppuration, or from *caries* or *necrosis* of bone. It is generally associated with similar lesions of the spleen and liver. The kidney in this form of disease presents but a slight increase of size, and is always considerably indurated. It is of a brownish-yellow colour. The cortical substance on section has a glossy waxy look, with a glistening appearance of the glomeruli when the light is allowed to fall upon them in an oblique direction. It is usually hard and brittle, and infiltrated with the albuminoid material. Gairdner, Sanders, and Virchow were the first to discover the true nature of this condition of the kidney. It is to be distinguished from the large white and soft kidney (of Bright) already described. It is much harder, tougher, and firmer. It is not easily torn; and its surface is uniform and smooth. The cortical substance, having a dim waxy appearance, is increased in extent, and anæmia predominates. It is this translucency, hardness, and uniformity of appearance which are sufficient characters by which to identify extreme cases; and in the more common and slighter cases the Malpighian glomeruli can be seen shining prominently like glistening grains on the cut surface. The iodine test, combined with microscopic examination, will alone, and are absolutely necessary to, clench the recognition of the lesion (Vol. I., p. 134). But without the application of any reagent, the Malpighian glomeruli appear as glistening bodies, having a clearness resembling the appearance of "frosted" glass or a globule of rough ice. Unless a microscope has been used, it cannot be said that lardaceous disease is not present. The capsule of the kidney is generally easily torn off; and there may be considerable loss of substance or atrophy present, indicated by depressions or hollows on the surface of the kidney beneath the capsule. The degeneration may be so slight that a microscopic examination is necessary to detect its commencement. Lardaceous disease of the kidney begins with the arterial capillaries; especially—(1.) The loops of the *Malpighian* tufts; (2.) their afferent vessels; (3.) the *vasa efferentia* and capillaries in the cortical part; (4.) the "*arteriolæ rectæ;*" and after the lesion has advanced to a certain point the injection of the cortical substance becomes impracticable; the arteries become impervious, the cortical substance anæmic, while hyperæmia increases in the pyramids; and hæmorrhage is apt to occur at times, owing to the increased pressure on the inelastic vascular walls, giving rise to reddish-brown streaks or spots. Ultimately the Malpighian glomeruli become converted into solid, homogeneous, translucent masses; and the extension of the disease to the tubes and other tissues produces at length the condition known as waxy kidney. Sometimes the degeneration begins in the capillaries of the *arteriolæ rectæ;* but rarely are the large vessels affected, and it is long before the epithelial tissues become affected. The lesion is most frequently combined with parenchymatous or interstitial nephritis, or the interstitial fatty kidney, especially in syphilitic cases. Professor T. Grainger Stewart has found great amelioration of the symptoms from the adoption of the following rules:—(1.) To attend to the nutrition of the patient, giving good nutritive food in the form best suited to the individual tastes and powers of digestion; (2.) to give such tonic medicines as may improve the appetite; (3.) to give such hæmic medicines as control the tendency to anæmia; and among these, pre-eminently, the syrup of the *iodide of iron*. The *syrup of the phosphate*, as well as the *syrup of the phosphates of iron*, *quinia*, and *strychnia*, are also most useful remedies. (4.) In all cases in which a syphilitic infection has been traced, and even in many others, *iodide of potassium* is advantageously combined with *carbonate of ammonia*, and is to be given in moderate and sustained doses. It diminishes the bulk of the liver in a remarkable manner (*Edin. Med. Journal*, August, 1864).

(4.) *Mixed Forms of Kidney Lesions in Bright's Disease.*—There are cases, when the patient has been ill for a long time, in which the kidney is found presenting the characters of more than one of the lesions described. The organs may be of ordinary size, granular, white, degenerating, and containing deposits visible to the naked eye, the processes leading to the lesions now described being engrafted, so to speak, the one upon the other. The kidneys are less pale than those which constitute the "large white kidney." The vessels are much more numerous, and more or less gorged with blood. The Malpighian tufts are red and solid, and the organ presents a very coarse and granular appearance (GOODFELLOW).

The College of Physicians has not adopted the view which regards Bright's disease as a general or constitutional malady. It is classed under "Diseases of the Urinary System;" and considered under two forms, namely,—(1.) *Acute Bright's disease*, of which the synonyms are "*acute albuminuria*," "*acute desquamative nephritis*," "*acute renal dropsy*," "*acute tubal nephritis;*" (2.) *Chronic Bright's disease*—Syn., "*chronic albuminuria*," with the following subdivisions:—(a.) *Granular kidney*—Syn., *contracted granular kidney, chronic desquamative nephritis;* (b.) *Gouty kidney;* (c.) *Fatty kidney;* (d.) *Lardaceous kidney*—Syn., *amyloid disease, waxy disease.*

I. ACUTE BRIGHT'S DISEASE—*Syn.*, ACUTE ALBUMINURIA; ACUTE DESQUAMATIVE NEPHRITIS; ACUTE RENAL DROPSY; ACUTE TUBAL NEPHRITIS.—**Symptoms.**—It is an intense febrile disease, which may come on after scarlatina and other exanthemata, or independent of these. It is marked by signs of intense congestion of the kidney, with exudation and hæmorrhage into the tubes, and desquamation of the epithelium The secondary phenomena are uræmic symptoms to a greater or les degree, and in the majority of cases general dropsy. The *urine* in th early stages and height of the disease presents intensely febrile characters It is small in quantity, deeply pigmented, and deposits urates. It contains a variable but usually a large amount of albumen and blood. Th sediments consist of desquamated kidney ureter, and bladder-structures voided renal cylinders, and sometimes large masses of coagulated fibrine or partly decolorised clots. Urea is augmented; and when it appears t be below the normal amount, its exit is impeded, and uræmic symptom are present, which generally increase and prove fatal. When the urin is very scanty, it becomes almost solid by heat. When the disease i about to end fatally, the quantity of water and of the solids decrease, th diminution of the solids being more considerable than that of the wate The albumen decreases least, and is very abundant till the last. Whe recovery is about to take place, *diuresis* usually occurs; and often a enormous quantity of water is passed, containing much *urea* and *chlori of sodium*. The albumen at the same time diminishes and disappear and the kidneys recover perfect health (PARKES, l. c., p. 378).

II. Chronic Bright's Disease—*Syn.*, Chronic Albuminuria.—**Symptoms.**—It is necessary to inquire into the history of each particular case of Bright's disease, so as (1.) to ascertain the precise period, if possible, when the general health began to be impaired; (2.) to determine the particular morbid condition in which the kidney exists. For this latter purpose it is necessary to make a chemical and microscopical examination of the urine from day to day, determining especially those points which have been enumerated in the preceding section. When Bright's disease is fully expressed there is characteristic anæmia, indicated by pallor of the countenance, a puffy face and œdematous limbs, anasarca or general dropsy, pain in the loins, with more or less albumen in the urine. Associated with these phenomena, the morbid states of the kidney are various in the extreme. There is scanty urine and frequent micturition; a sensation of heat and scalding on passing water, dryness, heat of skin, general feverishness and occasional chilliness of the surface, an irritable pulse, generally above the normal standard, emaciation, and progressive debility. Dyspeptic symptoms predominate. There is difficulty of breathing and anæmia-chlorosis.

The general symptoms indicative of the several states of the kidney, and comprised under the general term "Bright's disease," have been recently analysed and described with great care by Dr. Goodfellow, Lecturer on Medicine at the Middlesex Hospital. They may be comprised under the following heads:—

1. Although *anasarca* or *general dropsy*, either or both of which are usually prominent symptoms when a patient with "Bright's disease" seeks medical advice, yet in nearly every instance they have been preceded by other phenomena more or less definite—*e. g.*, febrile excitement, a dry and harsh state of the skin, a quick and hard pulse. It is only in the acute cases that the prominent and characteristic phenomena of anasarca come on with great rapidity, and commence generally with puffiness of the eyelids, or of the whole face, rapidly extending over the rest of the body. In the more chronic cases the œdema generally commences in the lower limbs; and it is at the same time associated with a pallid condition of the eyelids, and of the looser connective tissue of the face.

2. *Anæmia* is a prominent phenomenon. It is indicated by the pallor of the surface of the body and of the countenance. Its progressive appearance may even suggest a suspicion of renal disease before the anasarca sets in, especially in patients above the age of from thirty-five to forty, and whose urine ought therefore to be at once examined.

3. *Pain in the loins* may or may not be a sign of significance. Lumbar pains may be considerable in amount, and yet no renal disease may be capable of detection at the time. In most cases no unpleasant sensation is felt in the lumbar region till the anasarca becomes considerable.

4. In the earlier stages there is always *a considerable diminution in the quantity of urine passed.* But there are many exceptions to the rule, and in some cases the urine is more abundant than usual—*e. g.*, Parkes has measured more than 100 ounces; Rosenstein (quoted by Parkes) relates a case where an increase of 174 ounces was passed daily during eight days. Various circumstances concur in determining the greater or less

amount of water passed. (See under "Lardaceous Disease," Vol. I., p. 130). (*a.*) The anatomical condition of the kidneys influences the amount, and especially with or without anæmic symptoms. The mean of *six* cases without anæmic symptoms gave 61·5 ounces daily. The mean of *four* cases with uræmic symptoms gave 38 ounces daily, the specific gravity in both sets of cases being very nearly the same (PARKES). In the highly atrophic kidney, when many vessels are obliterated, the urinary water is on an average lessened; but on some particular days a large quantity may be excreted; for example, Dr. Parkes has known as much as *sixty ounces* passed in one day by a kidney under such a condition. (*b.*) The co-existence of dropsy with anæmia in an advanced period of the disease appears to be associated with lessened water; while, on the other hand, an improvement in dropsical symptoms is attended by more or less profuse diuresis. (*c.*) In a few cases the amount of urine is influenced by the presence or absence of fever. The quantity is very much smaller in amount on fever days as compared with fever-free days. (*d.*) Organic lesions of the liver, heart, and lungs may cause lessened rapidity of flow through the renal vessels, or may give rise to great variations in lateral pressure. (*e.*) One of the main causes of a variable amount of water is the spontaneous purging or vomiting which sometimes takes place, which would lessen the amount of urine; while, on the other hand, lessened skin exhalations will increase the flow.

Until the metamorphosis of the nitrogenous tissues in Bright's disease is better understood, the question as to the possible lessening of *formation of urea* and *uric acid* must remain undecided. Hitherto lessening of these products seems to have been the rule, to which, however, there are numerous exceptions (PARKES); and, on the whole, it appears probable that the effect of *a febrile* Bright's disease on the ordinary urinary constituents is chiefly owing to the physical condition of the kidney. There is no doubt that urea in the blood is increased; and it seems pretty clear that the urea in Bright's disease is still formed to a considerable amount, and that its lessening in the urine is in part owing to *retention* from simple impediment to diffusion through the diseased renal vessels and tubes; but it is yet undecided whether or not there is at the same time a *lessened formation* of urea (PARKES). The principal abnormal constituent of the urine in Bright's disease is dissolved albumen, which exists in various conditions, and gives different reactions with the usual tests, heat and nitric acid. Sometimes it entirely coagulates, and is precipitated by heat and a moderate quantity of nitric acid; at other times, after precipitation by heat *from an acid urine*, it is entirely resoluble in a moderate amount of nitric acid. A very minute quantity of nitric acid will also sometimes prevent its precipitation by heat, while, on the other hand, it is sometimes precipitated by nitric acid, and not by heat (various authorities quoted by PARKES). Its most usual condition, however, is that in which it is precipitated by heat from a urine which is naturally sharply acid, or which has been made so by a moderate quantity of nitric or acetic acid. O the apparently anomalous phenomena just described, the following explanation is given by Parkes:—"When albumen is experimented with, it can be made to pass through various phases of solubility and insolubility from the actions of acids, alkalies, and salts, without its real nature o composition being in any way altered; and as such influences will act on it more or less in every urine, it by no means follows, when the albumen in the urine of Bright's disease presents modifications in its reactions with reagents, that it is in any very peculiar or unusual condition" (*On the Urine*, p. 390). The amount of albumen varies much, ranging from 5 to 545 grains in the twenty-four hours (PARKES); and in any particular case the amount varies greatly from day to day. It is often increased during the day, from movement or from food, and Parkes has noticed it very greatly increased in the second and third hours after dinner. The albumen, however, is not constant, nor does it occur in acute cases of Bright's disease; though passed, it is often in a peculiar condition, being less perfectly coagulable by heat, and sometimes approaching in character the *albuminose* of Miahle. The albumen does not always increase as the disease advances; on the contrary, while there is a tendency to a larger excretion of albumen in the early stages of the disease than in the later ones, and while sometimes even at the last there is much albumen, yet it is sometimes entirely absent in the later period of the disease. In a certain number of cases the albumen may quite disappear from the urine; but it is important to notice that the detachment of fibrinous casts goes on even when the albumen has disappeared.

Fat in small quantities is very common in the urine derived directly from the kidneys. It is usually in the form of *oleine*, mixed up with albuminous substances, or contained in the epithelial or other cells, and microscopically visible; and it must be remembered, as Dr. Johnson was the first to determine, that some of the secreting or epithelial cells of the kidney inclose a minute quantity of fat, just as in the secreting cells of a healthy liver, as shown by Bowman.

In the more advanced stage of chronic Bright's disease, instead of the urine being scanty in quantity, and having a tolerably high specific gravity, the urine passed in twenty-four hours may amount to from *thirty-five* to *fifty* ounces or more, being equal to, and in some cases greater than, the average in health. The specific gravity is nearly always below the healthy average, the urine pale, and in very advanced cases almost colourless. A large flow of pale coloured urine, of low specific gravity, is one of the best marked evidences of chronic renal disease, with a persistent condition of high arterial tension. Occasionally the urine may be red, reddish-brown, pale smoke-brown, or "smoky," as it is commonly described. If the urine is highly fatty, it may have a milky appearance. On the other hand, the absence of a large flow of urine does not negative the presence of renal disease. From the yielding of the diseased heart texture, dilatation of the heart may result, with diminished arterial tension. Palpitation exists, Atheromatous changes occur in the arteries generally, so that hard. thickened, elongated, tortuous arteries are common in chronic Bright's disease. The large flow of urine characteristic of the earlier stages passes to a small amount of urine characteristic of heart failure.

Besides the albumen and other constituents of the serum of the blood, the urine may contain blood-corpuscles, casts containing blood, fibrinous filmy matters, and casts of tubes (granular, fatty, waxy, or hyaline), or simply of epithelium. A perfect acquaintance with these casts and with urinary deposits is essentially necessary; for by the characters of these casts, when taken in connection with the history and symptoms, the physician is able approximately to discriminate the morbid condition of the kidney in each particular case (BASHAM, GOODFELLOW). The *specific gravity of the urine* may range from 1,020 to 1,025, from two causes, namely—(1.) The presence of serum, which is heavier than urine; (2.) from the small amount of water (relatively) (GOODFELLOW).

5. There is nearly always *a frequent desire to micturate*, especially at night, when the patient is in the horizontal posture (WATSON), with a sensation of heat or scalding on passing water, accompanied with a discharge of mucus from the urethra, which possesses a more or less puriform character, and appears in the urine in the form of slight thin shreds (GOODFELLOW).

6. *Dryness of the skin* prevails, and the power of eliminating water by the skin is impaired. Heat of skin and general feverishness, with occasional chilliness of the surface, and a pulse above the natural standard, are common phenomena throughout the later stages of the disease, and when emaciation and debility are progressive.

7. *The Dyspeptic symptoms*, which prevail at an early period, indicate irritation of the gastro-intestinal mucous membrane. Loss of appetite, sometimes amounting to actual loathing of food, or a capricious and uncertain appetite, are amongst the most prominent phenomena. The food taken rests uneasily in the stomach, giving rise to stomachal and intestinal *pneumatosis* and acid eructations, the explosive force of the gas so generated being sometimes so great as to cause partial regurgitation of the food. *Gastralgia* and *pyrosis* may prevail; and there is very often nausea, retching, and sometimes vomiting, at occasional intervals. Attacks of diarrhœa are frequent, alternating with costiveness. When such phenomena are discovered to exist, the urine ought to be examined at once.

8. *Symptoms referable to the State of the Blood.*—The blood in Bright's disease being decidedly watery, and the red corpuscles deficient, the stimulus of healthy blood is not experienced by the heart; on the contrary, the morbid condition of the blood impairs the action of the heart and its circulation through the capillary blood-vessels. Palpitation is caused by very slight exertion, or by any mental emotion, and the heart beats *irritably*—the sounds being preternaturally sharp and abrupt, the rhythm disturbed, and the action irregular and intermittent. With the advance of the constitutional cachexia, the nervous and muscular structures of the heart are ill-nourished, and may become temporarily or permanently damaged; signs of pericardial effusion may occur, or even of pericardial or endocardial inflammation. The condition of the blood varies; but (1.) There is an excess of serum, the clot constituting no more than one-fourth part of the blood. (2.) The density of the serum returns to its normal standard, or even exceeds it; sometimes, however, it remains low, even in the advanced stages. (3.) The urea disappears as the disease advances, but reappears towards the fatal termination of the case, even in a larger amount than before. (4.) The fibrine, increased in the first stage, returns to its normal amount as the disease advances, and only becomes considerable again towards the close, especially if some of the intercurrent attacks of inflammation supervene, so common in

Bright's disease. (5.) The most remarkable change the blood undergoes is the great decrease of the red blood-globules. They are frequently reduced to *one-third* of the normal proportions; and this diminution progressively advances with the disease. "There is no disease of a chronic nature which so closely approaches hæmorrhage in its power of impoverishing the red particles of the blood as Bright's disease" (Sir ROBERT CHRISTISON). (6.) There are also present in variable, but always in considerable quantities, highly stimulating, irritating, perhaps toxic matters—pure excrementitious material—which can never be retained in the blood without more or less disastrous effects upon several great vital processes, and leading in some cases rapidly to death (GOODFELLOW). "The renal malady has a direct tendency, by its effect upon the blood, to generate disease of the heart. It induces anæmia, and thereby debility of the muscular texture of the heart, and leads to dilatation of its cavities. The weak muscle, becoming irritable also, grows thicker as it labours more" (Sir THOMAS WATSON). The experiments of Dr. Hammond, the late Director-General of the Medical Department of the Federal Army, have proved that urea, when retained in the blood, either by disease or extirpation of the kidneys, is sure sooner or later to kill. The blood, therefore, in Bright's disease, being poor, thin, watery, containing much less albumen, and fewer red corpuscles, than in health, and containing, moreover extraneous offensive matters in the form of urea and the extractives—more or less urinous excrement—is ill adapted to facilitate circulation, but, on the contrary, tends to retard its progress. Ultimately the blood is charged with further impurities. It begins to abound in fatty matter, especially cholesterine, which becomes deposited in several tissues, taking the place of their own proper elements of nutrition, and interfering with their function, as in the heart, the arteries, and the capillaries. "The body is poisoned in detail by the retention of its own excrements." The products of tissue waste and of superfluous peptones accumulate in the blood. The effect of this excess of waste products is to induce spasm of the muscular coats of the small arterioles of the system generally, the effect of which is to obstruct the flow forward of the arterial blood, and a rise in the blood pressure. Hypertrophy of the left ventricle is a consequent result. There is accentuation of the aortic second sound of the heart.

9. *Dyspnœa* is one of the earliest and most pathognomonic phenomena of chronic Bright's disease. It is quite apart from, and independent of, any bronchial disturbance in the first instance. The patient observes every now and then how short his breath has become. In an ordinary walk he finds he must stop to recover his breath. Occasionally the attacks occur at night, but chiefly during the day; and there is more or less palpitation during the paroxysms; while some wheezing at the chest may also attract the patient's notice. Weeks are thus apt to pass before the patient considers the symptoms sufficiently grave to seek medical advice (BASHAM). If the urine be examined at this early period, the presence of small quantities of albumen may be detected, and the sediment will contain granular casts, with more or less decayed cell-structures. Another cause of dyspnœa is the œdema which is apt to set in and pervade the base or more dependent parts of the lungs. It must also

be remembered that the lungs, the pericardium, and pleura are organs especially liable to the intercurrent attacks of inflammation in Bright's disease.

10. *Symptoms referable to the Nervous System.*—In the absence of anasarca these symptoms are of great importance when they occur coincidently with such other general symptoms as follow anæmia or puffiness of the face. Most of the phenomena are those present in *chloro-anæmia*—*e.g.*, a blowing noise, or a sensation of ringing noises in the ear, occasional dimness of vision, partial amaurosis, from paralysis of more or fewer of the papillæ of the retina, giving rise to the appearance of motes or small bodies floating before the eyes (*muscæ volitantes*). In some instances there may be flashes of light from irritation of the fibres of the optic nerve. Definite morbid changes are to be seen in the retina with the ophthalmoscope, such as hæmorrhage and retinitis (albuminuric). The loss of sight creeps on slowly and is permanent (see p. 524, *et. seq.*, *ante*). The more characteristic appearances are due to numerous white or yellowish-white brilliant looking spots or patches of irregular shape around the disc, with hæmorrhagic extravasations into the same locality. By union of the patches the disc may be surrounded with a white zone. Whitish streaks may also be seen radiating outwards along the vessels and nerves. Spots and hæmorrhages may also invade the disc. Both eyes are always involved (ALLBUTT). Throbbing of the temporal and other arteries, a sense of fulness or weight at the back of the head and in the nape of the neck, with a disposition to frequent cramp of the muscles of the back and sides of the neck; neuralgic pains in the face and head, or in other parts of the body; headache in the form of *hemicrania* or *megrim;* frequent attacks of giddiness, drowsiness, disposition to comatose sleep, and, in some cases, profound coma, alternating with convulsions, are all premonitory symptoms of chronic Bright's disease, and may be also fatal ones. The occurrence of what are called *head symptoms* may be frequent during the course of the disease, and death of the patient is so generally preceded by *coma,* with or without convulsions, that death by coma is almost the "natural termination" of Bright's disease, or the mode in which it proves fatal when life is not cut short by some other incidental or secondary affection (Sir ROBERT CHRISTISON). The pale and watery condition to which the blood is at last reduced may have something to do with the stupor and coma, as in *spurious hydrocephalus* similar phenomena are seen (Sir THOMAS WATSON). Giddiness is often a remarkable and characteristic phenomenon. A disposition to erysipelas has also been noticed. Anasarca is one of the earliest symptoms; in ninety-nine out of every one hundred cases it is the first intimation which the physician has of the disease; but if he could see the patient regularly before this symptom sets in, there are no doubt other phenomena, perhaps less definite, which he would have no difficulty in setting down as the probable antecedents of Bright's disease.

Diagnosis.—The specific gravity of the urine, the albumen it contains when persistent, and the amount of the solid constituents, are the first indications of the real nature of the disease. To determine the albumen, a small quantity of the urine in a test tube must be slowly and gently heated to the *boiling* point by the flame of a spirit-lamp, when, if albumen is present, it will appear in the form of a whitish cloud, of which the constituent particles multiply and collect, in proportion as the quantity is considerable, into small curdy fragments or flakes. These will gradually subside to the lower part of the tube when permitted to rest, leaving the supernatant liquor clear, and so indicate approximatively the amount of albumen present. A second specimen of the urine should be taken in another tube, and, after it has been thus boiled, an excess of *nitric acid* may be poured into the tube, when the albumen present will be precipitated in a flaky or pulpy form. This latter method is the best where the urine to be tested is alkaline; but both methods should be employed in every case, and they are sufficient to determine the presence of albumen. The following table, calculated by Dr. G. Bird, according to Sir Robert Christison's formula, affords results sufficiently accurate for the guidance of the student or practitioner, and shows at a glance the number of grains of solids, and the weight of a fluid ounce of urine, of every density from 1,010 to 1,040 :—

Specific Gravity.	Weight of 1 Fluid Ounce. Grains.	Solids in 1 Fluid Ounce. Grains.	Specific Gravity.	Weight of 1 Fluid Ounce. Grains.	Solids in 1 Fluid Ounce. Grains.
1010	441·8	10·283	1025	448·4	26·119
1011	442·3	11·337	1026	448·8	27·188
1012	442·7	12·377	1027	449·3	28·265
1013	443·1	13·421	1028	449·7	29·338
1014	443·6	14·470	1029	450·1	30·413
1015	444·0	15·517	1030	450·6	31·496
1016	444·5	16·570	1031	451·0	32·575
1017	444·9	17·622	1032	451·5	33·663
1018	445·2	18·671	1033	451·9	35·746
1019	445·8	19·735	1034	452·3	36·831
1020	446·2	20·792	1035	452·8	37·925
1021	446·6	21·852	1036	453·2	38·014
1022	447·1	22·918	1037	453·6	39·104
1023	447·5	23·981	1038	454·1	40·206
1024	448·0	24·051	1039	454·5	41·300

The amount of solid constituents may be determined approximatively at the bedside by the following formula:—If D = the density or specific gravity of the urine, and Δ = the difference between 1,000 and the density of the urine, the quantity of solids in 1,000 grains will be = Δ × 2·33 for diabetic urine, and by 2 for most cases. Supposing the specific gravity to be 10·20, then 20 × 2·33 = 46·60, which is the amount of solids in 1,000 grains of urine; or simply multiplying the last two figures of the specific gravity by 2 for most cases, and by 2·33 for diabetic urine, will give the amount *in grammes* of solids in 1,000 cubic centimetres of urine (Parkes). The formula of Trapp (the error of which, according to Vogel, cannot exceed one-tenth in healthy and one-fifth in morbid urine) is the best for urine *not diabetic*, and is as follows:—If Δ represents the excess of the specific gravity of urine above that of water, the amount of the solid constituents of 1,000 parts of urine will be represented by 2 Δ.

A sufficient approximation to the truth for clinical purposes is gained by such formulæ; but they can never supersede the exact processes necessary for scientific investigation (Christison).

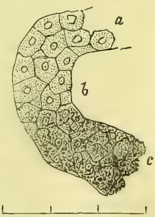
The hydræmia is indicated by the general paleness or anæmic appearance of the surface of the body; and even when no albumen can be discovered in the urine these phenomena are of great value in diagnosis. When they are observed in persons above the age of from thirty-five to forty-five, the physician should at once examine the patient and his urine closely from day to day, in order that he may detect as early as possible the other symptoms and conditions which are characteristic of Bright's disease. The specific gravity of the urine, the quantity passed daily, the amount of urea and of other solids contained in the daily discharge, should all be closely observed, and whether there be not slight puffiness of the eyelids in the morning, or indications of serum beneath the conjunctiva. The paleness is not a dry waxy paleness, as in chlorosis or anæmia-chlorosis. It is a paleness characteristic of dropsy.

When the disease is established, and its diagnosis confirmed, much information will be obtained as a guide to treatment, and as an aid to the diagnosis of the peculiar morbid condition in which the kidney may be by a daily microscopic examination of the urinary sediments. The specific cell-characters of the sediments have been found to be a more certain guide in prognosis than can be furnished by any other property of the urine, or by any other symptom exhibited by the patient. They undergo marked alteration and change as the renal disease advances. Under all circumstances it is difficult to form an opinion as to the rate of progress or advance of the lesion in the kidney; and hitherto the physician has been usually guided in his prognosis by the subsidence of the dropsy, or by the diminution of the amount of albumen in the urine. The microscopic examination of the cell-elements is now recognised as yielding more certainty to the opinion which may be formed as to the progress of the renal disease. The quantity of albumen present is an important point to be considered, along with the characters of the tubular or organic elements contained in the urine; and in private practice the amount of albumen passed is approximatively judged of by noting the *space* which the *coagulum* of albumen occupies in the tube after being allowed to rest. The phraseology recommended by Sir Robert Christison to express the proportions observed by the eye is as follows:—

1. *Gelatinous* by heat. 2. *Very strongly coagulable* by heat—nearly the whole tube. 3. *Strongly coagulable*—half the tube. 4. *Moderately coagulable*—one quarter of the tube. 5. *Slightly coagulable*—one-eighth. 6. *Feebly coagulable*—less than one-eighth. 7. *Hazy* by heat—no visible flakes of albumen.

The epithelial cells thrown off from the renal tubes, as well as the casts which accompany them, suffer material alteration as the disease of the kidney advances. These changes consist in deviations from the standard structure of healthy epithelium. The cells lining the straight tubes are the last to exhibit any character of degeneration; and in no case do the cells of the kidney-tubes appear in the urine except under the influence of disease. Any one single examination of the urine is not sufficient to determine the stage to which the disease has advanced. It is only by comparing the character of the deposit of one period with another, and noting carefully the altered appearance of the casts and cells, and the direction of whatever change is taking place, that an approximation of more or less certainty is made regarding the progress of the disease.

The sediment found in albuminous urine consists principally of cylindrical forms moulded in the uriniferous tubes of the kidney. These present different characters according to the stage of the disease or the intensity of the morbid process. They may be either coarsely granular, finely granular, or partly granular and transparent, or completely hyaline. Associated with these casts are various cell-structures—blood-cell, epithelium-cell, compound granule-cell, and various modifications of cell-growth, as may occur during the advance of acute or chronic disease. When hæmorrhage takes place in the uriniferous tubes from rupture of the Malpighian capillaries, the coagulum formed is washed away, and appears in the urine as a cylindrical cast of a granular appearance, entangling a number of blood-cells within its mould. These casts are usually stained of a red colour by the hæmatine of the effused blood. As the hæmorrhagic state subsides, epithelial cells appear in the cast, mixed with blood-corpuscles, which gradually become less numerous, and ultimately disappear. In favourable cases the casts become more and more finely granular, and daily more transparent, and the epithelium-cells less and less abundant. In less favourable cases various modifications of cell-development occur; resplendent granules appear, and free nuclei accompany the cast; compound granule-cells are discharged, abortive epithelial cells appear, with disintegrated granule-cells in the shape of grape-like clusters of nuclei; and other evidence of cell-transformation indicates the nature and direction which the renal disease may be taking. It is the *progressive* alteration of cell-formation which accompanies the casts of albuminous urine which must be watched and examined, day by day, to ascertain the progress of the disease. The first departure from the healthy type of the secreting kidney-cell is evinced by its becoming more granular, the single nucleus being either obscured or accompanied by other nuclear granules. The cells become larger and larger, and present a cloudy appearance; for a larger quantity of material than usual has been taken up by the cells. The entire uriniferous tubules are thereby rendered broader, and to the naked eye the tubes look white and opaque. The individual cells are difficult to isolate, because they adhere closely together, in consequence of the alteration of cohesion they have undergone. The few granules normally present in the cells have accumulated and multiplied in greater and greater numbers the greater the energy with which the morbid process is being carried on, so that even the nucleus gradually grows indistinct. This is the condition described by Virchow as that of *"cloudy swelling"* (trübe Schwellung), Fig. 73. It is the progressive and constant increase of such compound granule-cells in the urine, or of cells becoming more degenerate and compound, with a proportionate decrease of epithelium, which is the index of advancing disease. The occurrence of



1,000 of an inch × 350 diam.

Fig. 73.*

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* Convoluted urinary tubule from the cortex of a kidney in Bright's disease (after VIRCHOW). (*a.*) Tolerably normal epithelium; (*b.*) state of *"cloudy swelling;"* (*c.*) commencing fatty degeneration and disintegration.

such cells is usually preceded by a state of inflammatory congestion; and in proportion to the quantity of these cells, and the degree of degeneration to which they have advanced, so is the degenerative process in the kidney indicated. When groups of grape-like (botryoidal) clusters of resplendent granules appear in the urine, they are generally the nuclei of various cells, and indicate a deteriorated or atrophic state of cell-development which eventually becomes so powerless that nothing remains but a heap of granules. These accumulating in the tubes and Malpighian bodies, eventually constitute a fatty degeneration of the whole kidney. The occurrence of such clusters in the urine is generally associated with a subacute process of disease; and when the clusters become daily more numerous, they furnish very unfavourable indications. The large compound cell or inflammation-globule of Gluge never makes its appearance except as a sequel to a state of hyperæmia. There is a period in favourable cases where little or no epithelium is visible, and where transparent hyaline or waxy casts are alone seen. They are more abundant and more frequent in the chronic form of Bright's disease than in the acute; and are then present in the urine of the mildest and most tractable cases equally with the severest and most fatal. Their particular import depends on the cell-structures with which they are associated, and likewise on the relations as to time which other kinds of casts bear towards them. Although they have been named hyaline, yet occasionally they contain traces of granular matter or cell-elements. If they appear in the urine very shortly after hæmaturia, they may be accepted as indicating a state of catarrh of the renal tubes, which, in all probability, will prove remediable and tractable. In such cases an epithelium-cell is seen here and there in the urine, and sometimes a compound granule-cell; but these are not numerous, and do not increase. In other cases the glairy moulds of the tubes contain numerous abortive cells in almost daily increasing numbers. Compound granule-cells are then always present, clusters of granules (botryoidal or crescentic), and free nuclei are seen. These appearances are characteristic of a chronic subacute process, which progresses slowly, very insidiously, and is too often irremediable.

In a third variety of Bright's disease where these hyaline casts are seen, few or no epithelial cells are present, but the appearances are characteristic of broken up cell-structures; clusters of granules are seen, having more and more the appearance of fat granules. Many acquire a large size, and contain oil in abundance. In the more advanced cases the casts seem made up of these fatty and oily materials, and hence they have acquired the name of fatty or oily casts. They indicate the highest state of fatty degeneration.

There is still a fourth variety of the hyaline or transparent cast, and which, from the absence of all structure visible in it, has received the name of waxy cast. This cast may be formed either in the smaller convoluted tubes, or in the larger straight tubes of Bellini. They seem to be composed of some viscid material, and become faintly granular on the addition of dilute acetic acid.

The following is a general summary of the results relative to casts in the urine in Bright's disease:—(1.) *The blood casts* represent more or less active hyperæmia and hæmorrhage from the kidney. (2.) *The coarsely* *granular epithelial cast,* with its compound inflammation-corpuscle, accompanied by amorphous granular flakes stained with hæmatine, represent the period of inflammatory exudation. (3.) *The finely granular semi-transparent casts,* with scattered epithelium and granule-cells, represent the period of subsidence of the inflammatory process. (4.) *The transparent casts,* with compound cells, or with isolated transparent molecules and grape-like clusters of granules, represent a stage of chronic subacute disease of very grave import; and if these casts become more and more loaded with large and gradually increasing fat-granules and oil-drops, the progress of fatal fatty degeneration is clearly marked (BASHAM).

Deteriorating conditions such as have been here described are not limited to the kidney in Bright's disease. There is reason to believe that the nutrition of most of the textures and organs of the body proportionally fails; and although not so apparent, because not manifested in a manner capable of being demonstrated during life, yet the tissues of every organ become more or less degenerate and inefficient for the purposes of life. The cells of the liver are invariably loaded with an abnormal amount of fat in all fatal cases of Bright's disease; and the heart-fibre and arterial textures exhibit the microscopic characters of atrophy and granular or fatty degeneration. Inflammation of the serous surfaces is a common complication; and of the cutaneous surface, as erysipelas.

The origin of chronic Bright's disease cannot be traced with certainty in all cases. It is one of those diseases, like phthisis, in which the patient rarely applies to the physician till the disease has made considerable advances, often beyond any remedy or means of cure. The commencement of it cannot be recalled or described by any other terms than "a gradual failure of the general health," or as "a breaking-up of the constitution." If the urine be examined at the earliest stage, the presence of small quantities of albumen may be detected, and the sediment shows examples of granular casts, with more or less decayed and broken up cell-structure. These phenomena often manifest themselves long before any dropsical symptoms point to the existence of renal lesions. There are also exceptional cases in which the urine at this early stage is non-albuminous. It is important also to bear in mind the relation of the hyaline, transparent, and waxy casts to the early and remediable forms of Bright's disease. In favourable cases such casts become more and more translucent; but if the disease advances, numerous cell-forms occur, which are passed along with the casts; and when the casts continue to present, week after week, a great number of free nuclei, associated with hyaline cylinders—numerous compound granular cells, some with, some without, cell-walls—the progress of renal degeneration in the most obstinate and intractable form is fatally progressing. Without a microscopic examination of the urine from day to day it is impossible to distinguish between a case likely to improve under treatment, and one which may be viewed as hopeless; and without the daily use of the microscope the treatment becomes, at the best, but merely guess-work.

Treatment.—(*a.*) Of *Acute Bright's Disease*—(1.) Relieve the kidneys as much as possible from the labour of elimination, by avoiding exposure to cold, by keeping the patient at rest in bed, in a room of moderate uniform temperature. (2.) The food should be scanty, consisting of gruel, arrow-root, milk, or weak broth. Pure water is the best drink, and alcoholic fluids are not to be taken on any account. (3.) Free action of the skin and bowels must be maintained. The *hot air bath* and antimonial remedies are the best agents to effect the first of these conditions, and free perspiration is to be encouraged by bedding the patient in blankets. *Antimonial wine* may be given in doses of from fifteen to thirty drops every four or five hours. The bowels are to be kept open by the *compound jalap powder,* in doses of twenty to sixty grains, repeated daily or on alternate days. It may be alternated with *podophyllin,* or with extract of *colocynth.* Mercury is not to be given. (4.) *Cupping over the loins* relieves pain in the back, and the quantity of urine passed generally increases after eight or ten ounces of blood have been withdrawn in this way from an adult, or two or three ounces from a child three or four years old. (5.) When the tongue becomes clean and the general symptoms improve, mutton broth or good beef-tea may be indulged in; and, as the digestion improves, solid food may be eaten in small quantities, beginning with fish and fowl, and afterwards mutton or beef. (6.) Flannel must be worn next the skin. (7.) *Iron* is of great service during convalescence, for in such cases the anæmia becomes extreme. *Phosphate of iron,* in the form of *syrup,* or *citrate* of *iron* and *quinia,* or the *ferrum reductum,* are the most digestible forms, and they ought to be given in small doses repeated after every diet. (8.) *Diuretics* are not to be given. In a case where the urine was suppressed, fomentations, consisting of *infusion of the leaves of digitalis,* were found by Christison and Vogel to increase enormously the amount of urine. Parkes found the amount of the albumen to diminish markedly from the use of the *tincture of the sesquichloride of iron* (l. c., p. 379).

(*b.*) Of *Chronic Bright's Disease,* general principles only can be indicated, inasmuch as every case requires a special study, and a line of treatment in detail peculiar to itself. Whatever treatment be adopted, a long time is necessary before any appreciable results are obtained. It is, therefore, necessary to persist in one line of treatment steadily from week to week, and even from month to month; and it is obviously of great importance to be as accurate as possible in diagnosis as to the probable state of the kidney, so as to define the line of treatment from the first which may seem best adapted for the individual case.

It is a question of grave importance how far vomiting or diarrhœa ought to be checked. If either of these occurrences are suddenly stopped, the gastric and intestinal membrane acting at the time as an emunctory for the urea and other excreta of the urine, the patient may be suddenly cut off by convulsions, apoplexy, or effusion into some of the serous cavities, such as the pericardium, or the pleuræ, or the ventricles of the brain. It is necessary, therefore, in the first instance, to determine in all chronic cases the particular organ or tissue which seems in each case to be acting vicariously. The perspirations are often spontaneously profuse; and the skin is by far the safest emunctory for the vicarious elimination of urinary constituents. Therefore it is important to promote the action of the skin if it be deficient, and to encourage it even if it is already considerable. *Diaphoretics* are always of essential service. The best are *Dover's powder,* the warm bath, warm clothing, and, for convalescents especially, a moderately warm climate. In Sir Robert Christison's experience diaphoretics have always appeared most serviceable when they are so given as to excite a gentle perspiration during a part of the night. So it is also safe to promote the discharge of secretions from the intestinal canal, with due caution that they do not become excessive, so as to pass into permanent diarrhœa. Urea and other constituents of the urine are found in such discharges in large proportions. When general anasarca prevails, absorption may be promoted by gentle pressure, which at all times must be very cautiously applied, and the effects closely watched, for such effusions afford great temporary relief to important symptoms which indicate the involvement of vital organs. Bandaging to promote absorption is not justifiable so long as the anasarca is increasing. Patients ought to be encouraged to go about as long as they are able, care being taken that they are clothed with flannel and woollen garments, and otherwise well protected from chills or draughts of cold air.

In the subacute forms of the disease the action of the skin is especially to be promoted by such saline remedies as the *acetate* or *citrate of ammonia*, to which may be added *one*, *two*, or *three drachms of the infusion of digitalis*, and *ten* or *fifteen minims of antimonial wine.* In such cases, also, *ten* or *fifteen minims* of the *tincture of the perchloride of iron*, or from *five* to *ten* grains of the *citrate of iron and ammonia* may be given *every day* with one of the meals. *Every second day* a dose of the *compound jalap powder* may be given; and if hyperæmia of the kidneys prevail, it may be necessary to take from four to six ounces of blood from the loins by cupping or by leeches. As the urine becomes more free from blood-corpuscles and albumen, the iron medicines may be more frequently given, and the compound jalap powder less frequently. A good formula for the administration of salines and iron in the subacute and chronic cases is that given by Drs. Basham and Goodfellow, as follows:—

Liquor Ammoniæ Acetatis, ℥ii.; *Acidi Acetici diluti*, ℥i.; *Tinct. Ferri Perchloridi*, ʒiv.; *Aquæ*, ℥iss.; *misce.* Two teaspoonfuls for a dose.

Small doses frequently repeated seem to do better than larger ones given at longer intervals. These remedies tend to lessen the watery state of the blood; and the action of the chalybeate medicines is of no avail till after purgation has been free, and when the hot air or warm baths have caused the skin to act freely. A nutritious diet is then to be given, combined with the chalybeate remedy.

The quantity of urea passed by the urine should be determined day by day, to ascertain how far the kidneys are capable of secreting and eliminating these excrementitious products. According to the results obtained the diet must be regulated, and such measures taken as are calculated to reduce the quantity of urea, and other constituents formed daily, to the capacity of the diseased kidneys for the work they are able to do.

The further treatment of the chronic forms of Bright's disease is influenced by the amount of the anasarca present. If considerable anasarca is present, neither cupping nor leeches can be had recourse to, on account of the bruising and erysipelatous inflammation they are apt to induce. The chalybeate mixture already mentioned is still found of service, taken at least three times daily, and to it may be added from time to time *fifteen* or *twenty* minims of the *spiritus ætherus nitrici* (GOODFELLOW). The *citrate of iron and ammonia*, in from *five* to *ten* grain doses, with the *sulphuric* or *chloric ether*, are also good and useful remedies; and if the iron medicines are found too stimulating, *sulphate of zinc*, or *tannic* and *gallic acids*, may be used instead. The *sulphate of zinc* is to be given, in doses of *one* to *three* grains three times a day, in the form of a pill, made with *extract of hop*, and with or without a grain of the *extract of nux vomica;* or the same dose of *sulphate of zinc* may be given in a draught combined with *sulphuric* or *chloric ether* (GOODFELLOW). These latter medicines relieve considerably the flatulence and sensation of coldness in the stomach and bowels, so much complained of in such cases.

If the urine is "smoky," or if blood is seen on microscopic examination, the *gallic acid*, in *five* or *ten grain* doses, may be given, with a few drops of *diluted sulphuric acid* and a few drops of the *tincture of hops*, or other aromatic vegetable tincture, in an infusion of the same. The objection to these remedies is the constipation they are apt to induce. The bowels should always be kept relaxed, two or three loose evacuations being secured daily. The medicine most generally useful is the *compound jalap powder* of the London Pharmacopœia, which should be taken in the morning fasting, in half drachm or drachm doses, in a wine-glass of water. It does not induce prostration, but by repetition it is apt to lose its effect, when *elaterium* may be necessary in small but repeated doses; *two grains of elaterium* being dissolved in *sulphuric ether*, and *the eighth part of a grain* given every six hours till the desired effect is produced. Very abundant watery purgation is thus obtained, and a sensible impression is made upon the distended dropsical parts (BASHAM). If *elaterium* may not be deemed advisable, a saline draught of the following composition may prove efficient:—

℞. *Magnus. Sulph. vel Sodæ Sulph.*, ʒi. to ʒij.; *Ætheris Sulph.*, ♏ x.; *Acid. Sulph.* dil., ♏ x.; *Ferri Sulph.*, gr. i. to ii.; *Aq. Menthæ Vir.*, ℥iii. to ℥iv.

This draught is to be taken the first thing in the morning once or twice a week. It ought to produce two or three loose and watery evacuations (GOODFELLOW).

Diuretics are a most certain and speedy remedy when the *dropsical* effusions are considerable; and they are sometimes advisable when the urine is scanty, although they are seldom necessary in the treatment of the fundamental disease. Sir Robert Christison is convinced that the fears entertained by some of injury being produced by the stimulus of diuretics on the kidneys are visionary. In his experience, over more than forty years, the best diuretics are *digitalis*, *squill*, and *bitartrate of potash*, taken simultaneously; and, if these fail, Hollands spirits sometimes speedily establishes the diuresis.

When dyspeptic symptoms predominate, and there is considerable flatulence, the following pill is recommended by Dr. Goodfellow to be taken twice or thrice daily:—

℞. *Ferri Sulphatis*, gr. i.; *Ext. Nucis Vomicæ*, gr. $\frac{1}{2}$ to i.; *Mas. Pil. Galb. Co.*, gr. ii. to iii.; *misce, fiat pilula.*

And if there be co-existent bronchitis, a draught of the *acetate of ammonia*, with *ten, fifteen*, or *twenty* drops of the *spiritus ætheris nitrici*, and half a drachm of the *oxymel of squills*, is to be taken intermediately with the pills. If much nausea prevail, *three minims* of *dilute hydrocyanic acid* may be added to the draught, with the occasional application of mustard to the stomach.

Want of sleep is often complained of, but opiates are inadvisable, because they are apt to check the secretions and to occasion constipation. *Henbane* may be given in place of *opium*.

The patient should select a residence where the soil is sandy or chalky, where the air is mild and dry, so that as much open-air exercise may be taken as possible.

Diet should be light, but nutritious; no pastry should be eaten; and stimulant liquors must be used with caution, although, when exhaustion is great, their use may be unavoidable. The principal meal should be in the middle of the day, and not later than three o'clock; and the last meal should be taken two or three hours before bedtime, retiring early to bed, and rising early. Light but warm woollen clothing is to be always worn —in summer as well as in winter.

The complications of Bright's disease are extremely difficult to manage. The *diarrhœa* must not be suddenly checked. Thirty to sixty minims in water of the *spiritus ammoniæ aromaticus*, with half a drachm of the *tincture of kino* or of *catechu*, after every loose stool, will in general be all that is necessary. If there be much griping, the application of a linseed poultice over the abdomen, with two drachms or half an ounce of *tincture of opium* sprinkled over it, will give relief (GOODFELLOW). When it is found necessary to use measures for checking the diarrhœa, means should be taken that the skin acts freely, or that the urine flows increased in quantity. Lead and opium pill (*pilula plumbi cum opio*), in doses of five or ten grains twice or even thrice a day, is the best remedy for checking diarrhœa. These remedies may be aided by a *suppository* of the *hydrochlorate of morphia* occasionally, each suppository containing a quarter of a grain of the hydrochlorate of morphia (*morphiæ hydrochlor. suppositoriæ*). Vomiting may be controlled to some extent by *bismuth*, but more frequently by *morphia, hydrocyanic acid, creasote, rectified pyroxylic spirit, chloroform*, or *chlorodyne*, or little fragments of ice; and if these fail, a blister over the epigastrium has sometimes succeeded (Sir ROBERT CHRISTISON).

Intercurrent inflammatory attacks and effusions into cavities are still more difficult complications to manage. The application of a few leeches, or the abstraction of a few ounces of blood by cupping, may be borne well in the cases with head complications; but the greatest care and caution are required in the employment of similar remedies in the complication of *pericarditis*, on account of the great danger of death by syncope.

Bronchial complications are serious, and more or less constantly present. They are frequently the immediate cause of death in chronic Bright's disease. The least stimulating expectorants may be administered under such circumstances. The following formula is recommended by Dr. Goodfellow:—

℞. *Liquor Ammoniæ Acetatis*, ʒii. to ʒiii.; *Spiritus Ætheris Nitrici*, ♏ xx. to ʒss.; *Oxymellis Scillæ* (Lond.), ʒss.; *Aquæ Camphoræ et Aquæ*, āā ʒv.; *misce, fiat haustus.* To be taken every four, six, or eight hours.

If the expectoration be viscid and difficult to discharge, a few drops of antimonial wine may be added to the draught; or if there be much spasm of the bronchial tubes, as indicated by the asthmatic breathing, a few drops of *sulphuric* or *chloric ether* may be given. If, on the other hand, the expectoration be purulent and difficult, a *few grains of carbonate of ammonia* may be given with the *oxymel of squills.* Every precaution ought to be taken to prevent a fresh attack of bronchitis. So long as the patient is able to bear it, the *habit* of cold sponging and dry rubbing of the surface of the body every morning with a flesh brush or coarse towel is the best preventive, and being clad in warm woollen clothes. Flannel next the skin must be invariably insisted on.

Mercurial preparations are contraindicated in Bright's disease—so great is the tendency to salivation and the otherwise injurious effects they produce. *Podophyllin* (the active principle of the May apple) may be used instead. Its dose is from $\frac{1}{4}$ to 1 grain; and it ought not to be given in doses larger than one grain. It is best given in a fluid form (see p. 902, *ante*), or in a pill combined with soap and hyoscyamus. As an occasional mild aperient pill, the following is found to answer well:—

℞. *Mas. Pil. Rhei. Comp.*, gr. ii. to iii.; *Vel. Ext. Aloes, Aquosi*, gr. i.; *Ext. Nucis Vomicæ*, gr. i.; *Mas. Pil. Galb. Co.*, gr. ii.; *misce, fiat pilula* (GOODFELLOW).

SUPPURATIVE NEPHRITIS—RENAL ABSCESS.

LATIN EQ., *Nephritis Suppurans;* FRENCH EQ., *Néphrite Supurée;* GERMAN EQ., *Interstitielle Nephritis;* ITALIAN EQ., *Nephritide Suppurativa.*

Definition.—"*Inflammation, with suppuration of the substance of the kidney.*"

Pathology.—(*a.*) *Causation.*—The kidneys may suppurate—(1.) From morbid conditions of the blood, as from the excessive use of stimulant, alcoholic, or terebinthine fluids, or the use of cantharides and of acid diuretics; (2.) from external violence; (3.) from retention of urine; (4.) from calculi in the kidney, or other sources of direct irritation; (5.) *extension of inflammation and suppuration* by continuity, as from the bladder or urinary passages; (6.) *embolism;* (7.) the result of *general pyæmia* or *septicæmia;* (8.) the extension of inflammation from neighbouring parts (JOHNSON, *op. cit.*, p. 417).

(*b.*) *Morbid Anatomy.*—It is of importance, in studying the inflammations of the kidney, to determine—(1.) The tissue or tissues affected—

vessels, parenchyma, or interstitial connective-tissue; (2.) whether only the cortical substance, or the pyramidal, or the pelvis of the kidney, is affected, or all parts together; (3.) whether the affection is partial or diffused. In the commencement of the disease a definite tissue can generally be indicated by microscopic examination after death as the starting-point of the affection; but later it is not so easy. The several tissues are often consecutively attacked. There are two essentially different affections—(1.) *Parenchymatous nephritis;* (2.) *Interstitial nephritis;* and these may be complicated with the fatty degeneration or the lardaceous disease already noticed. All forms of lesion may be present at once, sometimes one and sometimes another occurring first; but *parenchymatous nephritis* is most generally the primary affection (RASMUSSEN, *Med.-Chir. Review,* July, 1863). The cells of the convoluted tubes appear to be larger and richer in albumen than those in the straight ones; and any disease of the former rendering them inactive, is of far more serious import than disease of the latter, producing an actual change in the urine. Affections of the pyramidal portion of the kidney only are usually of a catarrhal nature, and are described as "*papillary catarrh,*" or "*catarrhal nephritis.*" The catarrh affects principally the straight canals and papillæ, and may be continued from the bladder and urethra. It may be the starting-point of *parenchymatous nephritis,* and often complicates it. Such cases are generally suppurative, especially when secondary to bladder inflammation, or paralysis of the bladder in spinal affections. The suppuration is then generally most obvious and active in the cortical portion of the organ. The kidney is enlarged, and its surface covered with minute points of pus or small abscesses diffused throughout the cortical, to the exclusion of the pyramidal part. The mucous membrane of the pelvis is inflamed, and is described under the name of *pyelitis* (WILKS). When the capsule is highly vascular and involved in the process, it indicates a chronic affection, or is a result of a former inflammation. In cases of uncomplicated catarrhal nephritis, where suppuration does not occur in the cortical portion, the lesions are confined to the canals and papillæ. They present a whitish or yellowish striation, with hyperæmia of the intervening vessels, and bloody ecchymosis may be present over the whole kidney, especially if acid diuretics have been largely given. If the disease continues long, the distended urinary canals press on the blood-vessels, when the hyperæmia generally ceases. The productive effects of the inflammation are limited to cell-growth (nucleated, club-shaped, fusiform, or ramifying), mixed with mucous catarrhal products (mucin), sometimes combined with fatty metamorphosis or other degeneration of epithelium.

True parenchymatous nephritis is a hypertrophy or "cloudy swelling" of the large cells of the convoluted tubes. The cells take up large quantities of the *albuminates,* becoming distended, turbid, granular, and closely adherent to each other. Subsequently all the cell-forms vanish, and a large granular fatty mass is set free, which generally forms into "inflammatory globules." It often co-exists with "interstitial nephritis." When catarrh of the tubes exists, it is usually associated with the symptomatic phenomena of Bright's disease. Retardation of venous blood in the kidney is a common event in parenchymatous nephritis. *Thrombi* are then apt to form in the veins of the kidney, and being transported to the *vena cava* and heart, may find their way into the lungs. In the advanced stage of the disease, resolution or recovery corresponds to fatty degeneration of the cells, sometimes with loss of substance apparent on the surface, indurations, granulations, cysts, in connection with or separated from the urinary canals. The interspaces between the granular elevations consist of empty collapsed tubes. The granular cirrhotic appearance of the kidney is not similar in pathological origin to that of the liver. In the liver it is the interacinous connective-tissue which is first affected and causes the cirrhotic granulations; in the kidney, on the contrary, the parenchymatous cells of the tubes become first affected, and the lesion is afterwards complicated with *interstitial nephritis.* The Malpighian glanduli ultimately become small, corrugated, and surrounded by thickened capsules of connective-tissue. These capsules, with the epithelium, may eventually become fatty, lardaceous, or calcareous. With regard to *interstitial nephritis,* it has been doubted whether there is a connective-tissue or fibrous matrix to the kidney. I have never yet seen a human kidney in which it is not demonstrable. It becomes immensely hypertrophied, and minute cells grow up in its substance in large numbers. Atrophy is not uncommon at the expense of the cortical part. On section it is seen to be diminished between the pyramids and the surface. The edge is irregular, and the surface is granular. The granulations are composed of bundles of tubes lying between veins, and are not to be confounded with white specks of deposit. The capsule separates with great difficulty, tearing the tissue which adheres to it. The glomeruli appear to be much closer together than natural (DICKENSON, *Med.-Chir. Trans.*, 1863). Cysts are frequently met with on the surface. Sometimes the whole of the new interstitial material undergoes the fatty degeneration. Granulations are also formed by the connective-tissue contracting round the canals and glomeruli, and circulation is more or less obstructed. The tubes become constricted in a bead-like manner, and the *tunica propria* is thickened and streaked. The glomeruli become small, homogeneous, and more or less fatty.

Circumscribed interstitial nephritis often affects the pyramids only, as a result of syphilis; nodes are the consequence, which, passing away or diminishing, leave cicatrix-like depressions, not unlike those from hæmorrhagic infarctions (RASMUSSEN, WILKS, VIRCHOW, DICKENSON).

Symptoms.—Uncomplicated acute nephritis has hitherto been considered so rare that its symptoms have not been well described. Those mentioned by Baillie are more or less pain in the region of the kidneys, which commonly shoots along the ureters. There is a sense of numbness down the thigh, and in the male there is often retraction of the testicle, or a feeling of pain in it. When one kidney is affected, these symptoms are only felt on that side. The urine is voided frequently, and is sometimes of a pale, but more commonly of a deep-red colour. There are sicknes and vomiting. The bowels are at the same time often costive, and subjec to colicky pains. These symptoms are accompanied by more or les fever. When pus is formed, the event may be known by the repeate shiverings, and by the pus being mixed with the urine. Cases related b Mr. Stanley, however, by no means bear out this description. He give the case of a man who had retention of urine in consequence of a gonorrhœal discharge being stopped by injections. In this instance the kidneys were found extremely vascular and soft, with numerous minute depositions of pus throughout the cortical and tubular parts, and the infundibula and the pelvis were likewise filled with pus. The principal symptom was severe pain at the fifth lumbar vertebra. In another similar case, but not quite so acute, the kidneys were found so dark-coloured as to be almost black, and at the same time remarkably flaccid. This patient died paraplegic, the loss of motion being complete, and that of sensation nearly so. If *nephritis* passes to a chronic suppurative state, the pain in the loins is often severe and the appetite impaired, while pus may be found to a considerable amount in the urine; and if a calculus or gravel be the immediate cause, the urine may contain large portions of those substances mixed with blood. Dr. George Johnson has shown that the desquamative and suppurative processes in the kidney are much more closely allied than is generally supposed. They pass into each other by almost imperceptible gradations, the products appearing in the urine as purulent casts of the tubes.

Prognosis.—Acute affections of the kidney are of grave prognosis. The chronic forms are perhaps consistent with life, but in every case they greatly impair the functions of life, and are ultimately the cause of premature death. When pus-corpuscles take the place of renal epithelium, the prognosis must always be unfavourable; and the suppurative process in the kidney is the most rapidly destructive of the lesions. Dr. Johnson has not met with a case of suppurative nephritis which has terminated in recovery (*op. cit.*, p. 440).

Treatment.—The treatment of acute nephritis must be according to the usual principles of the treatment of inflammation, by keeping the patient at perfect rest, the application of hot fomentations and poultices, preceded or not by the local abstraction of blood or by dry-cupping. Blisters in such cases are dangerous, and ought to be avoided. The neutral salts, with opiates, are admissible in some cases, especially where calculi or gravel are sources of irritation. *Castor-oil*, or other purgative substances which do not act so immediately on the kidneys, are more useful, such as *compound jalap powder* or *podophyllin*. Should signs of abscess appear, the pus may be evacuated, as in the case of liver abscess, and with the same antiseptic precautions.

HÆMATURIA RENALIS.

Latin Eq., *Hœmaturia Renalis;* French Eq., *Hématurie Renalis;* German Eq., *Bluthamen*—Syn., *Nierenblutung;* Italian Eq., *Ematuria Renale.*

Definition.—*Hœmorrhages in which blood is mixed with the urine, whether it proceeds from the kidney, ureter, or bladder.*

Pathology.—(*a.*) *Causation.*—The usual causes of hæmaturia are, blows on he back or loins, the existence of renal or vesical calculi; also granular degenration of the kidney, suppurative nephritis, disease of the bladder, cancer, mbolism, parasites, and some morbid poisons, as that of *small-pox*, or of general blood diseases, such as *rheumatism* or *scurvy.* *Cantharides* and the *turpentines* also act specifically in the production of hæmaturia, by inducing active hyperæmia or congestion of the kidney. Villous cancer of the bladder may account for hæmaturia; and so may vicarious menstrual discharge.

(*b.*) *Morbid Anatomy.*—We are often unable to trace whether the blood passed with the urine has flowed from the *kidney, ureter,* or *bladder.* When hæmorrhage has taken place from the kidney, a small clot may remain after death, to mark the seat of lesion; also when it proceeds from the bladder, the coats of that viscus may be red, congested, and some blood may exude from them on pressure. The most usual organic diseases with which hæmaturia is associated are, *Bright's kidney,* especially *acute desquamative nephritis, fungus hæmatodes,* either of the kidney or bladder, *nephritic* and *vesical* calculi, and cancer of the bladder.

Intermittent hæmaturia is also a recognised morbid condition (Rayer, Elliotson), generally associated with malaria.

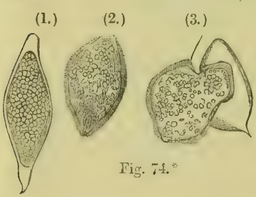

Fig. 74.*

Dr. John Harley has described a form of hæmaturia, already referred to in connection with the occurrence of the *distoma hæmatobium.* Its ova are then found in the urine; and its occurrence is endemic in Egypt, South Africa, and the Mauritius (Fig. 74).

Symptoms.—The hæmaturia may take place suddenly, or it may be preceded for a short time by pains in the loins, epigastrium, or bladder; and a burning pain is experienced on passing urine, which contains more or less blood. Sometimes the blood is deposited in clots in the bottom of the vessel, but more frequently it is thrown down in small portions of fibrine, like the sediment from beef-tea. In *renal hæmaturia* the urine is always of a smoky hue, or of a black colour, and albuminous. The blood is equally mixed with the urine in such cases, and hence is presumed to come from the kidney. Casts of renal tubes also indicate kidney lesion (blood casts). In grave cases, especially in old persons with disease of the prostate, the blood may coagulate in the bladder, and render it both necessary and difficult to pass the catheter. In hæmorrhage from the *pelvis* or *ureters,* the blood and urine are also intimately mixed; and there may be also moulded vermiform coagula of considerable length. In hæmorrhage from the bladder the blood is discharged chiefly or towards the end of the act of micturition; and when it comes from the urethra, it may escape or be pressed out independently of the urine, the first portion of which may be coloured from the blood which it forces out.

The hæmaturia often continues for many days, and even weeks, together, and the quantity of blood passed, though often trifling, yet occasionally amounts to some ounces in the course of twenty-four hours. The general symptoms depend, as in other hæmorrhages, on the quantity of blood lost.

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* (1.) Ovum of *distoma hæmatobium,* from hæmaturia of the Cape of Good Hope (Dr. John Harley); (2.) embryo (ciliated), from ovum capsule; (3.) embryo attached to the ovum capsule.

Diagnosis.—No certain symptom has yet been observed by which we can determine the particular seat of the hæmorrhage. We should be careful not to confound urine greatly loaded with uric acid with hæmaturia.

Prognosis.—Idiopathic hæmaturia is rarely a grave disease, except it arises from disease of the kidney, such as *carcinoma*, or other structural disease of the urinary organs, when it is the precursor of a fatal event.

Treatment.—*Tincture of the perchloride of iron* is one of the most useful of remedies, whether in debilitated patients or in those suffering from Bright's disease, and whether the blood comes from the kidneys or bladder. It is best given in doses of ♏x. to xx., three or four times daily, in combination with *glycerine;* and if there be much *cardiac* difficulty, with arterial excitement, it may be combined with *digitalis*, as in the following prescription:—

℞. *Tinct. ferri perchloridi*, ♏xxx.; *Tinct. digitalis*, ♏xv.; *Aq. menth. pip.*, *f*℥iss., repeated every four hours.

Gallic acid, in combination with *sulphuric acid*, may also be given; as also *ipecacuanha*, *tannin*, *acetate of lead* and *alum*, and *ergotine*, as already referred to under Hæmatemesis. *Quinine* and *arsenic* are remedies indicated in malarious intermittent cases. Idiopathic hæmorrhage often readily yields to *bitartrate of potash*, or to the *mineral acids.* Dr. Elliotson recommends, as also did John Hunter, the *ol. terebinthinæ*, in doses of ♏x., ♏xx., or ♏xxx., every two, or three, or four hours. *Injections of cold water* (or water in which twenty to forty grains of *alum* have been dissolved) into the bladder or up the rectum, and also a cold hip-bath, are useful applications. Dry-cupping over the loins is also indicated.

PAROXYSMAL OR INTERMITTENT HÆMATINURIA: PERIODIC HÆMOGLOBINURIA.

Definition.—*A condition in which the urine contains only the colouring matter of the blood with albumen, but no blood-corpuscles nor fibrine, coming on in sudden and irregular paroxysms.*

Pathology.—(*a.*) *Causation.*—It has been attributed to the influence of cold of sufficient intensity, or to getting wet. It has occurred after scarlet fever, and in connection with exposure to cold. There are also certain premonitory peculiarities in the recorded cases of this disease which point to a nervous rather than a nephritic origin, as severe neuralgia, sometimes of the trunk and sometimes of the extremities; also severe formication, quickly followed by lassitude and cold shivering (Robert and Knessner, *London Med. Record*, Dec. 15, 1878, p. 511): the occurrence of urticaria developed by the application of cold water, but not produced by tracing lines in the skin, or by any other form of mechanical irritation. It has also been found associated with the rheumatic constitution (Drs. R. W. Forrest, James Finlayson, and Adam, *Glasgow Medical Journal*, 1879). It has also been attributed to injury over the renal region, and to the influence of malaria; and in connection with septic fevers, purpura, and scurvy.

(*b.*) *Morbid Anatomy.*—The urine contains albumen, usually to a large amount, but no blood-corpuscles can be detected. They are believed to become disintegrated and dissolved.

Symptoms.—It comes on in sudden and irregular paroxysms, occurring during the day, and lasting from three to twelve hours. Certain premonitory phenomena already mentioned usher in an attack, also chills, a sense of weight in the bowels, dull pain or tenderness over the region of the kidneys. The temperature may fall below the normal. The urine passed is of a dark or red colour, resembling porter or port wine; the intensity of coloration varying with the intensity of the cold which produced the attack. It is turbid, and throws down an abundant sediment, and is highly albuminous. It rapidly recovers (within a few hours—twelve to twenty-four) its normal condition. In the intervals it is quite normal. Spectroscopic examination shows that *hæmoglobin* is present; while Drs. Forrest and Finlayson's examinations indicate the presence of two separate ingredients, as determined by spectrum analysis.

Treatment.—Improvement has taken place under the use of the *saccharated carbonate of iron* and *quinine* in full doses.

SUPPRESSION OF URINE—*Syn.*, ISCHURIA RENALIS.

LATIN EQ., *Urina Suppressa*—Idem valet, *Ischuria Renalis;* FRENCH EQ., *Suppression d'Urine;* GERMAN EQ., *Harnvelhaltung;* ITALIAN EQ., *Soppressione dell' Orina*—Syn., *Iscuria Renale.*

Definition.—*A complete or partial suspension of the functions of the kidney, by which the quantity of urine is greatly in defect, or its secretion entirely suppressed.*

Pathology.—(*a.*) *Causation.*—This affection may be caused by disease of the kidney itself, or it may be secondary, and arise from disease in other parts of the body. Among the latter are injuries of the head or spine, an attack of *pneumonia*, when the patient may pass only a few ounces of urine in twenty-four hours, or of hysteria, when it is often suppressed for several days together, and also of inflammation or irritation of the bladder. It may depend on inflammation of the kidney, caused by some poison acting on that organ, as that of *small-pox, scarlet fever, typhus, cholera*, or by *cantharides* or *turpentine.* The presence of a calculus in the kidney or the ureter is also an occasional remote cause. All ages are liable to this affection. Children from teething often suffer a complete suppression, or only pass a few drops of fiery urine in the twenty-four hours, and that at several hours' interval. In the adult it is due to gravel or stone; and in the aged from disease of the brain, or cord, or Bright's disease. The urine passed at such times is extremely high-coloured, stains the linen, and is passed with great pain, the child crying bitterly, as it scalds the surface over which it flows. In hysteria the urine is often suppressed for three or four days. A case in which no urine was secreted for nine or ten days is given by Dr. Laing, and yet the patient did well.

As extreme cases, Dr. Parr, in his *Medical Dictionary*, mentions a patient who made no water for twenty-two weeks, while Dr. Richardson speaks of another who up to seventeen years of age had never passed a drop of water in his life. In this case the ureters must, as in birds, have terminated in the large intestine, and the urine have been passed with the fæces. In general, however, it may be laid down as a maxim that, when suppression of urine depends on any acute or severe disease, the patient seldom survives this symptom more than three or four days. It is often the final result of such disease as *cholera, typhus, scarlet fever,* and other blood diseases. It is certain also that some cases are feigned; some women, for instance, are said to have had a vicarious discharge of urine from the stomach; and Nysten gives the case of a girl who vomited urine, but it was at length ascertained she first swallowed it. Rayer gives a similar case of a woman at La Charitié, who had an abdominal tumor, which was supposed to be connected with the kidneys. Many persons saw her vomit urine, and Guibourt detected it chemically in the matters thrown up; but it was found that she had first drank it, though for what motive, except notoriety, nobody could discover (Dr. ROBERT WILLIAMS).

Symptoms.—Among the symptoms of suppression of urine is an urinous odour of the perspiration from the axilla and umbilicus. There may be some pain in the back, or some irritability of the bladder; the patient becomes anxious and restless, till at last the brain is oppressed, and he may die comatose. In other cases there is nausea, with hiccough, and the body exhales an urinous odour. When the suppression is less complete, and depends on an affection of the bladder, the local sufferings of the patient, the forcing of the bladder, the tenesmus, and the general irritation of the sufferer are severe, and distressing to witness.

Diagnosis.—Suppression is to be distinguished from mere retention of urine in the bladder, or from *ischuria vesicalis,* by there being no fulness in the vesical region, and by no urine flowing when the catheter is passed.

Prognosis.—Many cases recover from a suppression of urine of not more than twenty-four to forty-eight hours; but, except in hysteria, few survive if the disease continues a longer period.

Treatment.—When suppression does not depend on any morbid condition of the blood, and is primary, the patient should be placed in a warm bath, and be purged by substances that act on the kidney, as the neutral salts. Indeed, if the case be slight, purging by any cathartic is sufficient. If this method should not succeed, ♏x. to xxx. of the *tinct. cantharides* should be tried every four or six hours, according to the urgency of the case. Many physicians prefer a tonic treatment, as the *camphor mixture* and *ether*, or the *tinct. ferri muriatis*, ♏ xxv. to l. *Belladonna* is a useful remedy, and so also is *digitalis* applied as a fomentation of the fresh leaves over the abdomen; or an ounce of *the tincture* may be added to a warm linseed poultice, or the *dried leaves* may be made into a poultice, to which half an ounce of the *tincture* may be added. It is chiefly in cases in which the pulse is rapid that digitalis is suitable; and the urine will not begin to flow till the digitalis has reduced the action of the heart (J. D. BROWN, in *Medical Times*, January 25, 1868). In the suppression of urine attending cholera, Dr. E. Goodeve recommends the following :—

℞. *Tinct. Digitalis*, ♏ v. to ♏ x.; *Spr. Æther. nit.*, ♏ xxx.; *Liq. Ammon. Acet.*, ♏ lx.; *Aq.*, *f*℥i.; *misce.* Such a draught may be taken every three or four hours. (Reynold's *System of Medicine*, Vol. I., p. 183.)

The treatment of *symptomatic anuria* resolves itself entirely into that treatment which will remove the primary disease.

FLOATING KIDNEY; MOVABLE KIDNEY.

Definition.—*A floating kidney is a condition in which the kidney has a mesentery or fold of peritoneum attaching it very loosely to the spine, by which it can be moved about to the extent of the length of its mesentery. A movable kidney merely slips a little under the fingers.*

Pathology.—*Causation.*—It is much more common in women than in men, and more common on the right than on the left side; and at ages between 25 and 40—*i.e.*, the child-bearing period of women. It may be due to the concurrence of several causes—(*a.*) the practice of tight-lacing (Cruveilhier); (*b.*) child-bearing with tight-lacing (Roberts); (*c.*) congestion and tumefaction during the menstrual period (Becquet); (*d.*) rapid emaciation after corpulence; (*e.*) a blow or violent concussion of the trunk (Fleming, Roberts); (*f.*) by far the larger number of cases have been women who have borne children. With regard to the greater frequency of floating right kidney, it is more depressed during deep inspiration than the left, probably from its relation to the liver (Jenner).

Symptoms.—The patient being recumbent *palpation* discovers a swelling which is rounded, smooth, and of the size and shape of the kidney. It can be moved in various directions, the movement being peculiarly slippery in character. The borders can be easily defined, except the inner concave edge, which is obscure. It lies more or less diagonally below the costal border, midway between the umbilicus and the tip of the last rib. It is most movable in a direction upwards, backwards, and outwards; or downwards and towards the middle line. The swelling descends when the upright posture is assumed. In the recumbent posture a deep inspiration will sometimes bring the floating kidney forwards, so that it can be felt: and it may fall to the right or left according to the inclination of the body. When both kidneys freely float, they may be both brought forward together, and their convex margins maintained in contact in the middle line. They bear manipulation well. Firm pressure causes pain. A dragging uneasy sense of weight may be experienced in the abdomen; and the movements of the floating organ may be felt by the patient, who (if a female) may believe the movements to be those of a child (Dr. James Sawyer).

Diagnosis is of this importance, that it enables us to give comforting assurances to patients, removing anxious fears, and preventing the adoption of useless, perhaps injurious measures of treatment. If unaware of the probability of the existence of floating kidneys, the practitioner may suppose the condition to result from various other causes, and so submit the patient to an injurious course of treatment (Sir WILLIAM JENNER, *Brit. Med. Jour.*, Jan., 1869).

Prognosis.—The condition never shortens life.

Treatment.—Wearing a tolerably tight, elastic abdominal bandage may completely remove the abnormal condition, and will at least relieve the dragging and uneasy sensations. Constriction of the thorax at the lower part by stays or waistbands must be avoided. Constipation, and the consequent straining, as invariably aggravating the mischief, must be relieved. Treatment generally must be of a tonic nature (SAWYER, l. c.)

SECTION VI.—DISEASES OF THE BLADDER.

CYSTITIS—*Syn.*, CATARRH OF THE BLADDER; VESICAL CATARRH.

LATIN EQ., *Cystitis*—Idem valet, *Catarrhus Vesicæ;* FRENCH EQ., *Cystite*—Syn., *Catarrhe de la Vessie;* GERMAN EQ., *Blasenentzündung;* ITALIAN EQ., *Cistitide*—Syn., *Catarro della Vescica.*

Definition.—*Inflammation of the urinary bladder.*

Pathology.—(*a.*) *Causation.*—(1.) The direct irritation of calculi or of morbid growths, or of conditions of the urine arising from the abuse of alcoholic or terebinthine fluids—gin, beer, and spirits, also the use of cantharides; the irritation also of ammoniacal urine, as in cases of paralysis of the bladder, in consequence of spinal disease or injury. (2.) Extension of inflammation by continuity, as from gonorrhœa. (3.) Exposure to cold and wet.

(*b.*) *Morbid Anatomy.*—The mucous membrane of the bladder is liable to the *diffuse*, the *serous*, the *adhesive*, the *suppurative*, and the *ulcerative* forms of inflammation; and these may be either *acute* or *chronic.* The inflammation may extend over the whole cavity, or be limited to a portion. The part most frequently inflamed is that near and around the neck. The inner membrane of the bladder in the dead body hardly shows any vessels which are large enough to carry red blood in its natural state; but when *diffusely* inflamed, it is crowded with a prodigious number of extremely fine blood-vessels, and among them may be seen small spots of extravasated blood. This state has many degrees, and the colour is usually of a venous red, while, in addition to this, the coats of the bladder generally are thickened. It may terminate by resolution, or it may pass into *serous* inflammation, or *catarrh of the bladder.* The mucus then secreted is at first small in quantity and extremely fluid, but is deposited as the urine cools. At a further stage of the disease it becomes abundant and thickens, surpassing the urine in quantity, which now resembles thick gruel, and is often mixed with blood or gravel, or both. The internal surface of the bladder has been seen coated with lymph more than a line in thickness, and similar to the false membrane of croup (ANDRAL). Lymph thus effused may become organised; and in this manner calculi have become encysted, and so removed out of the reach of the sound. Inflammation of the mucous membrane of the bladder may terminate in suppuration, and pus to a considerable amount may then be passed. Occasionally, instead of suppuration taking place at its free surface, an abscess forms: in either case ulceration may take place, sometimes superficially, and sometimes so burrowing as to perforate the bladder, and form a communication between it and the neighbouring parts, as the cavity of the abdomen, the rectum, or the vagina. When the communication is formed with the general cavity of the abdomen, the urine escapes into it, and produces general peritoneal inflammation.

The bladder is also liable to chronic inflammation, sometimes retaining its normal colour, and at other times being grey, brown, or black. It may then acquire a double or even triple thickness (hypertrophy of the mucous coat). One of the most ordinary changes is hypertrophy of its muscular coat. In a natural state the muscular coat of the bladder, when moderately distended, consists of thin layers of fibres running in different directions, and probably less than the eighth of an inch in thickness. It may be found, however, in some cases half an inch thick, owing to its efforts to overcome some resistance, as an enlargement of the prostate, or the presence of a calculus or of a stricture in the urethra to the passage of the urine. These efforts of the bladder to evacuate its contents have led to the mucous membrane being protruded through the intermuscular spaces, forming a pouch or hernial sac, in which a small calculus has been imbedded; but this form of lesion is extremely rare.

The mucous membrane of the ureter may participate in the inflammation of the bladder, and is liable to the diffuse, to the adhesive, to the ulcerative, and to the suppurative inflammations, and these also may be acute or chronic. The ureter is occasionally found highly vascular, and of venous colour after the passage of a calculus. There are repeated instances of adhesive inflammation of this canal. Andral quotes a case in which all the internal surface of the ureter was covered with a layer of lymph similar to the membrane in croup; and in some rare instances the ureter has been found obliterated or transformed into a fibrous cord. There are other well-marked effects of adhesive inflammation of the ureter, as when the delicate coats of this canal are increased, as in cases of severe chronic disease, to four to six lines in thickness. Suppurative inflammation also sometimes takes place in the ureter, and without breach of surface.

The **Symptoms** of inflammation of the bladder are pain felt in the perinæum and above the pubes, accompanied with a fulness or swelling, frequent attempts to make water, which is evacuated in small quantities and with great pain; or there is a total retention of urine, with a strong desire to void it. The rectum is affected, from its connection with the bladder, with tenesmus, and there may be nausea or vomiting. In some cases these symptoms are accompanied with much constitutional irritation and by delirium. When pus is formed, it is mixed in the urine evacuated. The slighter form of the disease is characterised by milder symptoms, which consist principally of local pain and irritation, and by the urine being loaded with mucus, which sinks to the bottom of the vessel, mixed with a large quantity of sandy precipitates, either of the phosphates, of the urates, or of both. The symptoms vary only in degree. The symptoms of inflammation and of other diseases of the ureter are probably the same as those of the similar diseases of the bladder, except, perhaps, that the pain is more strictly lumbar; and when these canals are greatly enlarged, it is possible they may be felt through the walls of the abdomen.

Diagnosis.—When the kidneys and ureters are diseased, the bladder very constantly sympathises with those diseases; and the affections of the bladder being much more painful than those of the ureters and pelvis of the kidneys, the sympathetic affection of the bladder may be mistaken for the primary disease. Morgagni first pointed out this fact, and he gives a case in which, from these sympathetic pains, it was believed that the patient laboured under disease of the bladder; yet after death the bladder was found perfectly healthy, while the kidneys were extensively diseased and filled with large calculi. Lowdell and also Howship give similar instances of the kidneys being diseased, when the symptoms of the bladder were so prominent as to be mistaken for the primary disease.

Prognosis.—The result of the acute forms of inflammation of the bladder or ureter is generally favourable. The chronic forms of *cystitis* are more formidable, and often ultimately cause the death of the patient. As a rule *cystitis* is a disease of long duration.

Treatment.—The causes must, if possible, be removed. *Purging*, together with *opiates*, *diluents*, and the *warm bath*, are the best means of curing the acute affections. Chronic inflammation of the bladder, and especially catarrh, is very difficult of cure, and often our best directed efforts are unsuccessful. *Opium* is the remedy of the greatest value, especially aided by hot hip-baths, hot fomentations over the abdomen, and linseed-meal poultices, with or without mustard or turpentine, over the hypogastric region. *Suppositories of morphia* are of great use, containing half a grain to a grain of opium; but if the pain be very great, much larger doses may be given—as much as two to four grains of *opium*, with ten to fifteen grains of *extract of hyoscyamus*, in a suppository, at the hour of sleep. Alkaline and demulcent drinks should be at the same time given. The state of the urine is one of the surest guides in our attempts to cure the patient. If the urine be acid, the best medicines are the *neutral salts* or the *pure alkalies*, with *opiates;* while, if the urine be alkaline, or greatly loaded with mucus, the *mineral acids* are of the most service, combined with an opiate. Thus the *infusio rosæ c. acidi sulph. dilut.* ♏ij. to v., *c. magnesiæ sulphatis*, ʒj., *c. tinct opii*, ♏iij. to v., every sixth hour, is one of our best and most useful remedies. Tonics often succeed when other remedies fail, and of these *salicine* is one of the best. It may be given in doses of ten grains every six hours. It must be admitted that much difference of opinion prevails as to the best tonic remedy, some preferring *uva ursi*, others *pareira*, others the *turpentines*, as the *Canadian balsam*, and others again the *infusio diosmæ*. In chronic catarrh, when the discharge is copious, the decoction of *uva ursi*, in doses of not less than half a pint daily, is very soothing. It is of most advantage in cases of irritability rather than of inflammation, prescribed in large doses, from one to two drachms of the extract daily, or from eight to sixteen ounces of the infusion as a drink, made as follows:—

℞. *Fol. uvæ ursi*, ℥i.; *Aq. Fervid.*, *f* ℥xviii. Macerate for two hours, and boil down to 16 ounces, and strain (Sir Benjamin Brodie).

CHAPTER XXI.

DISEASES OF DUCTLESS GLANDS.

Section I.—Diseases of the Thyroid Gland.

GOITRE.

Latin Eq., *Bronchocele;* French Eq., *Goître;* German Eq., *Kropf*—Syn., *Struma;* Italian Eq., *Gozzo.*

Definition.—*An enlargement of the thyroid gland, by some regarded as specific, and induced by the persistent use of water which has percolated through magnesian limestone rocks, or strata containing the soluble salts of lime in solution; or it is regarded as a circulatory disease, the twofold blood and nerve supply of the thyroid gland being favourable to its enlargement under conditions of excessive and constant muscular exertion.*

Pathology.—(*a.*) *Causation.*—The prevalence of the disease has hitherto been believed to be limited to certain districts of peculiar geological formation. Wherever it prevails popular opinion has regarded the water used for drinking as being in some way connected with its cause. Alike in England and in Oude, where goitre prevails, the people are convinced that water in some way is its cause. *Snow-water* from the summits of lofty mountains was at one time believed to contain the poisonous material which established *goitre* among the inhabitants of the valleys. The people of almost all the valleys of Switzerland drink snow-water, or the water of the glaciers; but *goitre* abounds in some of the valleys only; and in Greenland and Lapland, where snow-water is commonly used, goitre is unknown. The disease also exists in countries in which snow is never seen, as in Sumatra; and in places where snow never lies sufficiently long to be used as a drink, as in Derbyshire. It prevails also in places where *pump-water*, rather than *surface-water*, is used. At Nottingham, where this disease prevails in England, the people refer it to the *hardness* of the water—*i.e.*, to its impregnation with calcareous salts, *sulphate* or *carbonate of lime;* and it has been affirmed that the presence of magnesian limestone always implies the endemic co-existence of the disease (Inglis). In Captain Franklin's expedition to the Polar Sea, *goitre* was found to be prevalent at Edmonton, where the soil is calcareous, and contains numerous fragments of magnesian *limestone.* Those only suffered who drank from the *water* of the river (the Saskatchawan). In its worst form it is confined to the half-bred women and children who make use of the river-water. The men, who are often from home journeying through the valleys, drink the melted snow, and are less affected; and, should incipient symptoms of the disease come on in winter (when they live at home and use the water of the river), the annual summer voyage to the sea-coast generally effects a cure. The natives who use *snow-water* only, and drink from the rivulets which flow through the plains in summer, are exempt from the disease; but the residence of a single year at Edmonton, if the river-water of the Saskatchawan is used, is sufficient to render a whole family the subjects of goitre (RICHARDSON). The disease has been known to occur and to affect a family in a very short time, who, being free from the disease while using the *surface-water*, had a well dug, and obtained their water by tapping a limestone rock. After drinking from this well, the disease appeared among them. There are some waters in a goitrous district in Switzerland, issuing from the hollows of certain rocks, and trickling along crevices in the mountains, the drinking of which will produce *goitre*, or augment goitrous swellings, in eight or ten days, while the inhabitants who avoid these waters are free from the disease (BALLY, WATSON). The use of the hard pump-water in the lower streets of Geneva brings on goitre very speedily (Dr. COINDET). At Cluses, on the Arne, numerous cretins and goitrous persons are seen in the streets; while lofty cliffs of mountain limestone tower over the town, and through the crevices of these cliffs copious streams of water flow. In Yorkshire, Derbyshire, Nottingham, Hants, and Sussex, where the disease prevails in England, there is a ridge of magnesian limestone running from north to south through the centre of the district. All along that line goitre prevails to its greatest extent; and, diverging to either side, the disease is found to diminish (INGLIS, *Treatise on English Bronchocele*). Goitre has been found to prevail in one great section of the province of Kemaon, in India, south of the Himalayan Mountains, and to be almost entirely absent from another section of that district. Both of them agree in their external aspect, altitude, and climatology, but differ so remarkably in their geological formation that an examination of the rocks of the district into the villages where the disease abounded, or did not abound, enabled one to predict whether the inhabitants were affected with goitre or not. No instance occurred, in a district extending over 1,000 square miles, in which goitre prevailed to any extent where the villages were not situated on or close to limestone rocks (M'CLELLAND). Dr. M'Clelland visited 126 villages scattered promiscuously over an area of upwards of 1,000 miles, with the following results:—(1.) Five of these villages were built upon hornblende and mica slate, or on siliceous sandstone, or on green sandstone. They contained 290 inhabitants, not one of whom was a *cretin* or was affected with *goitre*. (2.) Seventy-one of the villages in the same district were built upon clay-slate. These contained 3,957 inhabitants; and among them there were twenty-two persons with goitre, or *one* in two hundred of the population. There was not a single cretin. (3.) Thirty-five of the villages, having a population of 1,160, were built upon Alpine limestone; and in them 390 persons, or more than *one-third* of the inhabitants, had goitre, while thirty-four of them were cretins, or about one person in every thirty-five.

Lastly, goitre is extremely frequent at Secrora, near Lucknow, and in all that district of Oude which stretches towards Nepaul and the Goruckpore district beyond the Gogra. In the jungles of the Teraie, at the base of the Nepaul Hills, the disease is very frequent; and in Nepaul itself, among the inhabitants of the *Cis* and *Trans*-Himalayan regions, it is constantly met with (BRAMLEY, GREENHOW; *Indian Annals* for 1857 and

1859). All along the line of the Teraie, on to Goruckpore, goitre is so prevalent that one in ten persons is afflicted with the disfigurement. The kingdom of Oude is geologically made up of the diluvial detritus of the Himalayan chain, which abounds in limestone; and the soil of the district contains abundance of lime, which is taken up by the waters that percolate through it from the rivers and from the rains and floods. The lime thus taken up and held in solution with carbonic acid gas, is frequently found deposited round nuclear fragments of flints or stones, and is then known by the name of "kunker;" so that wherever "kunker" abounds, there soluble salts of lime, silica, alumina, and sometimes magnesia and protoxide of iron, will be found (SLEEMAN, O'SHAUGHNESSY, GREENHOW). Goitre in Oude affects animals as well as man. At Hissawpore (a village about twelve miles distant from Secrora, on the Surjoo River) dogs, cats, and other animals are affected with it (GREENHOW). Mr. Bramley noticed the same fact in Nepaul.

An outbreak of *acute goitre* occurred, in 1860, in the garrison of Briançon (Hautes Alpes). The mean strength of the garrison during the year was forty-eight officers and 954 men; and from this force fifty-three cases of acute goitre (fifty-one soldiers and two *enfants de troupe*) were admitted in the year, and in the first three months of 1861. One case occurred in as short a time as *eight* days after arrival at Briançon, and one after *sixteen* months' stay; but the majority (thirty-nine) occurred after from *eight* to *eleven* months' sojourn. The rapidity of growth of the gland was remarkable. In some very predisposed subjects eight days sufficed to show a large increase; and the form of the tumor was most frequently bilateral. The sanguino-lymphatic temperaments and robust constitutions were those most attacked. There was no obvious hereditary tendency; but several of the men came from departments where goitre is more or less prevalent. The men who came from maritime places, and who were placed at Briançon under quite unusual conditions (4,285 *feet above the sea-level*), suffered most. M. Colin, who records the outbreak, does not say a word about the composition of the water; but the position of Briançon renders it highly probable that the water of the place is charged with lime and magnesian salts (PARKES, in *Army Med. Department Sanitary Report* for 1860, p. 385).

The disease is known to prevail at the base of lofty mountains in many parts of the globe. It is endemic at the foot of the Alps, where it is frequently associated with *cretinism*—a sort of idiocy associated with atrophy of the brain and deformity of the body. It is also endemic at the foot of the Apennines. It is common in Derbyshire, where it is called "Derbyshire Neck." It is met with in some flat situations in Norfolk, and in one village about five miles from Cambridge it is extremely common (WATSON). In South America goitre is met with both in the upper and lower course of the Magdalen River, and in the flat high country of Bogota, 6,000 feet above the level of the stream (HUMBOLDT). It is also common at the base of the South American Andes. In North America many cases occur in the vicinity of the Blue Ridge, in Virginia. It is prevalent in the mountainous regions of Pennsylvania, New York, New Hampshire, and Vermont (DUNGLISON). In India it prevails in Oude, and along the line of the Himalayan range. It seems to be more common in females than in males, and is rarely seen before the age of puberty; but in districts where the disease abounds, it is on record that children are sometimes born goitrous of goitrous parents (GODELLE, WATSON). The evidence of hereditary transmission, in the strict sense of the term, appears to be doubtful; but predisposition may exist in some rather than in others, to the development of the disease.

From such evidence it has been concluded, that in association with lime and magnesia in geological formation, premature and abnormal ossification and thickening of the base of the cranium takes place from the unusual quantities of lime taken into the system by the water, tending to diminish the size of the foramina for blood-vessels (KÖLLIKER, VIRCHOW). Almost all writers who have written on the subject agree that, in some way or other, the condition of the water has to do with the production of goitre. Remarkable instances are known wherein the exchange of well for rain-water for drinking purposes, has been followed by the best effects, and even by the disappearance of goitrous tumors. In Oude, where the water of wells believed to be injurious, in consequence of their excessive impregnation with lime, has been given up, and other water used instead for drinking, great benefit has been felt, and goitres have decreased in size, even though the subjects of them have continued living in the same village as before (GREENHOW). Certain wells were known to be deleterious, and the natives of the villages avoided them accordingly, having learned to do so from experience. The water of the wells most shunned by the natives were tested, and found to contain a great excess of lime; and Dr. Greenhow concludes, from his own investigations in connection with others, that the use of drinking-water containing lime is the main cause of goitre. How it acts on the system is as yet unknown.

On the other hand, there is a want of chemical analysis in support of the hypothesis that there is no relation between the hardness of water and goitre. Prompted by this remark of Parkes in his *Manual of Hygiene* (which has so greatly stimulated original research), Dr. J. B. Wilson, of the Army Medical Department, has carried out some independent inquiries into the causes of goitre at Bhagsoo, Dhurmsala, a hill station of the Punjaub for British troops (*Indian Annals of Med. Science,* No. XXXII.) The whole district is hilly, and is about 6,100 feet above the level of the sea. Through the sandstone hills, chiefly formed of rich red clay overlaid with vegetable mould, numerous extensive veins of limestone run and seams of fine slate. Amongst the native inhabitants of this district goitre prevails. Dr. Wilson analysed specimens of the drinking water obtained from this district within a radius of ten miles. It was more than usually pure, and did not contain either lime, magnesia, nor iron. Three specimens only indicated a small amount of lime. Hence it is not absolutely necessary for people to drink hard water before they can become affected with ordinary goitre. The disease is not connected with the composition of the water. An excess of the disease occurs amongst those whose daily duties are most laborious. Of the subjects of goitre, 52 per cent. were labourers whose duties were to carry heavy loads strung upon the shoulders and back, or to carry for long distances in a *dooly* European passengers; or to be employed in carrying and cutting stones, building walls, and cultivating land. The attidude which the labourer assumes is peculiar. He is rarely seen in the erect posture, unless conveying loads. In 42 per cent. of the cases it appears between the ages of ten and twenty, the time of life when labour is most actively pursued, and when the constitution receives the new impulse and modifications of puberty. Next in order of susceptibility to the disease is the period of infancy and childhood. In a registration of the cases according to age, Dr. Wilson found invariably a marked excess in the frequency of arterial pulsation; and that the subjects of goitre were liable to cardiac palpitation. In all acounts of this disease its frequency seems to be universal in hilly and mountainous districts. The complex trophic functions of the thyroid gland, as shown by its twofold blood and nerve supply, must be regarded as an agent which regulates intracranial circulation (see page 50, *ante*)—an agency which becomes apparent when under excessive muscular exertion the veins of the neck are compressed, and the thyroid gland becomes swollen by the strong influx of arterial blood (GUYON). The late Professor Laycock was the first to call attention to the clinical relation of these facts to goitre and cretinism (*Mind and Brain*, 2nd Ed., p. 476); and now Dr. Wilson's observations strongly confirm the theory that ordinary goitre (like the exophthalmic variety) is entirely a circulatory disease; that its tendency to occur is encouraged and may be induced by conditions of the constitution in a reduced state favourable to the disease—namely, (1.) Active and laborious occupation in constrained positions. (2.) Violent exercise in its effects upon the heart and larger blood-vessels in their relation to the thyroid gland. (3.) Its remarkably large supply of blood, its dense capillary structure, and consequent ready liabilities to enlargement from dilatation of its vessels under violent and prolonged exercise. (4.) The effects of elevation above the sea level upon the circulatory functions seem favourable to the production of goitre; and the frequent accession of palpitation, giddiness, frequent bleedings at the nose, increase of frequency of the pulse, are all symptoms which point to disturbance of the circulatory system favourable to the development of goitre. (5.) The prevalence of this disease recorded to have taken place in prison, may have arisen not from the water, but from excessive exercise at the tread-mill, or some other violent labour, whilst the inmates were on prison rations (Dr. J. B. WILSON).

(*b.*) *Morbid Anatomy.*—The characters of the swelling of the thyroid gland, associated with goitre, appear to be different at different stages. At first the tumor is soft, but it gradually acquires a firm and even a cartilaginous consistence. In the soft condition the cell-elements of the gland secrete a thick, ropy, viscid, gelatinous fluid; but when the consistence increases, the hypertrophy of the cell-elements is generally more obvious than the fluid secretion. Its blood-vessels are increased in size and number, and ultimately cysts become developed, in which the glairy fluid abounds. In more advanced cases, osseous, or at least calcareous, deposits occur, and occasionally the whole gland is transformed into a osseous-like capsule, filled with matter of various kinds, which has been likened to jelly, suet, and honey. Sometimes the gland preserves its original lobulated form, its relative proportions being maintained; at other times there is no distinction of parts or lobules. The right lobe is more often enlarged than the left (Alibert, Rickwood, Greenhow). Solid aggregations of calcareous particles have been found in the goitrous gland (Ceely). The tumor, originally of a simple nature, is liable to enlargement of its arteries, to expansion of its cells, or the formation of cysts on its surface and in its interior, or to inflammation and ulceration of its component tissues. Such ulceration is apt to assume a malignant scirrhus-like appearance.

Treatment.—The indications are—(1.) To remove from the district where the disease prevails; (2.) improvement of the water used as drink; (3.) elimination of the goitrous poison, or change of constitution. As with the poison of *mercury* and of *lead*, so with goitre, *iodide of potassium* has a wonderful effect in subduing the swelling, and probably in eliminating the poison which produces goitre, whatever that may be. *Iodine* has thus acquired the reputation of being almost a specific against goitre (Coindet, Straub, Gairdner, Manson, Lugol). In robust subjects it has been advised that the administration of *iodine* should be preceded by general blood-letting, on the principle that the abstraction of blood favours absorption; but this should not be done unless the *iodine* does not seem to be producing any effect; and whatever may be the preparation of *iodine* employed, it must be persevered in for a length of time, and the dose gradually increased (Dunglison). The best form of administration is that in which the *iodine* is mixed with and dissolved by *iodide of potassium*. It is then held in solution, and is sometimes known as *Lugol's solution*. It is made by dissolving *one part of iodine* (five grains) with *two parts of iodide of potassium* (ten grains), and adding water (to the extent of *one pint*). It contains *one grain* of iodine in four ounces of the solution. The treatment ought not to commence with a larger dose than *one drachm* three times a day. Small unirritating or alterative doses are the most efficient, and they may be gradually increased when necessary. The remedy may also be employed externally, in the form of a liniment or ointment—the *unguentum iodum compositum*, of which a small portion may be rubbed upon the swelling night and morning. In some parts of India the application of an ointment of the *biniodide of mercury* has been found efficacious. The ointment is prepared by adding finely-powdered *biniodide of mercury* to melted lard or mutton suet or *vasaline* (in the proportion of 15 grs. of *biniodide of mercury* to 1 ounce of lard. See p. 819, *ante*). This ointment is then applied to the goitre about an hour after sunrise, and is rubbed in, by means of an ivory spatula, for about ten minutes—*the patient sitting with his goitre held well up to the rays of the sun as long as he can bear the exposure*. The ointment may produce a blistering effect, although no vesicles appear on the skin; and in the course of the day it should be gently rubbed in again, and the patient sent home, with orders not to touch it with his hands, but to allow the ointment to be gradually absorbed. A second application is sometimes necessary in very bad cases. In 1855 no less than 500 or 600 persons were sometimes treated in a single day; and it is estimated that altogether about 60,000 patients have been so treated; so that the cases n the district are now far less numerous than formerly, and the disorder s thus being extinguished (Mouat, in *Indian Annals* for April, 1857). When medical treatment fails, surgeons have attempted to give relief by ne of three operations; but so long as the disease does not interfere with ny of the important functions of the body, nor produce serious discomfort, surgical interference is not warrantable. These operations are,—(1.) The introduction of setons through the tissue of the diseased gland—an operation which has been successful. A thin double wire is to be passed through the gland, and left there for a week (QUADRI, TANNER, JAMES). (2.) Tying the thyroid arteries which supply the goitre with blood, and so starving the tumor, has been attended with varied success (COATES, BRODIE, EARLE, WICKHAM). These means having failed, (3.) the gland has been extirpated—an operation which few surgeons would now think of undertaking.

Regarding these surgical interferences for the radical cure of goitre, Sir Thomas Watson observes, that "there is not one of which the *average* results have been sufficiently prosperous to warrant its repetition, except in cases where life is put in jeopardy, or made miserable by the swelling; and where other methods, and particularly the treatment by *iodine*, have been tried and have failed" (*Principles and Practice of Physic*, Vol. I., p. 795). He makes, however, an exception in favour of puncturing any cell or cyst containing fluid, which sometimes makes up a considerable portion of the tumor. Such cysts may be punctured without much risk, and with great relief to the patient.

EXOPHTHALMIC BRONCHOCELE.

LATIN EQ., *Bronchocele Exophthalmica;* FRENCH EQ., *Goitre Exophthalmique;* GERMAN EQ., *Klotzaugenkropf*—Syn., *Basdow'sche Krankheit;* ITALIAN EQ., *Broncocele Esoftalmica.*

Definition.—"*Enlargement, with vascular turgescence of the thyroid gland, accompanied by protrusion of the eyeballs, anæmia, and palpitation.*"

Pathology.—(*a.*) *Causation*—This form of *goitre* is totally different from the endemic form, and has received the name of *exophthalmic goitre*, the *goitre* of *anæmia* or *spanæmia*. It is rare in children; more common in females than in males; and co-exists with wasting discharges, or supervenes upon them; such as leucorrhœa and menorrhagia in females, and hemorrhoids in males. It is sometimes associated with heart disease. Palsy of the vaso-motor nerves connected with the carotids, thyroid gland, and heart, is the explanation given of this disease, which is far more common among women than among men. The thyroid body seems to act as a diverticulum.

(*b.*) *Morbid Anatomy.*—Simple hyperæmia prevails in the gland, which subsequently becomes hypertrophied and very large, partly from dilatation of its vessels, and partly from infiltration of the tissues with serum, or from simple hyperplasia. Sometimes cysts form with serous or colloid contents.

The exophthalmic state is similarly induced; continued distension of the intraorbital vessels, with growth of fat, presses the eyeballs forwards; and the exophthalmic state may be reduced by pressure applied to the carotids. In fatal cases the eyes are observed to recede within the orbit after death (BEGBIE). Vision is rarely impaired. Dilatation of the cavities of the heart is the lesion which is induced in that organ, and dilatation o the veins is the last expression of the disease (FLETCHER, in *Brit. Med. Journ.*, 23rd May, 1863).

Symptoms.—The normal nutrition of the nerve-centres is obviously impaired. Sleep is disturbed and unrefreshing. Digestion is impaired. Pallor and anæmia ensue, with excessively frequent, long-continued palpitation of the heart and carotid arteries. A systolic *bruit* may be heard over the region of the heart, and sometimes also over the carotids; and during any excitement, a rushing or throbbing feeling is experienced in the head. The pulse is frequent (120 to 140), and excessive vascular excitement of the arteries of the upper extremities and carotids, especially with enlargement of the neck and prominence of eyeballs, are the earliest signs of this disease. A whistling noise is heard over the region of the thyroid, and a peculiar sensation may be felt by the hand laid over the gland. The general symptoms are very irregular; and the disease generally drags on for months or years.

Prognosis.—The result may be unfavourable, by reason of dilatation of the heart, with diminished functional power. The patient becomes cyanotic and dropsical, with dyspnœa. Death takes place more rarely by cerebral symptoms. As a rule, however, the disease ends in recovery.

Treatment.—The use of iron and tonics generally are indicated; and considering what has been already said under diseases of the heart, the use of *digitalis* may be indicated in connection with the cardiac symptoms. The indications for treatment are, to allay the irritability of the stomach by the use of ice; to give bland, unstimulating, nutritious food in small quantities and at short intervals; to produce sound and refreshing sleep by morphia, or any such stimulating soporific; to administer digitalis; to steady the weak heart and control its excitement. Iron may improve the state of the blood; but the *hygienic* conditions in which the patient lives are mainly to be rectified (FLETCHER, l. c.). Dr. J. Warburton Begbie recommended the use of *belladonna* in combination with iron (*Dublin Quarterly Journal of Medical Science,* Nov., 1865, by Dr. W. MOORE).

SECTION II.—DISEASES OF THE SPLEEN.*

The following diseases of the spleen are named by the College of Physicians:—*Splenitis; abscess* (*including pyœmic inflammation and abscess*); *congestion*, synonymous *with ague cake; fibrinous deposits; hypertrophy* (*one form of which is associated with leucocythœmia*); *lardaceous disease of the spleen*, synonymous with *amyloid disease and waxy spleen; cancerous, colloid, and tubercular diseases; and parasitic affections.* Under the head of hypertrophy a peculiar enlargement of the spleen and lymphatic glands, originally described by Dr. Hodgkin, ought to find a place. It has been described under the name of "Hodgkin's disease;" and although it has not been recognised by the College of Physicians in their nomenclature, it is an affection which presents very striking peculiarities, and therefore is deserving of a distinct appellation and description. Of these in their order,

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* As the spleen is a ductless gland, its diseases are treated of in this chapter rather than among diseases of the digestive system, as in the classification of the College of Physicians. The origin of the spleen is from a special aggregation of cells belonging to the middle germinal layer of the embryo, and quite unconnected with the digestive organs. These cells are subsequently differentiated into the several structures of which the spleen is composed.

SPLENITIS.

LATIN EQ., *Splenitis;* FRENCH EQ., *Splénite;* GERMAN EQ., *Milzentzündung;* ITALIAN EQ., *Splenitide.*

Definition.—*A primary acute inflammation of the substance of the spleen.*

Pathology.—(*a.*) *Causation*—Primary inflammation is a disease which is extremely rare in this country. It is usually limited to the fenny districts, as Cambridgeshire, Essex, or other paludal places. It is common in the East Indies, especially in the low marshy districts of Bengal, and occurs in the paludal districts of other parts of the world. Now and then it is said to originate from a blow or other accidental violence, injuries which are much more apt to cause rupture than inflammation. Hæmorrhagic infarctions, occurring during the course of contagious fevers and general contagious diseases, blood-poisoning, diseases of the heart with fibrine on the valves and embolism, are the most frequent causes of consecutive inflammation and suppuration of the spleen (NIEMEYER). *Secondary splenitis* is generally the result of pyæmia or blood-poisoning, as in typhus ending in abscess (pyæmic), the pyæmic blocks or infarcts resembling the fibrinous infarcts in shape, only more irregular, because the process extends beyond the limits of the area of the terminal twigs of the blood-vessels; and they rapidly proceed to suppuration with the constant inflammation of the capsule of the spleen over the block of fibrine, probably due to the septic properties of the infarcts. In such cases there seems to be spontaneous coagulation of the blood—which itself is in a morbid state—as in the contagious fevers, especially typhus, without any evidence of embolism.

(*b.*) *Morbid Anatomy.*—The few cases of disease of the spleen occurring in this country may account for its pathology having been little studied. In diffuse inflammation we find it enlarged, of a deep venous colour, and its tissue so softened as to be readily broken down, or even reduced to little more than the consistency of coagulated blood. Hæmorrhagic infarctions are roundish or wedge-shaped, well-defined collections of fibrine, varying in size from that of a pea to a hen's egg. The base of the wedge is towards the periphery, and the apex is directed inwards towards the *hilus* of the spleen. A border of acute inflammatory reaction is often well marked round their margins. These fibrinous infarcts correspond to the areas which mark the terminal divisions of the branches of the splenic arteries, where they break up into the bushes of small twigs (*pencilli*), like the hairs of a paint-brush. At first they are brown-dark, or brownish-red, and quite hard. The lesion is sometimes seen in children under ten years of age. Diffuse inflammation may terminate by resolution, or pus may form; and in this case one or more abscesses, often containing several ounces of pus, have been formed. The abscesses sometimes make their way to the surface; and also to open into the left thoracic cavity, the stomach, the transverse colon, and the cavity of the peritoneum, when circumscribed peritonitis forms a sac for the pus. The process may also terminate by the accumulation caseating or becoming a mass of fibro-cellular material, which, gradually shrinking up, leaves a cicatrix-like contraction on the surface, in which calcification may occur. The spleen has also been found in a state of gangrene (BAILLIE).

The Symptoms and Physical Signs of splenitis are mainly due to the presence and changes induced by such infarcts. The hyperæmia and inflammation cause the gland to swell. In cardiac disease, with embolia from valve vegetations, the infarcts are often numerous, and the swelling therefore is proportionately considerable, with tumefaction, some pain in the side, and probably accompanied with dropsy. But such splenitis may go on to suppuration without marked local symptoms. The enlargement of the spleen, or "splenic tumor," can generally be made out by palpation and percussion. Its form is that of the spleen exaggerated. The lobulation or notching of its swollen margin can sometimes be felt through the parietes of the abdomen, if the patient has not too much subcutaneous fat. The enlarged gland, coming from beneath the margin of the ribs on the left side, can sometimes thus be traced extended low down, even as far as the pelvic region, and well over on the right side of the linea alba, and backwards towards the spine. Its lower margin may be felt rounded. Weight and uneasiness (rather than soreness) are present; and the tumor is movable in all directions by manipulation and change of posture when adhesions are not present. Hæmorrhage from the stomach and bowels is not unfrequent towards the fatal end of such cases, often so profuse as rapidly to hasten dissolution. The countenance is sallow; emaciation progresses, and the bowels are irritable, accompanied by œdema of the limbs or ascites.

The **Diagnosis** of enlarged spleen resulting from splenitis arising from one or other of the causes referred to, requires to be made out as between lardaceous disease, malignant swellings, or other tumors, or a swollen condition of the spleen, especially near the cardiac end of the stomach or tail of the pancreas, as in Hodgkin's disease, enlarged left lobe of lung, or omental suprarenal or kidney tumors.

Treatment.—Bleeding in *splenitis* will not effect a cure, while mercury is most pernicious. A *spleen mixture*, not dissimilar to that recommended by Celsus, is most in vogue in India. The best, according to Mr. Twining, consists of the following:—

R. *Pulv. Jalap.; Pulv. Rhei; Pulv. Calumba; Pulv. Zingib; Potass. Supertart.*, ā ā ʒj.; *Ferri Sulphat.*, ℈ss.; *Tinct. Sennæ*, ʒss.; *Aquæ Menthæ Pip.*, ℥ixss. Of this mixture an ounce or an ounce and a half is to be taken twice a day, or such quantity as may produce three or four stools in the twenty-four hours.

The *iodide of potassium* and the *bromide of potash* have been more recently recommended. The dose of the bromide of potash is gr. v. to x. *ter die*, combined with *camphor mixture*. The marked influence of the *biniodide of mercury*, in the form of an ointment rubbed into the skin over the surface of a *goitre*, in reducing its enlargement, has been already noticed (p. 1011, *ante*). It has been similarly used in cases of enlarged spleen; and the invalid soldiers who suffer from enlarged spleens invariably apply for some to take away with them when they are discharged from Netley Hospital.

HYPERTROPHY AND CONGESTION OF THE SPLEEN.

LATIN EQ., *Hypertrophia ;* FRENCH EQ., *Hypertrophie ;* GERMAN EQ., *Hypertrophie ;* ITALIAN EQ., *Ipertrofia.*

The spleen is enlarged under a great variety of circumstances, especially as a result of malaria or of *leucocythæmia*, or as a result of Hodgkin's disease; but true hypertrophy in its simple form is a rare occurrence, in which nothing abnormal is to be seen in the spleen or in the blood. Enlargement with hyperæmia occurs as a result or concomitant of pyrexia in many fevers, notably in enteric fever and malarious fevers, erysipelas, puerperal fever, pyæmia, and acute tuberculosis. The capsules of spleens so enlarged appear very tense, so that the gland feels plump and elastic; but on section its substance is generally soft, pulpy, or almost liquid, very full of blood, and of a dark red colour. Sometimes, however, it is so fine that a more or less smooth or coarsely granular surface is seen on section, with an abundant new formation of small lymph cells and nuclei, many of them contained in large mother cells (compound splenic corpuscles). These are to be seen especially in the spleen pulp and splenic vein. This condition constitutes the nearest approach to true hypertrophy, in which, with an increase in the quantity of diffuse granular matter, the enlargement is less due to hyperæmia simply, than to mere structural increase of normal constituents. Such simple hypertrophy sometimes results from long-continued obstructive disease of the heart, or *mechanical* congestion, following any obstruction to the portal circulation.

The largest spleen I have weighed was 10 lbs. 15 ounces in a boy soldier of eighteen years of age. It measured 14 inches long, $7\frac{6}{10}$ inches in breadth, and $4\frac{9}{10}$ inches in thickness. Its increase was mainly due to connective-tissue growth. The heaviest spleens are those affected with *lardaceous disease* and *leucocythæmia.* The large tumefied and simply congested spleens sometimes subside very rapidly. Abercrombie mentions one that subsided in a week after the ague on which it depended had been arrested. The hypertrophied spleen is generally more or less indurated. The spleen is occasionally atrophied, so that little more than a rudimentary spleen remains. The lowest weight I have had occasion to record is an ounce and three quarters, and of firm texture. The patient died of dysentery at the age of twenty-four, and the smallest spleens are those of patients dying of dysentery. It may be indurated; or greatly softened. Hydatids have been found in the spleen. In a few instances small portions of the spleen, about the size of a nut, are found indurated and nearly white. These appearances generally arise from embolism.

The following morbid condition is regarded by the College of Physicians as furnishing one form of splenic hypertrophy, namely:—(*a.*) Leucocythæmia; but, as will be seen, the disease here described as *leucocythæmia* has a much more extended pathology.

LEUCOCYTHÆMIA.

LATIN EQ., *Leucocythæmia;* FRENCH EQ., *Leucocythémie;* GERMAN EQ., *Leukæmie*—Syn., *Leucocythæmie;* ITALIAN EQ., *Leucocitemia.*

Definition.—*A disease* sui generis, *in which the number of white corpuscles in the blood is greatly increased, with a simultaneous diminution of the red. This state is brought about by chronic exhausting diseases, exposure to cold and wet, or serious acute affections—such as typhus fever, pneumonia, puerperal fever, affections of the lymphatic glands or of the spleen, and is attended sometimes by cough or diarrhœa, epistaxis, hæmorrhagic effusions, furunculus or pustulous eruptions.*

Pathology.—(*a.*) *History.*—In the sixty-fourth volume of the *Edinburgh Medical and Surgical Journal* a case of disease of the spleen is described by Dr. Craigie, "in which death took place in consequence of purulent matter in the blood." The case occurred in 1841, and proved fatal on the 1st of April of that year. The late Dr. John Reid, "on examining the blood of the veins of the abdomen and sinuses of the brain by the microscope," found "that it contained globules of purulent matter and lymph." Dr. Craigie inferred "that, by some means or other, purulent matter and lymph had been mixed with the blood, and, circulating with it, had given rise to the peculiar febrile and inflammatory symptoms which occurred during life, and to death in the manner in which it had taken place." He inferred that the spleen was the only organ from which the purulent matter and lymph could have proceeded, it having been for several weeks in a state of chronic inflammation; that in this form of disease of the spleen the pus-cells are secreted, and, being mixed with the blood, they cause much disorder in the circulatory system, and finally destroy the patient. Dr. Craigie thus recognised a connection between the diseased state of the spleen and the changed condition of the blood; and that "it was in some respects new." On this account he made a correct description of it, expecting at some future period that the chief facts might be confirmed. He kept the case unpublished till 1845, and it was only published then in consequence of the occurrence, to another physician in the same hospital, of a case "in many, if not in all, respects similar," which led Dr. Craigie to anticipate similar results, and which went far, as he thought, to confirm his conclusions deduced from the first case (*Edin. Med. and Surg. Journal* for 1845, p. 400, *et seq.*) The details of the second case referred to by Dr. Craigie were published by Dr. John Hughes Bennett in the same volume of the *Edinburgh Medical and Surgical Journal* immediately after the account of Dr. Craigie's case. Dr. Bennett describes his case as one of "*Hypertrophy of the Spleen and Liver, in which death took place from suppuration of the Blood;*" and although the most evident lesion during life was enlargement of the spleen, Dr. Bennett agrees with Dr. Craigie "in thinking that the immediate cause of death was owing to the presence of purulent matter in the blood, notwithstanding the absence of any recent inflammation or collection of pus in the tissues," and that it produced the febrile symptoms. In these valuable papers Drs. Bennett and Craigie are at issue, however, about the source of the pus in the blood. Dr. Bennett considered his case particularly valuable, because he believed it demonstrated "the existence of *true pus*, formed universally within the vascular system, independent of any local purulent collection from which it could be derived" (l. c., p. 414). He believed the white corpuscles he saw in the blood "were true pus-globules," and he then was of opinion that they were formed in the *liquor sanguinis* within the vessels, independent of inflammation or of phlebitis, or what was then understood by *pyæmia;* that the transformation had taken place throughout the system, and that the whole mass of blood was affected. The case appeared to him capable, in this respect, of furnishing an important fact which may throw light on the doctrine of *Zymosis*, as applied in Pathology (l. c., p. 423). In the same year (1845), about a month after the publication of Dr. Bennett's case, Professor Virchow, of Berlin, described and explained, in *Froriep's Journal*, of November, 1845, a disease of the blood as due to an increased development of white blood-cells. No inflammation of veins was observed; and the hypothesis of any spontaneous formation of pus in the blood (pyæmia) was contrary to the pathological doctrines of Virchow. To this form of disease he gave the name of "white" or "colourless blood." As to its cause, he points out, *as primary*, the condition of the spleen, entertaining the idea of an increased formation of colourless blood-cells through the functional relations of that organ. In 1845-46 several cases of leucocythæmia during life were recognised in this country. Dr. Fuller, of St. George's Hospital, in December, 1845, was the first to determine this condition during life; and it was also diagnosed by Drs. T. K. Chambers and Walshe, in London, and by Dr. Douglas, in Edinburgh, in 1846. Virchow continued to oppose the view of Dr. Bennett as to this affection having its origin in the formation of pus in the blood, or that its formation is in any way of the nature of a *Zymosis;* maintaining, on the contrary, the correctness of his first opinion, that the essence of the disease consisted in an increase of the colourless blood-cells (*Medicinische Zeitung*, August and September, Berlin, 1846). In 1847 Virchow collected and described cases from the older authors, and compared them with those observed in England and with those seen by himself; and finding also a further basis in favour of his views in the theory of Hewson and Donné regarding the function of the spleen in the propagation of the blood, he naturally became more confirmed in his opinion, that from some altered relation of the spleen to the blood-cells might be established those conditions under which "white blood" would be produced. In the *Archives of Pathological Anatomy* of the same year a case of great value is described by him as a new form of this "white-blood" disease. In this form the spleen presented no change whatever, whilst the lymphatic glands were enormously enlarged. He was now able to declare with more confidence that the "white-blood" disease proceeded from a primary affection of the spleen and lymphatic glands, as a direct consequence of which an increased development of colourless blood-cells takes place; and thus he considers that the disease, in its turn, throws light on the physiological functions of these glands as eliminating organs of the blood. In 1851-52 Dr. Bennett gave a most interesting and systematic view of the whole subject, first in the *Monthly Journal* of 1851, and afterwards in a separate work, entitled *Leucocythæmia, or White-cell Blood*, in which the explanation of the pathology of this disease is no longer referred to the spontaneous development of pus in the blood. Although Dr. Bennett originally set out upon what has since been believed to be an error in the interpretation of the phenomena of leucocythæmia, yet he was the first to show that these phenomena were new to science; while the more correct generalisation and elaboration of the subject seem, from the chronological history of the disease, to be mainly due to Virchow. He was the first to recognise the colourless cells in leuchæmia or leucocythæmia as identical with the colourless globules of the blood—the view at present generally entertained. More recent observation, pointing to the formation of pus from the white corpuscles of the blood, may (if found correct) again change the views now generally entertained. It is greatly to be regretted, however, that so much personality and ill-feeling had been imported into the historical records of this interesting discovery; and if the reader desires to read the several sides of the lengthened controversy, let him consult—(1.) The original cases of Craigie and Bennett in the sixty-fourth volume of the *Edin. Med. and Surg. Journal;* (2.) A review of Virchow's *Handbook of Pathology*, in the June number of the *Edin. Monthly Med. Journal* for 1854, p. 546; (3.) An able letter by Professor Kölliker, in the October number of the same journal, p. 374; (4.) A reply by Dr. Bennett to the same, p. 377; (5.) Dr. Bennett's work on *Leucocythæmia;* (6.) Dr. Bennett's Lecture (vi.) in the *Lancet* of April 4, 1863.

(*b.*) *Causation.*—The causes which bring about leucocythæmia are not well recognised. It seems several times to have suggested itself to Virchow that acute inflammatory processes may lay the foundation of the morbid state; and in an interesting review of his writings in *The Brit. and For. Med.-Chir. Review* for July, 1857, there is related a case of the lymphatic form of the disease, whose origin obviously dates from inflammatory swellings of the lymphatic glands after exposure to cold and wet. I saw a similar case in Guy's Hospital in July, 1863. It occurred to a man after exposure to cold and wet on Epsom Downs at the time of the races there. My friend Dr. Robert Paterson, of Leith, recently President of the Edinburgh College of Physicians, from whose teachings in the Edinburgh Royal Infirmary I profited as a student, records several cases of leucocythæmia in connection with pregnancy in the *Edinburgh Medical Journal* for June, 1870, p. 1073. He there shows how this disease is able to linger long in the constitution in a masked or subacute form, unmarked by any easily recognised symptom, until the occurrence of some change-producing event in the constitution, such as childbirth, after which the rapidity of its fatal course was to be measured, not by days, but by hours. His observations are borne out by Vidal, who says that pregnancy in four cases out of ten is the commencement of leucocythæmia. The age at which the disease has been most common is between thirty and forty; but it has been met with under ten and over sixty.

(*c.*) *Morbid Anatomy.*—The morbid state expressed in the definition has been observed by many physicians and pathologists under various complex conditions; and hence a variety of opinions has been entertained regarding it in Germany, England, and France, by Vogel, Remak, Henle, Nasse, Weber, Rokitansky, Kölliker, Parkes, Jenner, Gulliver, Piorry, Bichat, Velpeau, and others, who have described cases since Bennett and Virchow first wrote on the subject. The increase of the colourless corpuscles of the blood, which is the prominent character of this disease, does not seem in any case to have existed by itself. There is usually a diminution also of the red corpuscles. Other morbid states, or some change-producing event in the constitution, such as childbirth (ROBERT PATERSON), precede, co-exist with, or succeed the augmentation of the colourless corpuscles. The most frequent complication consists in the enlargement of the spleen. This enlargement is so constant that its existence, if not otherwise accounted for, would at once indicate that leucocythæmia prevailed, and would suggest a microscopic examination of the blood. In nineteen cases Vogel writes that the splenic enlargement was present in sixteen; and in three cases the weight of the spleen was estimated at more than seven pounds. White corpuscles accumulated in the spleen and in the marrow of the bones. Constituents foreign to normal blood have been found by Scherer in a qualitative analysis of the blood in a case of leucocythæmia where the spleen was enlarged. These consisted of lactic, acetic, and formic acids, gelatine, and a peculiar substance (hypoxanthin), to the amount of from ·4 to ·6 per cent. This same substance exists naturally in the pulp of the healthy spleen (SCHERER). This form of the disease has been named *splenœmia* by Virchow. In it the globules (white) predominate, which are peculiar to the elements of the spleen. The *liver* is also frequently (thirteen times out of nineteen cases) enlarged or otherwise altered, but not to so remarkable a degree as the spleen. Affections of the *lymphatic glands* predominate in some cases rather than enlargements of the *liver* or the *spleen.* Virchow considers that some kind of lymphatic *diathesis* prevails,—that there is a progressive inclination of the lymphatic system to the formation of the lymphatic elements. In some instances observed by him there was a new formation of glandular tissue, or the glandular tissue tended to grow beyond the pre-existing boundaries of the glands. He has observed this development of lymphatic gland-tissue to take place in the liver in a remarkable case which contained numerous small whitish granules, about the size of the natural lobules of the liver, and which exhibited under the microscope nuclear and cellular elements quite like those of the lymphatic glands. This infiltration of the liver followed the ramifications of the portal vein. In one of these cases he observed a similar alteration in the kidney. This constitutes the *lymphatic* form of *leuchœmia,* or *lymphœmia,* first described by Virchow in 1847. In such cases the elements of the lymphatic glands prevail in the blood in the form of innumerable round granulated nuclei, generally provided with nucleoli of the size of the usual nuclei of the lymphatic glands. Here and there are also to be seen cells, consisting of such a nucleus, surrounded by a membrane closely attached to it. There are three possible conditions under which these elementary cells in the blood may originate, —(1.) They may multiply in the blood by the subdivision of pre-existing cells; (2.) they may be primarily introduced into the blood through the lymph or chyle, which is conceived to convey the developed as well as the undeveloped globules derived from the lymphatic glands, the spleen, and its connecting tissue; (3.) that they are formed on and detached from the walls of the blood-vessels has not yet been proved (VIRCHOW).

Virchow regards the colourless blood-corpuscles as simple cells, without any specific character, whose transformation into red globules cannot take place; that they therefore form a relatively superfluous constituent of the blood—a kind of excess or waste. The transformation of lymph-globules into red globules takes place before passing into the general circulation; and it appears that if a certain cell, when passing into the blood, has gone beyond that stage of development, it is ever afterwards unfit to undergo its specific coloured metamorphosis. The idea that the cells are of a purulent nature has been now abandoned; and there is no evidence to support the doctrine that they are the result of *pyœmia.* Besides the spleen and the lymphatic glands, the other blood-glands, such as the *thyroid gland* and *suprarenal capsules,* are occasionally degenerated, as well as Peyer's glands and the mesenteric glands; and leucocythæmia may arise from disease in them as well as from the spleen or lymph glands.

Symptoms.—In the majority of cases there are indications of general ill-health; and the most prominent symptom has been tumefaction of the abdomen, depending upon an enlarged spleen and liver. Ascites and anasarca of the lower half of the body are not unfrequently present; and a tendency to œdema may commonly be observed, the general surface of the body being usually pale. Transitory pains are frequently experienced in the abdomen. Intestinal disorders are also present, such as vomiting, constipation, or diarrhœa, and jaundice is not unfrequent; but diarrhœa is one of the most dangerous complications, and the most difficult to arrest or control. A considerable amount of dyspnœa may prevail, which cannot be accounted for by elevation of the diaphragm merely. Hæmorrhage often occurs in the form of epistaxis, or takes place from the gums. A persistent increased secretion of uric acid has been observed in the urine. The disease generally runs a chronic course, and a high degree of emaciation accompanies it. Leucocythæmia is usually well established before it is noticed, and before any remarkable disturbances in the general health have occurred. It is not till towards the fatal termination that fever sets in, which then assumes the hectic type.

The **Diagnosis** consists in demonstrating the extreme increase of the colourless blood-cells, with diminution of the red, which may be done as follows:—(1.) By microscopic examination of the blood, for which a single drop is sufficient, most conveniently taken from a needle puncture in the finger, and examined under a power of at least 250 diameters. If the disease exists, the colourless corpuscles will be seen to form a sixth, a fourth, or even a half or more of the numbers of the whole corpuscles. (2.) If a large quantity of blood is obtainable by venesection or by the cupping-glasses, and freed from fibrine by heating, and placed in a high narrow little glass, so that the corpuscles sink to the bottom, the upper part of the mass looks whitish-coloured, like milk. The milky character does not vanish on agitation with ether, and is not produced by fat-globules suspended in the blood-serum, but by the prodigious number of the colourless blood-corpuscles. (3.) The clot of leucocythæmic blood shows on its surface greyish-white granulations, which, being observed under the microscope, are seen to consist almost entirely of colourless corpuscles, distinguishing the condition in leucocythæmia from the ordinary buffy coat; and the separated serum being clear and not turbid, distinguishes the condition from a fatty condition of the blood. (4.) In the dead subject there are found in the heart and in the great veins large soft semi-fluid greyish-yellow coagula, which, on microscopic examination, are seen to consist almost entirely of colourless corpuscles (VOGEL).

Prognosis.—Hitherto no instance of cure is known. All the cases progress to a fatal end in about thirteen months—the minimum duration being three months, and the maximum about four years. Death takes place gradually from asthenia and exhaustion.

Treatment.—The most varied remedies have been tried without checking the increased formation of colourless corpuscles; but it is suggested that if it is possible to discover the glandular or splenic affection early, before the alteration of the blood has made much progress, it is probable that the disease may be averted. Tonics, nutrients, and stimulants are indicated to support the system. The use of the nitro-muriatic bath ought not to be neglected, and the indications given under anæmia and chlorosis may be followed. *Phosphorus* has been administered with some success by Drs. Broadbent and Wilson Fox. Sir Wm. Jenner administered the drug in capsules containing one-thirtieth of a grain of phosphorus, two or three times a day for four months, without appreciable benefit to the patient. The results generally are unsatisfactory as regards any treatment for these cases.

PECULIAR ENLARGEMENT OF THE SPLEEN AND GENERAL HYPERTROPHY OF THE LYMPHATIC GLANDS—*Syn.*, HODGKIN'S DISEASE, GENERALISED LYMPHADENOMA.*

Definition.—*A disease characterised by a peculiar white deposit in the spleen, sometimes also in the liver, kidney, and lungs* (WILKS), *and by an enormous enlargement of the lymphatic glands throughout the body, accompanied during life by a remarkable anæmia and disposition to anasarca.*

Pathology.—(*a.*) *History.*—Although this disease has not been recognised in the nomenclature of the College of Physicians, yet it seems to be an affection presenting as striking peculiarities as any in the Nosology, and therefore deserving of a distinct appellation and description (WILKS). It arrested the attention of Drs. Bright and Hodgkin many years ago; and they also recognised the frequent association of diseased lymphatic glands with it. Dr. Bright, indeed, refers to the circumstance as having been originally pointed out by Dr. Hodgkin in Vol. XVII. of the *Medico-Chirurgical Transactions* (1832). Dr. Wilks considers that the subject has got into a false position, mainly in consequence of having been referred to in connection with lardaceous disease. I fear I have aided in this confusion; and therefore I hope to aid Dr. Wilks, by thus endeavouring to remove the subject from the false position in which it has been placed by giving here a summary of the pathology of the affection as given by him (*Guy's Hospital Report*, Vol. XI., p. 56).

(*b.*) *Causation.*—Whether the disease is constitutional from the commencement, or whether the system is infected from a local source, is a question yet unsolved. It may commence in one part, and from this, as a focus of contamination, be propagated through the body; or it may be constitutional from the beginning. It occurs at all ages up to thirty years.

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* A disease not recognised in the nomenclature of the College.

(*c.*) *The Morbid Anatomy* of this disease is not to be confounded with lardaceous disease or any other morbid process. The enlarged glands may be met with in various forms of disease, and sometimes with lardaceous disease; and thus the two affections may seem to have a relationship they may not really possess. The lesion appears to have a likeness to tubercle on the one hand, and to cancer on the other. It is a disease *sui generis*. The enlargement of the glands "appears to be a primitive affection of these bodies; and there is no reason to suppose it is due to *inflammation* or *scrofula*, nor attributable to the formation of any adventitious structure. It appears, in nearly all the cases, to consist of a pretty uniform texture throughout, and rather the consequence of a general increase of every part of the gland, than of a new structure developed in it (HODGKIN). More recently it has been found that a marked increase in the interstitial stroma of the lymphatic glands takes place. The condition is that of fibroid indurations, or irritative hypertrophy, the corpuscular development of the growth taking the form of the adenoid tissue of His. The gland-cells have large round nuclei. The enlarged lymphatic glands may attain an enormous size, and the spleen at the same time be greatly enlarged. "In conjunction with this affection of the absorbent glands is the state of the spleen, which is strictly pervaded by defined bodies. We might suspect that the bodies in the spleen, like the enlarged glands, were due to an enlargement of a pre-existing structure—an idea which may derive some support from the fact that, although in the human spleen no glandular structure is distinguishable, in those of some inferior animals a multitude of minute bodies exist which appear to be of that nature. Malpighi, indeed, considered the acini to be glands" (WILKS, *loc. cit.*, p. 59). Generally, the condition is one of hypertrophy of the splenic pulp, with increased growth of the reticulum (SANDBY). In the cases observed by Dr. Wilks, the new material has been found in the liver, kidney, and lungs besides in the spleen. The peculiarity of the affection consists in the whole glandular system being specially affected; and it must take its place in the rank of malignant diseases, or amongst those affections which are characterised by new growths in the system. The peculiar symptoms are to be referred to the universal affection of the glands. They appear to be affected for a considerable period, perhaps many years, before the system suffers, and that subsequently the spleen becomes specially involved, and afterwards the other organs. It is possible that propagation to the spleen takes place in the course of the lymphatics, thereby affecting the corpuscles of the spleen, which are intimately connected with the absorbent system. The deposit in Glisson's capsule of the liver may be transmitted through the same channel. In malignancy the disease takes a place between cancer and tubercle. An increase of white blood-cells is not uncommon in cases of cancer. Dr. Wilks has never met with well-marked *leucocythæmia* in Hodgkin's disease, except in one case where the spleen was hypertrophied; and only one case where lymphatic enlargement and lardaceous disease were associated.

Symptoms.—General ill-health, paleness, and sallowness of complexion precede any other signs. Lymphatic glands are subsequently found enlarged in the neck, axillæ, and groins. Weakness is felt, which increases, and the patient can no longer walk nor run up a stair. He totters in the legs from feebleness. Sexual appetite is lost, and flesh is lost; so that emaciation with marked anæmia exists—a pale sclerotic and a feeble pulse. The legs become œdematous. Irregular and high temperature forms an element in the clinical characteristics of the disease. Dr. Murchison (*Pathological Transactions*, Vol. XXI., page 372) records a case in which the lymphatic hypertrophy was remarkably general throughout various organs of the body, and the periods of growth of the glandular enlargements were marked by almost *periodic febrile paroxysms*, continuing through a period longer than two years, each paroxysm being of ten days' duration, and recurring about every four weeks. In the single paroxysm observed, the evening temperature attained 104°. In another case, by the same writer (*Ibid.*, Vol. XX., p. 198), a temperature is recorded varying between 101° and 104° through fourteen days. Again, in a patient of Dr. Bennett's (*Ibid.*, Vol. XXII., p. 70), the usual temperature varied from 100° to 102°, but 104° was once registered, the range of temperature maintaining a relation with the bronchial irritation and dyspnœa. In Dr. Taylor's case (*Ibid.*, Vol. XXV., p. 246), the temperature through twenty-three days varied between 100° and 102°. In one of Dr. Greenfield's cases (*Ibid.*, Vol. XXVII., p. 275), during the second month, through which the patient was under observation, the temperature varied from 100° to 102·6°, with no constancy as to the greater elevation in the morning or evening; and in a case by Mr. Haward (*Clinical Soc. Trans.*, December 10, 1875), the evening temperature during the latter part of the case was usually 102°, and subsequently 103°; in the morning, 98°. In Dr. Russell's case, the record of temperature extended over sixty-six days, with a temperature of 102° Fahr., rising to 105° through the next ten days, the morning temperature being generally 1° or 1·5° lower. In thirty-six hours a fall of 7° Fahr. took place—then again a rise for seven days up to 104°. Pulse varied from 100 to 112.

Prognosis is generally unfavourable; and no special treatment can be indicated beyond that laid down in cases of anæmia and of leucocythæmia. Death occurs through the derangement of the structure and functions of the glands.

LARDACEOUS SPLEEN—*Syn.*, AMYLOID DISEASE; WAXY SPLEEN.

LATIN EQ., *Lien Lardaceus*—Idem valent, *Morbus Amylodes*, *Lien Cereus*; FRENCH EQ., *Dégénérescence Lardacée*—Syn., *Maladie Amyloïde*; GERMAN EQ., *Speckige Entartung der Milz*—Syn., *Amyloide Entartung*; ITALIAN EQ., *Milza Lardacea*—Syn., *Malattia Amiloide*.

Definition.—*A disease in which the texture of the spleen, and especially the Malpighian sacculi, are filled with lardaceous material, so that it is much larger and heavier than in health.*

Pathology.—(*a.*) *Morbid Anatomy.*—This condition of the spleen is frequently associated with a similar condition of other organs, especially the liver and the kidney. It is characterised by great firmness, a peculiar waxy-like consistence, and with a distinctness and transparency of the Malpighian sacculi which are not usually obvious (W. T. GAIRDNER). Of all the viscera and tissues of the body, the spleen is the one which is comparatively the most frequently affected by this lesion, although it was generally unknown up till 1853–4. Of the bodies examined in the Royal Infirmary of Edinburgh it was observed in 10 per cent. of all. In the most extreme cases the spleen is enlarged, and has a swollen aspect. Its weight and density are greatly increased. It feels to the touch like the consistence of wax and lard, and its section shows a dry and smooth surface. After exposure to the air, its pulp may become of a bright red colour; and then the Malpighian sacculi appear large and distinct, forming round, greyish granules about the size of a pin's head, slightly prominent, and so hard that they may be picked out by a knife. The pulp is diminished in quantity, and may be entirely absent; so that the sacculi are crowded together, and the tissue in their vicinity appears as a continuous mass of a globular form. A small arterial twig can sometimes be seen passing into or through these Malpighian sacculi at their central part. Under a low power (40 to 60 diameters) the Malpighian sacculi appear as large clear spaces, of a circular or oval form, surrounded by the dark-red pulp. Under the higher powers (above 250) the alteration is in the corpuscles of these sacculi, which are converted into, replaced, or added to, by masses of a colourless, dense, highly translucent and homogeneous material. A careful examination may disclose the outlines of irregular cell-forms. But these masses, owing to their roughness, are not easily broken up into the particles which compose them (SANDERS). The translucent parts are little acted on by reagents (acids, alkalies, alcohol); but if alkalies, such as *liquor potassæ,* be first applied to a microscopic section the iodine test will fail until the section is neutralised by acid. Its characters are marked and permanent, so that there is no difficulty at any time in recognising or identifying the characters of lardaceous disease in the spleen. Several varieties may be noticed. Two forms at least are distinctly indicated by Virchow, Sanders, and Wilks (although the latter observer says three) :—(1.) *The form in which the Malpighian sacculi are the structures prominently affected.*—They are sometimes large, and sometimes very small. In the former state we recognise the sago-like granules described by Virchow. In the latter case they are best seen after exposure to the air for a little time, or after having been well washed in water, and then put in alcohol or into a solution of chloride of zinc, which brings out the waxy appearance very distinctly. In the most extreme cases they never occupy more than about half the bulk of the organ, the intervening pulp-substance being healthy. (2.) *A form in which the pulp-substance and trabeculæ are mainly affected, leaving the sacculi intact.*—It presents the same waxy consistence, the smooth dry section, and other characters of the lesion; but the sacculi are obscured by the peculiar translucent substance which pervades the pulp, and which looks as if melted tallow had been poured into the trabecular spaces (WILKS). Histologically, there is no real difference between these two forms, because the corpuscles of the sacculi and of the surrounding pulp-substance are now known to be identical (Busk and Huxley, in Wedl, p. 247).

The disease seems to commence—(1.) In the arterial capillaries, where the little lateral bulgings are in connection with the Malpighian corpuscles. (2.) These sacculi contain at first normal splenic cells; but at a later stage the contents of the sacculi become irregular and granular, and corpuscles of a gelatinous lymphy appearance become changed into the minute masses of the waxy substance. (3.) Subsequently the trabeculæ and pulp-struma become affected (Busk and Huxley, as above).

Some cases of albuminoid disease related by Dr. Jenner are evidently of this second nature.

From this description, and from what has been written in Part I., Vol. I., on the subject of *Lardaceous Disease* (q. v.), the term "*degeneration*," which has hitherto been generally used, is quite inapplicable. Rindfleisch more correctly describes it as an infiltration. The spleen is added to in bulk and weight by a new material; its textural elements are not replaced, which is the nature of degeneration to effect. Neither has it been shown that the new material is "*amyloid;*" but it is rather of the nature of albumen. The phrase "*amyloid degeneration*," therefore, is one which is incorrect in both its terms (Wilks), and one which ought no longer to be used. It is only by an extended inquiry into the class of cases in which lardaceous disease is found that we can hope to learn anything satisfactory as to its pathology; and the reader is referred to the excellent reports upon ninety-six cases already given by Dr. Wilks, in Vol. IV. (1856) and Vol. XI. (1865) of *Guy's Hospital Reports;* and by Dr. Pavy, in Vol. X. of the same valuable series. These cases prove indisputably that lardaceous disease of the spleen, or any other organ, is one implying a long-standing and deep-seated cachexia; and is seen, in its most intense form, after a protracted caries and necrosis of bone, having its origin in scrofula, or syphilis, or even external injury, where the injury leads to protracted bone disease. Hence the question is still undecided, whether the disease arises from a local source, such as the injured bone and the morbid processes going on in it, or whether it is a constitutional general disease? Wilks gives several instances, such as the following:—"A strong young man, in good health, falls and strikes his hip; a disease is thus set up which causes his death at the end of two years: his organs were then found to be lardaceous" (No. 89 of cases in *Guy's Hospital Reports*, Vol. XI., p. 47). It is evident that the question is one of great practical importance to the surgeon.

Symptoms.—As regards the spleen, lardaceous disease is only known to exist by its discovery after death—no symptoms that can be recognised (unless enlargement and hardness) having been perceived as constantly associated with the lardaceous affection.

SECTION III.—DISEASE OF SUPRARENAL CAPSULES.

ADDISON'S DISEASE—*Syn.*, BRONZED SKIN, MELASMA ADDISONII.

LATIN EQ., *Morbus Addisoni*—Idem valent, *Cutis Ærea, Melasma Addisoni;* FRENCH EQ., *Maladie d'Addison, Melasma Addisonii;* GERMAN EQ., *Addison'sche Krankheit*—Syn., *Malasma Addisonii;* ITALIAN EQ., *Malattia dell' Addison*—Syn., *Melasma dell' Addison.*

Definition.—*Disease of the suprarenal capsules, with discoloration of the skin; or, a morbid state which establishes itself with extreme insidiousness, whose characteristic features are anœmia, general languor and debility, and extreme prostration, expressed by loss of muscular power, weakness of pulse, remarkable feebleness of the heart's action, breathlessness upon slight exertion, dimness of sight, functional weakness and irritability of the stomach, and a peculiar uniform discoloration of the skin, which becomes of a brownish olive-green hue, like that of a mulatto, occurring in connection with a certain diseased condition of the suprarenal capsules. The progress of the disease is very slow, extending on an average over one year and a half, but may be prolonged over four or five. The tendency to death is by asthenia, the heart becoming utterly powerless, as if its natural stimulus—the blood—had ceased to act.*

Pathology.—(*a.*) *History.*—The pathological significance of morbid states of the suprarenal capsules was brought prominently before the profession, both in this country and on the Continent, by the original observations of the late Dr. Addison, then the Senior Physician of Guy's Hospital. The cases recorded in the medical journals since Dr. Addison wrote, which connect uniform discoloration of the skin (a condition now known by the name of "bronzing") with various morbid states of the *suprarenal* bodies, are now so numerous that, as a clinical fact, the connection cannot be disputed; but the exact relationship and pathological significance of the morbid states thus connected are still open questions, especially as regards the pathology of the constitutional cachexia which exists. Morbid states of the *suprarenal* capsules are not always attended with bronzing of the skin. If the cases recorded are carefully analysed, symptoms and phenomena of a very important kind have been lost sight of in describing this constitutional disease, while an undue importance has been placed upon the bronzing of the skin. It is to the cachexia that Dr Addison calls special attention; but his commentators have been carried away by the inquiry regarding the colour of the skin and its connection with the capsular disease. These writers have overlooked the more important portion of his observations, and have been induced to consider rather the *causes* and *nature* of the bronzing of the skin. This coloration when established may be received as a most valuable symptom of a prevailing constitutional cachexia, in which the abnormal deposit of pigment is associated with anæmia and intense prostration, with the phenomena stated in the definition. But this bronzing must appear of very secondary importance compared with the symptoms and pathology of that peculiar cachexia which attends the cases of *suprarenal* capsular disease, as described by Drs. Addison and Wilks. There seems to be no doubt that the cases Dr.

Addison described belong to the class of diseases marked by anæmia, and that they are similar in many respects to forms of anæmia already noticed, and more especially to leucocythæmia. "In almost all the cases there would seem to have been great deprivation of the coloured constituents of the blood, as manifested by the pallor of those parts of the skin not involved in the bronzing, the great flabbiness of the muscles, and the pearly state of the conjunctivæ" (HUTCHINSON). The blood has been subjected to but few examinations. "In two only," of the cases, writes the same author, "was the blood examined, and in both of them it was found to be loaded with white corpuscles." In all the cases a most remarkable and fatal cachexia prevails, and the value of Dr. Addison's observations consists in showing that a peculiar bronzing of the skin, combined with *asthenia* —of which emaciation is not a necessary accompaniment—attends this cachexia, and indicates organic disease of the *suprarenal* capsules associated with this constitutional state. His observations are at the same time of the greatest value, as showing how well-directed pathological inquiry may advance the science of physiology; for the cases detailed throw some light on the influence, at least, of the *suprarenal* capsules in the maintenance of health.

In the volume of *Guy's Hospital Reports* for 1862, Dr. Wilks has given a most able, interesting and impartial account of the progress of our knowledge regarding this disease since the time Dr. Addison wrote. His own observations entirely uphold the argument which Dr. Addison attempted to develop; and the cases brought forward by him in that report appear to substantiate, in a great measure, the original facts on which Dr. Addison's history was based. Nevertheless, Addison's views have by no means received the support of the profession at large. Dr. Wilks thinks that Dr. Addison in some measure contributed to this scepticism, by including among its original cases some which did not present the true features of the disease; and the great merit of Dr. Wilks's paper is, that it more clearly defines and renders precise the pathological characters of the disease which Dr. Addison desired to describe. He believed, at the time he published his work, that *any disease* which affected the integrity of the *suprarenal capsules* would be attended by the remarkable phenomena originally described by him. This was an error: for all subsequent observations have shown that no recorded instance of the affection has been connected with cancer, or with any other kind of disease of the organ than that found in the genuine cases of the disease which he first described, and which constitutes the true form of the malady, as Dr. Wilks has demonstrated.

(*b.*) *Causation.*—It has been described as due to extension of inflammation from neighbouring diseased or injured parts to the suprarenal capsules; to strains or blows in the back; over-exertion; also, to nervous shock, grief, or anxiety. It is more common among males than females, and especially in connection with such manual labour as entails exposure to bodily injury from accident or over-exertion.

(*c.*) *Morbid Anatomy.*—In malarious, malignant, and cachectic diseases it is usual for the serum of the blood to assume a dark and dirty hue, and ultimately the cutaneous surface comes closely to approach the colour of jaundice, differing from it only in being more lurid and dusky; and it is believed that the hue of the skin, which becomes of so dark a tinge in some malignant organic diseases, is due to the admixture of morbid matters absorbed from the seat of local mischief, which so tinges the serum of the blood that the *rete mucosum* is rendered dark. If it is true that in this cachexia the suprarenal capsules are always diseased, and if it be true, as M. Vulpian has found (*Med. Times,* October 4, 1856), that the *suprarenal capsules* differ from all other organs in the body, in the presence of a substance which has two peculiar reactions—one with the perchloride of iron, and the other with the tincture of iodine, the first of which gives a dark-blue tint—is it not probable that the coloration of the skin in *suprarenal capsule* diseases may result partly from—(1.) The cachectic state; (2.) the organic lesion of the *suprarenal capsules;* and, (3.) the reaction of the peculiar substance in these bodies upon the iron of the blood, which the morbid organic changes in them have allowed to mingle with the circulating fluid? It is, unfortunately, the *suprarenal capsules* only which have been carefully examined in the cases recorded; and in connection with the cachexia and the "bronzing," the lesions they exhibit have been described as of a variable character, consisting of—(*a.*) Acute or recent inflammation ending in abscess; (*b.*) atrophy with concretions; (*c.*) the conversion of the organs into indurated fibroid enlargements; (*d.*) tuberculous deposition in various stages; (*e.*) carcinomatous deposition. Sometimes the lesion appears to have been secondary to morbid conditions, apparently of a similar nature, in other parts, all of which must therefore be interpreted as several local expressions of the constitutional disease which prevails; and occasionally the capsules seem to have been the only structures in which lesions were detected; while the degree of "bronzing" of the skin appears to have been proportionate to the length of time which the *suprarenal capsules* are presumed to have been diseased; but neither the time nor the number of cases in which such a proportion can be traced is accurately made out. The general symptoms are those of a person constitutionally diseased, *not* always proportionate in their severity either to the nature or to the extent of the disease in the *suprarenal capsules.* The *post-mortem* appearances seem to have been in some instances associated with the tuberculous diathesis: but in the best-marked cases there has been no appearance of tubercle in any part of the body (WILKS); and in no case of general tuberculosis has Dr. Wilks ever met with an instance where the *suprarenal capsules* were affected in the manner of Addison's disease. In some cases the spleen was much enlarged, the kidneys pale, and in the last stage of fatty degeneration (TAYLOR). Out of 500 cases of *post-mortem* examinations of all kinds of cases, made at Guy's Hospital by Dr. Wilks, only two instances were observed in which, the *suprarenal capsules* being morbid, the skin did not betray the lesion by "bronzing." In come cases the mesenteric glands are stated to have been enlarged; and calcareous concretions have been noticed in the *medulla oblongata.*

In one case which, through the kindness of my first teacher, the late Dr. William Munro, of Dundee, I had an opportunity of carefully inspecting after death, in June, 1856, the following lesions existed:—"The body generally was anæmic. The dark colouring of the skin was most conspicuous in the vicinity of the knees, and on the lateral and posterior regions of the neck. The deposits of pigment gave to the parts a dirty sordes-like appearance where the mucous membrane of the skin meets the lips, and especially at the angles of the mouth. The heart was small and flabby. Tuberculous deposits were sparingly disseminated throughout the apices of both lungs. The spleen, the liver, and the kidneys were severally adherent to the adjacent parts, but their structures appeared normal. The suprarenal capsules were morbid, and the sympathetic nerves from the lesser *splanchnic* were greatly increased in size, as were the ganglia of the *solar plexus* towards the side of the suprarenal most diseased and in contact with it. The texture of the nervous parts was of a bright rosy hue, as if under the influence of vascular excitement. The mucous membrane of the mouth was thin, pale, and bloodless, the labial and buccal glands shining prominently through. The stomach and glandular substance of the intestinal tube were uniformly thin throughout. In the stomach the solitary gastric glands were remarkably prominent, while the mucous membrane generally was wasted and atrophic. Microscopic sections from the jejunum and ileum showed the villi remarkably attenuated, and the mucous membrane very readily separated from the adjacent muscular part of the gut. The tubular glands of the mucous membrane of various parts examined were almost entirely gone, and their place supplied by granular amorphous material. The average specific gravity of the mucous membrane of the intestines was 1·040.

The special lesion of the capsules in Addison's disease is of one form only—namely, that which was seen in the earlier cases described by Addison, and which he simply styled as "scrofulous" (WILKS). Judging from the material alone, it would be difficult to form an opinion of its nature, seeing that a degenerating inflammatory substance would produce a very similar appearance to a so-called scrofulous one. It is only in exceptional cases that any well-marked tubercular deposit has been found in other viscera. "When the disease is recent, the organ is somewhat enlarged, and changed into a material which is semi-translucent, of a grey colour, softish, homogeneous, and which, when examined microscopically, is found to be without structure, or sometimes slightly fibrillated, or containing a few abortive nuclei or cells. This lardaceous kind of material is the first deposited, and resembles what is often seen in the early stages of scrofulous enlargement of the lymphatic glands; subsequently it undergoes a decay or degeneration, as in these glands, and changes into an opaque yellowish substance; and thus the two materials are constantly found associated. At a later period, as in a scrofulous gland, this may soften into a putty-like matter, or it may dry up, leaving the mineral part as a chalky deposit scattered through the organs. These, then, are the changes —first, the deposition of a translucent, softish, homogeneous substance; subsequently the degeneration of this into a yellowish-white opaque matter; and afterwards a softening into a so-called abscess, or drying up into a chalky mass. Occasionally, also, some fibrous tissue may be found round the organs, being the product of an inflammation which has united them to the kidney, liver, and adjacent parts" (WILKS). Some years are necessary for the production of these changes. The state of the skin which has been termed "bronzing" resembles the colour of a mulatto, or of a bronzed statue from which the gloss has been rubbed off. It has been examined microscopically by Dr. Wilks, Mr. Tuffen West, and M.

Robin, of Paris. The sections show a layer of very distinct pigment-granules in the *rete mucosum,* limited to that structure, and exactly resembling that of a negro. The pigment is deposited in granules, but in some instances coloured cells are visible.

Before detailing the symptoms observed in the remarkable cachexia from which these lesions result, it is necessary to draw attention to our incomplete knowledge regarding many points in its pathology, and especially regarding the state of the blood, and the morbid tendencies of the relatives and progenitors of the patient. While the apparent connection of bronzing of the skin with lesions in the *suprarenal capsules* appears to be evident, our information is of the most deficient kind regarding the chronological sequence of the various symptoms which indicate the establishment of the constitutional disease.

Symptoms.—As in the forms of anæmia already described, so in this disease the patient has considerable difficulty in assigning the number of weeks, or even months, that may have elapsed since he first experienced indications of failing health and strength; and the rapidity with which the local lesions develop themselves varies in different cases—a few weeks being sometimes sufficient to break up the powers of the constitution, or even to destroy life—an event the more speedy in proportion to the rapid and extensive destruction of the *suprarenal bodies* (Dr. ADDISON). The important features of the disease are,—a progressive feebleness of the patient, without any apparent or known cause (*asthenia*), anæmia, general languor and debility, remarkable feebleness of the heart's action, irritability and weakness of the stomach, and a peculiar change of colour of the skin. In most of the cases the early sequence of symptoms appears to have been gradual and almost imperceptible indications of failing health and strength, consisting chiefly of languor, weakness, and indisposition to either bodily or mental exertion, the appetite being impaired or entirely lost, the white of the eyes becoming pearly, the pulse small and feeble, perhaps somewhat large, but excessively soft and compressible. The body wastes, without presenting extreme emaciation or the dry and shrivelled skin usually associated with protracted malignant disease. Slight pain or uneasiness is from time to time referred to the region of the stomach, and there is occasionally actual vomiting. With every sign of feeble circulation, anæmia, and general prostration, "neither the most diligent inquiry nor the most careful physical examination throws the slightest gleam of light upon the precise nature of the patient's malady; nor do we succeed in fixing upon any special lesion as the cause of this gradual and extraordinary constitutional change" (ADDISON). With a more or less manifestation of the symptoms already enumerated, a most remarkable and characteristic discoloration takes place in the skin. It pervades the whole surface of the body, but is commonly most strongly manifested on the face, neck, superior extremities, penis, and scrotum, in the flexures of the axilla, and round the navel. It presents a dingy or smoky appearance, of various tints or shades of deep amber or chestnut brown; and in one instance the skin was so universally and deeply darkened as to resemble a mulatto. This distribution of pigment is not confined to the skin, but is also visible in the mucous tracts, as well as in some other structures. An interesting case occurred in the practice of Professor Biermer, of Berne (November, 1861), in a weakly girl, eighteen years of age. After continued illness of a tubercular kind, the dark colour of the skin commenced, and increased in intensity with the decrease in the weight of the body till death followed. The eyes of this patient happened to be examined with the ophthalmoscope, although vision was not disturbed. A bluish-black colour was everywhere visible through the sclerotic, and a peculiar pigment existed in the interior of the eye. In a plane of a uniform red-brown colour there were bluish-black spots, corresponding to the intermediate spaces of the *vasa-vorticosa,* from which the retina was distinguished by its delicate white appearance. The disease thus originally described by Addison may be associated, like "*morbus Brightii,*" with the expression of several distinct local lesions. The discoloration consists of stains in the lining of the cheeks, and a decidedly blackish tinge of the mucous membrane of the lower lips, as if after eating mulberries. Dark areolæ become developed beneath the orbits, much marked towards the middle line of the face. In one case, under Dr. Barlow, loss of consciousness and what are termed "fainting fits" were the earliest symptoms noticed (*Med. Times,* January 24, 1857).

Although the change of colour of the skin would seem to be a most marked and constant symptom, still it does not appear that this change is one of the earliest symptoms of the disease. On the contrary, there are good grounds for believing, both from the history of the cases and from physiological experiment and observation, that the change in the colour of the skin, which has been termed "*bronzing,*" does not come on for a long time; that from its nature as a pigmentary change it is of slow production; that while, in almost all the well-marked cases in which it has occurred, the symptoms had existed from one to three years, in other cases where it did not exist it has been alleged that time was not afforded between the establishment of lesions in the suprarenal bodies and the fatal issue for the production of the pigmentary deposit. The change of colour seems to depend on the chronicity of the disease; so that, if the disease progresses rapidly, no discoloration is observed, the phenomena being simply those of asthenia. One very important point is thus remarkably deficient of illustration—namely, *the early symptoms of this cachexia* independent of *bronzing of the skin.*

Gastro-intestinal disturbance prevails during life; and a condition of mucous membrane is found after death which may be associated in its pathology with this irritation. The stomach is often ecchymosed, and the glands of the intestines enlarged. In several cases Brunner's glands in the duodenum, and the solitary glands in the lower end of the ileum and in the colon, were very prominent.

Prognosis.—From what has been shown relative to the disease, it would appear that when *bronzing* of the skin has been established, a sign of disease has been discovered when it is too late to be of any service, for all the cases appear to have terminated fatally in which this state was unequivocally established. There are good grounds for believing that Addison himself entertained the belief that death in such cases may be due to the implication eventually of the ganglionic nerves. Some of the symptoms point to this; and of the special phenomena associated with the cachexia, it may be said that the nervous centres are at least impaired. The suprarenal capsules have a large supply of nerves, intimately connected with the trunk of the *sympathetic*, with the *phrenic* and *pneumogastric* nerves, and through these with the *cerebro-spinal centres;* and, as already noticed, the morbid changes tend to implicate these nerves, extending even to the *semilunar ganglia* and *solar plexus* (see p. 1030, *ante*). It has occurred to me also that, in cases of this disease, all the viscera ought to be examined as to the reaction of their minute blood-vessels with iodine, as there are some phenomena which seem to indicate the not improbable co-existence of *amyloid* degeneration; and the lesion in the *suprarenal capsule* ought especially to be investigated in this direction.

Treatment.—If the disease be recognised in its earliest stages, its progress may to some extent be delayed. The asthenia, the depression, the evidence of local irritation about the suprarenal capsules, and the pathology of the disease generally, point to the necessity for tonic treatment and nutritive diet, the avoidance of all causes of depression, and the benefit of rest in bed, and of such medicinal agents as may relieve the vomiting. *Glycerine*, in two drachm doses, combined with fifteen or twenty minims of the *spirit of chloroform*, and of the *tincture of the sesquichloride of iron*, have been of service (E. H. GREENHOW). This may be varied by the substitution of twenty to thirty minims of the *syrup of phosphates of iron*, *quinine*, and *strychnia*, in place of the *sesquichloride of iron*. The greatest caution is necessary in using purgative remedies, as fatal collapse is apt to follow cathartic medicine.

CHAPTER XXII.

DISEASES OF THE CUTANEOUS SYSTEM.

SECTION I.—GENERAL PATHOLOGY AND CLASSIFICATION OF DISEASES OF THE SKIN.

DISEASES of the skin have been regarded too much as a specialty. But they are now beginning to be looked upon as a class of diseases whose pathology is capable of being investigated and studied like other diseases. Skin diseases are undoubtedly various in appearance; and the same disease does not always continue in the same simple or elementary form.

The *classification* of skin diseases hitherto in use is that which is comprehended in the eight orders of Willan and Bateman; and the characteristics of these orders are embraced in the following definitions of terms in common use in the description of skin diseases:—

ORDER I. PIMPLES.—*Papulæ* are simple solid acuminated elevations of the cuticle, resembling enlarged *papillæ* of the skin. They commonly terminate in a scurf, and sometimes, though seldom, in slight ulceration of their summits.

ORDER II. SCALES.—*Squamæ* consist of cuticle in patches, plates, or laminæ, in which the cells of the epidermis are morbidly adherent, hard, thickened, whitish, and opaque. These scales cover either small papillæ, red elevations, or larger deep red and dry surfaces.

ORDER III. RASHES.—*Exanthemata* are composed of superficial red patches of irregular size, and variously diffused. They disappear under pressure, and terminate by desquamation.

ORDER IV. BLEBS.—*Miniature Blisters*—*Bullæ* differ from vesicles in size, being larger. A large portion of cuticle is detached from the skin by the interposition of a watery fluid, usually transparent. The skin is red and inflamed beneath the blebs.

ORDER V. PUSTULES.—*Pustulæ* consist in circumscribed elevations of the cuticle, and contain pus. They have red and inflamed bases, and are succeeded by an elevated scab, which may or may not be followed by a cicatrix.

ORDER VI. VESICLES.—*Vesiculæ* are small acuminated or orbicular elevations of the cuticle, containing lymph, which, at first clear and colourless, may become amber-coloured, opaque, or pearl-like. Vesicles are succeeded by a scurf or a laminated scab.

ORDER VII. TUBERCLES.—*Tuberculæ* are small, hard, indolent elevations of the skin, sometimes suppurating partially, sometimes ulcerating at their summit.

ORDER VIII. SPOTS.—*Maculæ* are permanent discolorations or stains of some portions of the skin, often with a change of structure. They may be whitish, dusky, or dark.

Such definitions have given the key to the classifications of cutaneous diseases, which are mainly anatomical. They do not attempt to throw any light on the causes producing the various diseases, which are contemplated as so many distinct and individual "unities," their mutual relations being of secondary importance. Clear and accurate in detail, these anatomical definitions have not yet been surpassed; and they have laid the foundation of the objective study of cutaneous diseases.

The basis of classes, as proposed by Willan and Bateman, furnishes outward marks or anatomical characters, which are useful in describing the morbid anatomy of skin diseases; but it has no relation to the causes, to the pathology, nor to the treatment of such diseases. An affection which is papular to-day may be vesicular to-morrow, and pustular eventually. Under the same division, or class, maladies are brought together which nature has stamped with broad and obvious marks of distinction. Febrile diseases are associated with non-febrile; and ailments which are local and trivial are associated with diseases of grave import and deeply rooted in the system. On the other hand, distempers which nature has plainly brought together and connected by striking analogies and resemblances, the methodical arrangement of Willan and Bateman puts widely asunder (WATSON).

In an admirable paper "On the Theory and Classification of Inflammations of the Skin," by the late Dr. A. B. Buchanan, Physician to the Dispensary for Skin Diseases in Glasgow, it is shown that any classification resting on one principle of division only, runs the risk of being, to a greater or less extent, *artificial* and untrue; and to secure a *natural* system, several principles must be taken into account, though greater importance may be attached to some of these than to others (*Edin. Med. Journal*, January, 1863). Skin diseases, like all other diseases, ought to be classed according to their *nature* or *pathology.* The cause of a disease, when known, gives a more accurate indication of its true nature and its means of cure than a knowledge of its anatomical appearances or symptoms, which are mere effects of the cause. So far as can be done, therefore, skin diseases should be classified according to their causes. Not knowing the cause, some other principles must be sought for under which groups may be formed; and in the pathological processes recognised as *inflammations, new formations, hæmorrhages,* skin diseases may be arranged into three or more groups:—(1.) *The inflammatory skin diseases* are those which affect the "*pars papillaries*" of the corium, as well as the "*pars reticularis*" and the subjacent connective-tissue; but the process is not confined to these, for it affects even the epidermis, whose cells are changed in formation and in structure in the same way as all cells are altered by the process of inflammation. (2.) *The skin diseases characterised by new formations* may be regarded as—(*a.*) The productive effects of inflammation; or, (*b.*) the productive effects of such constitutional states as cancer, scrofula, and Bright's disease. (3.) *The hæmorrhagic skin diseases* arise from the escape of blood in small quantities, owing to the rupture of capillary vessels, or to the escape of colouring matter along with the exudations. They are affections peculiar to the cutis; for, although blood may be effused among the epidermic cells, hæmorrhage can only originate from the cutis. On these principles the following classification has been suggested:—

CLASSIFICATION OF SKIN DISEASES (DR. A. B. BUCHANAN).

Class I.—Inflammations.

GROUP I.—SIMPLE INFLAMMATIONS (ALLIED TO *Simple Dermatitis*).

(1.) ERYTHEMA ([a.] *simplex*; [b.] *multiforme;* [c.] *chronicum*—comprehending *papulatum, nodosum, strophulus, squamosum, pityriasis furfuracea, membranacea, rubra*). (2.) HERPES (*simplex* and *zoster*). (3.) URTICARIA (idiopathic, from ingestion of particular kinds of food; from uterine affections; or persistent). (4.) DERMATITIS (idiopathic, as from *burns*, or from *frost-bite;* or symptomatic, as of *erysipelas;* or phlegmonodes, as *furunculus, anthrax, Aleppo tubercle*). (5.) PEMPHIGUS (benign, persistent, and foliaceous).

GROUP II.—ECZEMATOUS INFLAMMATIONS (ALLIED TO *Eczema*).

(1.) ECZEMA (*erythematodes; E. papulosum,* comprising *lichen simplex* and *prurigo; E. vesiculare*; *E. rubrum; E. pustulosum,* comprising *impetigo sparsa, figurata,* and *pilaris; E. lichen; E. squamosum; E. pityriasis*). (2.) ACNE (comprising *A. simplex; A. pilaris; A. rosacea*). (3.) ECTHYMA ([a.] *simplex;* [b.] *chronicum=rupia;* [c.] *gangrenosum*). (4.) PSORIASIS (*punctata, guttata, nummularis, circinata* [lepra], *gyrata, confluens*).

GROUP III.—ULCERS.

(1.) IDIOPATHIC. (2.) SYMPATHETIC. (3.) CONSTITUTIONAL.

Class II.—New Formations.

GROUP I.—HOMOLOGOUS NEW FORMATIONS.

(1.) EPIDERMIC (*epithelial growths,* comprising *callositus, clavus ichthyosis, cornu cutaneum*). (2.) PIGMENTARY (*lentigo, ephelis, moles, melanosis, chloasma, silver stain, leucopathia albinism*). (3.) DERMIC (*cicatrix normal* or *cheloid, cutaneous tumor* [wens], *multiple tumors* [mycosis], *molluscum simplex, condylomata, verruca vulgaris, verruca mollis*).

GROUP II.—HETEROLOGOUS NEW FORMATIONS.

(1.) PSEUDOPLASMS (*lupus,* comprehending *maculosus, tuberculosis, hypertrophicus, exedens, serpiginosus;* and *lepra,* comprehending *maculosa, tuberculosa, anæsthetica, exulcerans*). (2.) NEOPLASMS (*epithelioma, carcinoma*).

Class III.—Hæmorrhages—e. g., *petechiæ, vibices, ecchymosis, purpura.*

Class IV.—Diseases of Accessory Organs—(*a.*) *hair,* e. g., *sycosis, alopecia, calosities, fragilitas, polytrichia;* (*b.*) *nails,* e. g., *atrophy, hypertrophy, onychia;* (*c.*) *sweat glands,* e. g., *miliaria, sudamina, lichen tropicus, dysidrosis, hyperidrosis, anidrosis, chromidrosis;* (*d.*) *sebaceous fluids, seborrhœa, comedo, acne milium, moluscum centagiosum, xanthelasma, steatoma.*

Class V.—Diseases Defined by Uniform Causes.

GROUP I.—PARASITIC DISEASES.
GROUP II.—SYPHILITIC ERUPTIONS.
GROUP III.—ERUPTIONS OF SPECIFIC FEVERS.
GROUP IV.—SCROFULODERMATA.

In the following section, however, the names of skin diseases and the arrangement followed will be that which is given in the nomenclature of the College of Physicians :—

SECTION II.—DESCRIPTION IN DETAIL OF THE MORE COMMON DISEASES OF THE SKIN.

ERYTHEMA.

LATIN EQ., *Erythema;* FRENCH EQ., *Erythème;* GERMAN EQ., *Erythema;* ITALIAN EQ., *Eritema.*

Definition.—*Uniform redness simply, with puffiness of the skin, distributed in distinct patches of some size.*

Pathology.—It is accompanied by little constitutional disturbance; and if febrile phenomena are decided, it betokens more severe areolar inflammation than *erythema,* and a more grave disease. It is thus apt to be mistaken for *erysipelas.* The varieties of erythema are,—*E. læve; E. fugax* —Syn., *E. volaticum; E. marginatum; E. papulatum; E. tuberculatum; E. nodosum.* Of these varieties *erythema nodosum* is perhaps the most important. The indisposition which precedes the eruption is generally associated with a slight degree of fever. Red oval patches, considerably elevated and very tender, appear on the fore part of the legs, sometimes on the arms; and the long diameter of these patches is generally parallel to the axis of the limb. They may form bumps an inch and a half long and an inch broad on the anterior aspect of the leg. After a few days the red colour changes to a blue, the patches become soft, and although something like fluctuation may be felt, yet suppuration does not occur. Thus the bumps of erythema gradually subside. Sometimes the disease is seen in feeble boys; but it is most common in young women, in whom it seems associated with disordered menstruation, or with rheumatism (RAYER, WATSON). In the chronic stage desquamation invariably occurs; and to this stage of the disease the name *pityriasis* has been given.

Treatment. — *Rest, quinine,* and *iron,* after aperients; *carbonate of ammonia,* after gentle purgation, in doses of 5 to 10 grains three times a day, are the most useful remedies. *Oxide of zinc,* in fine powder, dusted over the surface, will sometimes allay the local irritation.

URTICARIA—*Syn.*, NETTLE-RASH.

LATIN EQ., *Urticaria;* FRENCH EQ., *Urticaire;* GERMAN EQ., *Nesselausschlag;* ITALIAN EQ., *Orticaria.*

Definition.—*An eruption of little solid elastic eminences, roundish or oblong, pale in the centre, and red at the circumference. These are commonly called "wheals," similar to the results from the strokes of a lash.*

Pathology.—The eruption is attended with an intense heat, a burning, tingling, or pricking sensation, very much like that produced by the stinging of a nettle. It is due to rapid serous infiltration of the lower layer of cells of the epidermis. There are several varieties of *urticaria*—(*a.*) *Urticaria acuta;* (*b.*) *Urticaria chronica.* The acute forms are generally connected with the ingestion of some kinds of food—oatmeal, bitter almonds, shell-fish, &c. The more chronic and intermittent forms are associated with uterine or other affections.

Treatment.—Emetics and purgatives in the first instance; afterwards the correction of faulty digestion. The surface of the eruption may be dusted over with flour; or the following lotion may be used:—

℞. *Carbonatis Ammoniæ,* ʒi.; *Plumb. Acetatis,* ʒii.; *Aquæ Rosarum,* ℥viii.;

In the chronic form, especially associated with uterine irritation or ovarian tumors, I have found *bromide of potassium* of much benefit. It ought to be given in a full dose of ten grains twice daily, which is to be doubled at bedtime. *Quinine* is also useful, combined with *rhubarb* and *carbonate of ammonia.*

℞. *Sulphatis quiniæ,* gr. xii.; *Solve in Glycerinæ,* ♏ii.; *Pulv. Rhei.,* gr. xxiv.; *Carb. Ammon.,* gr. xviii.; *misce et divide in pil.* xii.—*Signa, "One three times a day."*

LICHEN.

Latin Eq., *Lichen;* French Eq., *Lichen;* German Eq., *Lichen;* Italian Eq., *Lichene.*

Definition.—*An eruption commencing as small red papulæ, either isolated or confluent. These becoming excoriated, give vent to a serous fluid in considerable abundance, which ultimately concretes into a crust.*

Pathology.—This disease has been regarded as a form of *eczema.* It includes five varieties,—(1.) *Lichen simplex;* (2.) *Lichen pilaris;* (3.) *Lichen circumscriptus;* (4.) *Lichen agrius;* (5.) *Lichen tropicus*—Syn., *Prickly heat.* If the summits of the papulæ become torn by the nails in scratching, yellow or blackish crusts form, due to the mixture of blood and serum, and abrasions of the skin occur. The disease is then known as *prurigo* (*eczema lichenoides* or *eczema pruriginosum*). It occurs more or less in all chronic cases of *scabies* or *phthiriasis,* and sometimes in *urticaria.* The disease attacks by preference the scrofulous and the debilitated; and is associated with improper, insufficient, or bad food. A too liberal diet, and stimulants in food or drink, similarly predispose to the disease. Exposure of the skin to the heat of the sun, or to acrid substances, also brings about the disease—*e.g.,* in cooks, grocers, bakers, and the like. Fissures of the skin are a frequent complication, and occur where the skin is naturally thrown into folds, as at the arms, the angles of the mouth, the joints, the palms of the hands, and flexures of the fingers. They are apt to increase in depth as the disease continues, sometimes bleeding, and causing excruciating pain. When deep, the fissures are red and raw-looking, and serum or blood exudes from them, giving rise to crusts which partially fill them up (*eczema rimosum* of T. M. Anderson and Buchanan —the *eczema fendillé* of the French).

Treatment.—*Glycerine* in the following formula is recommended by Mr. Startin:—

℞. *Acid. Nit. dil.,* ʒss. to ʒi.; *Bismuth sub. nit.,* ʒss.; *Tinct. digital.,* ʒi.; *Glycerini,* ℥i.; *Aq. Rosæ, f* ℥vii. ss. "To be applied as a lotion frequently to the affected parts."

The alkalies internally and externally are of service. *Bicarbonate of soda,* in fifteen grain doses in some bitter infusion, to be increased by eight grains daily till ʒi. is taken in the twenty-four hours (Devergie). Alkaline baths and lotions are to be used at the same time—the lotions containing two to three drachms of the salt to twenty ounces of water.

PSORIASIS.

Latin Eq., *Psoriasis;* French Eq., *Psoriasis;* German Eq., *Psoriasis;* Italian Eq., *Psoriasi.*

Definition.—*An eruption characterised by the development of irregularly formed patches of a dusky red or coppery hue, slightly raised above the level of the skin, and covered with thin, dry, white scales.*

Pathology.—The affection comprehends *Psora leprosa,* and *Lepra vulgaris* or *dry tetter.* The term *lepra* is now quite given up on the Continent, being restricted to true leprosy. The patches may be distinct, small, and scattered; or larger, confounded together, and irregular; or they may be so extended as to make a continuous surface. Hence the names of varieties of psoriasis—(*a.*) *Psoriasis vulgaris;* (*b.*) *P. guttata;* (*c.*) *P. diffusa;* (*d.*) *P. gyrata;* (*e.*) *P. inveterata.* The intense itchiness and the eruption are always preceded and accompanied by that form of dyspepsia or impaired digestion in which there is a superabundance of acidity, much formation of lithates, and an obvious constitutional tendency to gout. Anæmic persons, and those in whom the circulation is languid, with a dry skin, are those in whom the disease is prevalent. Sometimes it is localised in patches, as on the back, between the shoulders, the lips, the eyelids, the palms of the hands, the scrotum, or the pudenda. Sometimes it seems hereditary. Intemperance, the use of highly salted food, fish, or indigestible substances, are apt to induce an attack. The depressing passions, anxiety, and grief may also be followed by *psoriasis.* The papillæ of the true skin are enlarged, from twelve to fifteen times their normal size; and the whole cuticle, including the Malpighian layer, is greatly hypertrophied —the corium being filled with abundant caudate cells arranged round the blood-vessels of the papillæ (NEWMANN).

Treatment.—Preparations of arsenic are found to be of great service in this squamous form of eczematous inflammation. A form of arsenical remedy, known as the Asiatic pill, is recommended by Cazenave. Its composition is as follows:

℞. *Arsenici Protoxidi,* gr. i.; *Pip. Nig.,* gr. xii.; *Palv. Acac.,* gr. ii.; *Aq. Distill.,* q. s.—*Divide in pil.* xii. *vel.* xvi. One pill to be taken once, twice, or thrice daily after meals.

It is of the greatest importance that the meals and the diet should be regulated. Small doses of *pilulæ hydrargyri,* or of *pilulæ calomelanos composita,* or *hydrargyra c. cretâ,* may be given at bedtime for a few days, followed each morning by a *drachm of magnesia,* given in combination with a teaspoonful of *lemon juice.* The vascular excitement of the stomach is best subdued by *dilute hydrocyanic acid,* in the following formula (A. T. THOMSON):—

℞. *Potassæ Carbonatis,* ʒss.; *Succi Limonis Recentis,* *f*ʒiv.; *Acid Hydrocyanici Diluti,* ♏iv.; *Vini Sem. Colchici,* ♏xv.; *Aquæ Distillatæ,* *f*ʒvi.; *misce. Fiat haustus* 4*ta. quâque horæ sumendus.*

The tone of the stomach is to be restored by small doses of *bicarbonate of potassa,* with from twelve to fifteen minims of the *tincture of henbane,* in a fluid ounce and a half of the *infusion of cinchona.* The eruption and itching disappear as the mucous membrane of the stomach and bowels return to a healthy state. The diet should be absolutely free from stimulants; and tepid baths, at a temperature of 96° Fahr., used every morning for half an hour, are most serviceable. The bowels should be regularly relieved by such mild ingredients as are contained in the following formula for pills:

℞. *Pilulæ Hydrargyri*, gr. vi.; *Pulveris Ipecacuanhæ*, gr. vi.; *Extracti Colocynthidis Comp.*, gr. xii.; *Ext. Hyoscyami*, gr. xviii.; *misce. Fiat pilulæ duodecem. Sum.* ij. h. s. *quotidie* (A. T. Thomson).

Glycerine and emollient lotions are useful local applications. I have found the itchiness greatly relieved by the following lotion of bitter almonds, rubbed night and morning over the parts affected :—

℞. *Amygdal. Amar. Numero*, xx. *vel.* xxx.; *Aquæ Rosæ*, ℥vii. *Contunde et tere simul, dein cola, et adde Hyd. Corrosiv. Sublim.*, gr. xii.; *Ammoniæ Hydrochloratis*, gr. xii.; *Spt. Vini Rect.*, ℥i.; or, *Dilute hydrocyanic acid* may be used to the extent of two drachms to the eight ounces, omitting the *corrosive sublimate;* or let *borax* replace the *hydrocyanic acid*, and omit the *spirits of wine.*

When the lotion has dried off, *oxide of zinc ointment* or *glycerine starch* ought to be applied to the parts all night.

MILIARIA.

Latin Eq., *Miliaria;* French Eq., *Miliaria;* German Eq., *Miliaria;* Italian Eq., *Miliare.*

Definition.—*An eruption of innumerable minute pimples, with white summits, occurring in successive crops upon the skin of the trunk and extremities, preceded and accompanied with fever, anxietas, oppression of respiration, copious sweats of a rank, sour, fetid odour, peculiar to the disease. The base of the pimples and the skin around are red and irritable.*

Pathology.—As to the specific nature of this disease pathologists are not agreed. All physicians are not disposed to admit that in *miliaria* a peculiar specific disease exists, with a characteristic eruption and definite course, such as the variolous pustules and course of small-pox exhibit. By many it is believed that "this affection is almost invariably symptomatic." Certain it is, however, that a peculiar epidemic disease prevailed in different parts of Europe at different periods in the world's history, the nature of which is described in the definition; and although in this country it seems to have disappeared, yet there can be no doubt that a specific disease of this description prevails epidemically from time to time in many parts of continental Europe and Asia. It has been described under the various names "sweating sickness," "miliary fever," "sudatoria," "miliaria," and the like. Rayer has given the most accurate account of it; and I had an opportunity of witnessing a great number of cases of it amongst the Turks in their military hospitals at Scutari, during the war against Russia in 1854-1856. The temperature and physical climate of that place, combined with the relaxed habits of the Turks, appear to be favourable to the development of such a disease. The best accounts of it are those of Borsieri and Rayer. The true *sudamina*, or *miliaria crystallina*, are always symptomatic of some continued fever, *acute rheumatism*, or other general disease. The fluid occurs in a small cavity within the layers of dry cuticle; and as a sudoriferous gland always opens into the vesicle, the fluid is justly regarded as retained sweat (VON BARENSPRUNG). A true miliary vesicle is thus analogous as to its anatomy with the formation of a *comedo* from the obstruction of a sebaceous follicle.

Symptoms.—The fever which precedes the eruption is ushered in by chills, intense and general, shivering, anxietas, oppression of the chest, restlessness, a sense of great feebleness and imminent fainting, with pains in the head, loins, and limbs. In a few hours, nausea, flushing, and profuse sweating supervene, but without any diminution of the dyspnœa, the anxietas, or pectoral oppression, but rather with an aggravation, in the form of short, irregular, panting, and sighing breathing, as if proceeding from a sense of weight under the sternum, with a feeling of internal heat, wandering pains, and sometimes cramps of the hands and calves of the legs. The pulse is generally rapid, small, and feeble, in a few cases hard; often variable, irregular, or intermittent at every ninth, twelfth, or sixteenth beat. The tongue is foul, being coated with a white or yellow fur, indicative of a sluggish condition of the alimentary canal; and the bowels are constipated. The sweat is profuse, and emits a peculiar smell of a rank, sour, fetid odour. From the fifth or sixth day up to the twenty-first, an itching sensation is felt in the mammary and epigastric regions, and inner surface of the arms, and the skin of those parts is found to be diffusely red, rough, and irregular, with numerous elevations not larger than pin-heads. In a short time the summits of these become pearly-white, the cuticle being elevated by a slight opaque sero-albuminous fluid; crop after crop breaks out, and continues from three to seven days, followed by a corresponding desquamation of the cuticle. This eruption is generally confined to the neck, breast, mammary and epigastric regions, and the inner surface of the loins and legs. In severe cases, miliary vesicles appear at the junctions of the skin and mucous membranes, and there they are apt to become aphthous. A deranged state of the gastro-enteric mucous membrane, indicated by nausea and vomiting of bilious matter, acid eructations, flatulence, and diarrhœa, frequently complicate the disease. Two forms have been described—namely, a mild and malignant. The malignant is rendered so by the occurrence of violent inflammation in some of the internal organs, especially of the stomach, lungs, kidneys, or brain; and the danger of the disease is chiefly due to these complications. Such malignant forms have been known to prove fatal in two or three days, but more frequently in from seven to twenty-one.

The **Treatment** of the disease appears to consist in cooling drinks, purgatives, and antiphlogistics.

HERPES.

LATIN EQ., *Herpes;* FRENCH EQ., *Herpes;* GERMAN EQ., *Herpes;*
ITALIAN EQ., *Erpete.*

Definition.—*This lesion is expressed by red patches on the skin, of irregular form and variable size, upon each of which there arises a group, cluster, or crop of extremely minute vesicles, for the most part in a corymbiform arrangement.*

Pathology.—(*a.*) *Causation.*—This is one of the most interesting of cutaneous lesions; for while its associations, on the one hand, are with *neuroses*, and on the other with specific *exanthemata*, it seems really as much a disease *sui generis*, as any with which we are acquainted (HUTCHINSON). A special mystery envelops its origin. In one case it seems an instance of an inflammation of the skin produced directly by nervous influence. In another case it is merely a local lesion, to be regarded as a sign or symptom of disturbance, beginning at some part of a nerve-trunk, or possibly in the very nerve-centres themselves. The varieties of *herpes* mentioned by the College of Physicians are—(*a.*) *Herpes phlyctenodes;* (*b.*) *Herpes circinatus;* (*c.*) *Herpes iris;* (*d.*) *Herpes zoster*,—Syn., *shingles, zona, ignea, cingulum.*

Besides *Herpes zoster*, the other forms of *herpes* are *symptomatic*, as when it occurs on the *lips, nose, cheeks, prepuce.* It is often rudely symmetrical. It goes through stages exactly like *herpes zoster*; but it may occur over and over again in the same person, and rarely or never leaves scars beyond a grazed-like mark for some time, unless ulceration from irritant applications has been deep. It may be observed in any illness in which a rigor occurs—*e. g.*, after *catheterism;* in *erysipelas;* in *ague;* in *pneumonia;* in *fever;* any inflammation of a shut sac, such as of the *pleura, peritoneum, tunica vaginalis.* It is a common lesion on the lips, associated with a common catarrh. A very common and troublesome form of the disease affects the foreskin—*herpes preputialis.* Its effects are often mistaken for chancres, and by unprincipled persons may be treated as such. It has nothing to do with syphilis, and its history is a sufficient guide to diagnosis. The disease may be prolonged by ulceration of the vesicles, or by irritant applications, or when the scabs are prematurely chafed off.

Herpes zoster occurs at almost any age, and in either sex; and no special condition of general ill health can be stated as predisposing to an attack of shingles. It is not contagious, and does not occur twice in the same person. The eruption is rarely, if ever, symmetrical; but syphilitic rashes have sometimes been known to resemble *herpes*, and so give rise to the belief that it is symmetrical (*Syphilitic Shingles* of Mr. HUTCHINSON). It occurs with equal frequency on the two sides of the body. The parts affected usually correspond to the cutaneous distribution of a particular nerve; but Mr. Hutchinson has in one case seen the *iris* implicated in a form of iritis. Referring to *herpes* as symptomatic of irritation of the nerves, Dr. W. Moore, of Dublin, regards intra-thoracic tumors producing such nerve-irritation as a probable cause of *herpes;* and hence that the occurrence of *herpes zoster* might suggest the existence of such tumors. He relates, in illustration, two cases of *herpes* eruption which occurred in patients suffering from thoracic aneurism (*Dublin Quarterly Journal of Med. Science*, Feb., 1868).

(*b.*) *Morbid Anatomy.*—The normal papillæ of the skin are enlarged by serous infiltration, and by multiplication of minute round exudation-corpuscles. The connective-tissue cells appear swollen, and the fusiform cells of the *rete Malpighii* increase in number, and spread through the capillary layer of cutis and the mucous layer of epidermis. These cells increase in number, forming a network throughout the papules, in the meshes of which exudation-corpuscles appear, and rapidly multiply, so as to push aside the normal epithelial cells. The papillary vessels beneath are meanwhile becoming dilated, and the contents of the network gradually assume the character of pus. The neurilemma of the affected nerves are shown to be inflamed and filled with small round nucleated cells (DANIELSSEN, VON BARENSPRUNG, NEUMANN, BIESIADECKI). A partial division of the vesicle and pustule of *herpes* into *loculi*, formed by fusiform nucleated cells, has also been described and figured by Dr. Haight. The serum is inclosed in this network between the horny layer of cuticle and the rete mucosum. In the papillary layer of the corium are round granular cells, like white blood-corpuscles in size; and as the vesicles change to pustules, these increase in number and fill the network above (BIESIADECKI and NEUMANN, in *Brit. and For. Med.-Chir. Review*, July, 1870, p. 32).

Symptoms.—The phenomena of *herpes zoster*, or *shingles*, are as follow:—A certain belt of skin on one side is felt to be tender and painful, but when looked at nothing is to be seen, the skin is not even red. Next day, however, or sooner or later, red points may be seen arranged in long oval groups on the painful parts, and very quickly each point shows a small clear vesicle. The vesicles at first are beautifully pellucid, and often a number are heaped together, not positively confluent, for divisions between them may still be seen, but much in the manner that a number of hills constituting one range are piled together. At a later stage the vesicles may contain a blood-stained serum, and later still opaque pus. The groups of *herpes* are on one side of the body only. If the area of the terminal branches of the fifth nerve are affected, then the eruption appears on the one side of the forehead, or side of the temple, eyelid, side of the nose, or upper lip; if one of the intercostals, then it appears on one side, before or behind, of the chest or abdomen; and so of the two extremities (JONATHAN HUTCHINSON). Commencing from behind at the spinous processes, the groups of vesicles in *herpes zoster* arrange themselves in a curved line, passing downwards and forwards on the trunk, and sometimes approaching the middle line in front. The affected parts continue very painful during the eruption, and for a considerable time afterwards. After the eruption has lasted a few days it begins to fade; and in a week or ten days it will have wholly disappeared, leaving however, perhaps, some troublesome ulcers. The eruption disappears at a stated time as certainly as the eruption of measles does, and leaves the patient free of liability to another attack as a rule, to which, however, there are rare exceptions. The eruption never relapses, as skin eruptions usually do. After the inflammation subsides the skin remains tender, and then numb; and the numerous little scars left show that the deep tissues of the true skin were involved (HUTCHINSON). The constitutional disturbance is rarely more than the pain will account for; but the eruption is often preceded by considerable local pain, or by cutaneous pain over the region which is about to be the seat of eruption, or by pain resembling neuralgia in the neighbouring parts, which may continue after the eruption has disappeared. It runs a very definite course, and when not interfered with, its development and defervescence are completed in about ten days. Now and then the patient has a slight rigor before the attack.

Treatment.—Dry applications, such as enveloping the parts in cotton wool after sprinkling with *oxide of zinc* in fine powder, or with starch, I have always found better than wet applications. *Tincture of aconite* and *tincture of opium* have been applied to relieve the severe intercostal pain. The intolerable burning has been relieved by painting over the vesicated patches with *collodion.* It tends to prevent the rupture of the vesicles, and so to prevent ulceration by promoting healing of the parts under a scab when inflammation has subsided. An opiate application, such as Dr. Fuller recommends in *sciatica,* may be of use in some cases of shingles, namely:—

℞. *Tinct. opii, sp. Æther. Sulph. co.; Glycerine,* ā ā ʒiii.; *Ext. Belladonna,* gr. xx.; *misce.* A strip of flannel soaked in this should be applied along the course of the deep-seated pain, and covered with oil-silk, to prevent evaporation (Waring).

The state of the stomach and digestive organs, and diet generally, should be regulated. Malt liquors should be avoided; and, if pain is severe, opiate fomentations may be applied. *Herpes preputialis* requires no treatment beyond careful ablution with tepid water, and the interposition of a piece of wet lint between the prepuce and the *glans penis.* If there is much pain, the lint should be moistened with a watery solution of opium.

PEMPHIGUS—*Syn.*, POMPHOLOX.

Latin Eq., *Pompholox;* French Eq., *Pemphigus;* German Eq., *Pemphigus* —Syn., *Pompholox;* Italian Eq., *Penfigo*—Syn., *Ponfolice.*

Definition.—*An eruption of large vesicles, filled with serous fluid, known as bullæ.*

Pathology.—These *bullæ* vary in magnitude, and are generally distinct and numerous. They spring up in successive crops, generally in the forearms, legs, and feet, surrounded by redness of the skin. The vesicles, originally transparent, gradually become opaque, pearl-coloured, and ultimately of a pale red colour. The *bullæ* of pemphigus differ from the *vesicles* of variola and herpes. The fluid of the *bullæ* is not situated between the mucus and the horny stratum of the epidermis, but between the layers of the latter, for the floor as well as the roof of the bulla-cavity is formed of flat cells, which show no nuclei, and do not absorb carmine. The cavity is simply filled with serum, and shows no locular network of spindle-shaped cells (Neumann, *Med.-Chir. Review,* July, 1870). Usually the disease is prolonged over weeks, months, or years, and is not attended by any decided febrile disturbances. Relapses are to be expected in children. A slight pricking sensation indicates a red spot, where the eruption commences. In the centre of each of these spots the cuticle begins to rise like a blister; and as the circumference of the redness increases, the blister-like vesiculæ are so rapidly formed that in a few hours each vesicle may be as big as a hazel-nut or walnut. The *blebs* are then apt to burst, when a straw-coloured serum exudes; and the epidermis contracts into folds or wrinkles, the surface underneath being red, painful, and smarting. If the vesicles do not burst, small brownish flat crusts form. The disease is usually associated with debility, intemperance, and bad or insufficient food. In one case of chronic pemphigus, quoted by Neumann, Dr. Hertz found extensive lardaceous disease of the viscera. The following varieties are to be distinguished—(*a.*) *Pemphigus acutus;* (*b.*) *Pemphigus chronicus;* (*c.*) *Pemphigus solitarius;* and, (*d.*) *Pemphigus foliaceous.*

Pemphigus foliaceous commences on the front of the chest, and when fully developed covers the whole body. It is almost always fatal.

Treatment.—Dietetic and tonic, especially by *quinine* and the *mineral acids. Arsenic* is said to be of use in obstinate cases, improving the general health. Emollient lotions, with or without opium, are useful local applications; also enveloping the parts with india-rubber cloth dressed with sulphur.

ECZEMA.

LATIN EQ., *Eczema;* FRENCH EQ., *Eczema;* GERMAN EQ., *Eczem*—Syn., *Nässende Flechte;* ITALIAN EQ., *Eczema.*

Definition.—*An eruption characterised by*—(1.) *Infiltration of the skin;* (2.) *exudation on its surface;* (3.) *the formation of crusts;* (4.) *itching.*

Pathology.—(*a.*) *Morbid Anatomy.*—The condition of the skin in different stages of *eczema,* as shown by fine sections made perpendicular to the surface, has been investigated by Biesiadecki, and is quoted by Neumann. In the early stages it resembles the description already given under *herpes.* The papillæ are enlarged and filled with serum and exudation cells, while the normal connective-tissue corpuscles of the cutis are swollen, and the same fusiform cells are seen in the *rete Malpighii.* When the formation of cells in the papillæ is rapid, and fluid is rapidly infused into the network of fusiform corpuscles and epidermic cells which form the eczematous papule, a *vesicle* is the result. The fusiform cells take on the same office as ordinary connective-tissue corpuscles in conveying the liquid exudation from the inflamed papilla beneath to the epidermis, where it collects under the horny layer of cuticle. When the cuticle falls of, the same channels convey the continually secreted eczematous fluid from the papillary layer of the cutis to the *rete Malpighii,* now laid bare, and so keep up the characteristic moisture of eczematous lesions. As the disease becomes chronic, the increase in size of the papillæ becomes more marked than ever, and their solid fibrous structure is hypertrophied, until at last they become visible to the naked eye. The whole cutis is thickened; and in long standing cases bands of dense fibrous tissue stretch down among the fat cells of the subcutaneous fascia. The cutaneous glands share in the changes of the surrounding structures. The fusiform cells of the epidermis, discovered by Biesiadecki, are always increased in number whenever the formation of cuticle becomes excessive—*i.e.,* in most chronic as well as in acute affections of the skin (PAGENSTECHER). They pass by autogenic movement from the papillary layer to the *rete Malpighii,* and they may often be seen half in the *corium* and half in the *Malpighian stratum.* They are known as "*migratory*" or "*wandering cells*" (wanderzellen). With regard to the exudation-cells first found so remarkably arranged round the vessels of the papilla, it is suggested by the able reviewer of Neumann's work in the *British and Foreign Review*, that they may probably be corpuscles which have just made their transit as the offspring of connective-tissue cells, or the result of spontaneous molecular aggregation. Like the serous fluid in which they float, they have passed into the *rete Malpighii* from the subjacent vessels of the cutis, instead of being generated where we find them by the epithelial cells themselves, or the fusiform corpuscles of Biesiadecki. This explanation is derived from the remarkable observations of Stricker and Cohnheim, already referred to in Vol. I., under *Inflammation*, on the passage of white corpuscles through the walls of the blood-vessels in a state of irritation. Practically, a papular, a vesicular, a pustular, a weeping, and a squamous condition of eczematous lesions may be recognised, as Hebra, Willan, and Neumann describe. Every case of eczema will, at some period or other of its progress, develop *vesicles;* but the infiltration of the skin, the character of the secretion, the thickening of the texture of the skin, the formation of crusts, the itching, and the seats of election, where the eruption is found, are sufficiently diagnostic and characteristic evidences of this very common disease. The forms of *eczema* recognised by the College of Physicians are:—(*a.*) *Eczema simplex;* (*b.*) *E. rubrum;* (*c.*) *E. impetiginodes;* (*d.*) *E. chronicum.*

Symptoms.—The elementary lesions of the skin in eczema are—(1.) An erythematous state of the skin; (2.) vesicles; (3.) pustules; (4.) papules; (5.) fissures; (6.) a mixture of several or of all of these lesions; and in the fully expressed disease there may not be the merest vestige of either a vesicle, a pustule, or a papule; but the skin is red and smooth on the surface, having a brilliantly polished and shining appearance, the tissue being loaded with infiltration. The vesicles, when they occur, are usually developed at the orifices of the cutaneous follicles. They are small and closely set together. They usually rupture early, and the serosity concretes into crusts. The more the skin is infiltrated the less likely are vesicles to be seen. In the absence of vesicles, when the disease is at its height, the infiltrated patches are red and inflamed. The redness is not uniform: it is studded with innumerable points of a deeper red, giving a punctated appearance to the part (DEVERGIE). These points correspond to the orifices of the glands, like the vesicles which may have preceded them; and their distinct appearance is due to the greater congestion of the skin which surrounds the glandular orifices. Minute excoriations are apt to occur at these spots, the result of rupture of the vesicles. From these excoriated points serous fluid continues to exude, which afterwards concretes into scabs (*eczema vesiculorum*). Eczematous skin feels thick when pinched up into a fold. It has a doughy feel, and on pressure the redness disappears, and shows a yellowish hue. The exudation is watery, sometimes purulent, and sometimes mixed with blood. It stains and stiffens underclothing with which it may come in contact. Becoming concrete, it forms crusts, and their appearances vary with the character and nature of the exudation. The itching is always aggravated by touching the part, and, at the same time, an irresistible desire to scratch is excited. Scratching is sometimes thus persevered in till blood flows. The disease is thereby greatly aggravated—a copious eruption of newly formed vesicles and fissures being apt to form.

The *impetigo* of authors is the pustular form of *eczema*, and the pustules form upon erythematous patches of skin, just as the vesicles did before them. They are most common on the head and chin. Vesicles and pustules are often seen on the same patch (*eczema impetiginodes* or *eczema pustulosum*).

Any part of the skin may be the seat of *eczema;* but there are certain localities where it is most apt to occur. These are the head, the hairy portions of the face, the edges of the eyelids, the nostrils, the lips, the external auditory passages and ears, the hands, feet, legs, genitals, perineum, *mammæ*, umbilicus, and parts of the skin which are naturally in contact with one another. On the head and hairy parts of the face the disease occurs most frequently in the pustular form (*impetigo capitis*); and collections of pus are apt to form beneath the crusts and scabs, attended sometimes by little cutaneous abscesses and enlargements of the lymphatic glands. Each pustule of eczema is situated at the orifice of a hair follicle; and the hair may be seen to pass through the centre of each pustule. These pustules dry up into small yellow crusts when the disease affects the hairy parts of the face; and the skin on which the pustules are developed assumes a dusky red tint, becoming gradually more thickened and infiltrated (*impetigo menti—eczema pilare facici*). In the *eczema* of the internal auditory passages the disease usually affects the auricles, commencing on the skin of these parts, and gradually extending inwards. The calibre of the meatus is narrowed, often so much so that it is impossible to see the *membrani tympani.* A milky or watery fluid exudes from the meatus, and moistens the pillow at night. The discharge is apt to have a very bad odour if the ear is not frequently washed out (ANDERSON, *Med. Times and Gazette*, June, 1863).

Diagnosis.—The *pruritus* or itchiness of simple cutaneous irritation, without any papular or other eruption, ought to be separated from true *prurigo*, and from the *pruritus* of eczema.

Treatment of eczema must be constitutional in the first instance; and local applications suited to the nature of the part affected are to be carefully used. Derangements of the digestive organs must be especially rectified. If the tongue is loaded, the appetite bad, the liver torpid, as indicated by light clay-coloured evacuations and costive bowels, small doses of *grey powder* in combination with *quinine* are indicated. Dr. T. M. Anderson suggests the following:—

℞. *Sulphatis Quiniæ*, gr. xii.; *Pulv. Rhei.*, gr. xxxvi.; *Hydrarg. c. Cretâ*, gr. xxx.; *Sacchari Albi*, ʒi.; *misce, et divide in Pulv.* xii.

Two of these powders are to be taken daily by an adult; but at any age the dose must be so adjusted that the patient has at least one full natural evacuation daily. If the patient is robust, but with the functions of the liver and bowels impaired, occasional doses of *calomel*, alone or in combination with *scammony*, will stimulate the torpidity of the digestive organs; and at the same time the cutaneous inflammation will diminish. If the patient is a full feeder, and will not be persuaded to live more sparingly, *one drachm to two drachms of the sulphate of magnesia* may be given twice daily, with *a sixth to half a grain of tartar emetic* added to each dose. The effect of this remedy will be to diminish desire for food, and at the same time keep the bowels freely open. If the patient is scrofulous, or debilitated from insufficient food, or food not nutritive enough, more nourishing food must be given, combined with tonics containing iron and cod-liver oil. Children a few months old, reduced to "skin and bone," recover wonderfully under the influence of twenty drops of the *syrup of the iodide of iron* in a teaspoonful of *cod-liver oil* repeated three times daily, the dose of the oil being gradually increased to a table-spoonful (ANDERSON, l. c., June, 27, 1863). *Syrup of the phosphate of iron*, or of the *phosphates of iron, quinia, strychnia*, should be alternated with the *iodide of iron.* The treatment must be steadily maintained for at least six weeks or two months. In severe cases the oil may be rubbed into the skin, in addition to giving it internally. *Cod-liver oil* and *iron* preparations are almost equally serviceable to eczematous adults. When there is a difficulty of taking the oil in its pure state, the emulsion of it will often be found useful: when the appetite is very deficient, a pure tonic, such as *quinine* and *aromatic sulphuric acid,* may be of service, as in the following:—

℞. *Sulphatis Quiniæ*, gr. xvi.; *Acid Sulphurici Aromatici*, ʒiv.; *Syrupi Limonum*, ℥ss.; *Infus. Cascarillæ*, ad ℥viii.; *misce, et cola per chartam.* A table-spoonful to be taken three times a day half an hour before food.

If the eczematous patients are robust and plethoric, the local abstraction of blood by leeches or scarification is often beneficial; but in general the action of *calomel purgation* is sufficient to reduce the inflammatory action, combined with a regulated abstemious diet of mixed animal and vegetable food simply dressed. Dishes of pastry, pickles, spices, strong tea, and coffee, are particularly to be avoided; while the use of wine, spirits, and malt liquors must be entirely suspended. If the patient has been in the habit of using stimulants, the eczema will be more difficult to subdue than if he had been an abstainer from these fluids. It is sometimes necessary to prescribe milk diet for a time, all animal food being proscribed.

Of internal medicines, *arsenic*, *sulphur*, and the *alkalies* are especially useful (STARTIN, ANDERSON). Of Fowler's solution (the *liquor arsenicalis* of the Edinburgh Pharmacopœia) an adult may commence with *five* minims thrice daily; and at the end of a week the dose should be increased by *one drop* every second or third day, till the disease begins to yield, or the medicine to disagree (ANDERSON). "In order to secure the virtues of arsenic as an alterative, it will be necessary to push the medicine to the full development of the phenomena which first indicate its peculiar action on the system. Arsenic as a remedy is too often suspended, or altogether abandoned, at the very moment its curative powers are coming into play. The earliest manifestation of its physiological action is apt to be looked upon as its poisonous operation; and the patient declares that the medicine has disagreed with him. Forthwith the physician shares his fears; the prescription is changed, and another case is added to the many in which arsenic is said to have failed after a fair trial of its efficacy"

(BEGBIE, *Contributions to Practical Medicine*, p. 270). *Arsenical solutions* should be given immediately after food; and in persons whose digestive organs are weak, a tonic infusion, such as the infusion of *cascarilla* or *gentian,* is the best vehicle for its administration; and a few drops of *morphia* may sometimes be added, as in the following :—

℞. *Solut. Fowleri; Solut. Muriatis Morphiæ,* ā ā ʒii.; *Syrupi Limonum,* ℥ss.; *Tinct. Cocci Cacti,* ʒss.; *Infus. Cascarilla,* ad ℥xii.; *misce.* A tablespoonful of this mixture to be taken thrice daily immediately after food.

The *liquor sodæ arsenias,* in doses of five to ten minims, is said to cure eczema with less gastric disturbance and with less irritability of the conjunctiva than the *liquor arsenicalis.* As the disease yields, the dose may be diminished; but the use of the remedy should not be suspended till some time after the complete removal of the eruption. In the case of infants at the breast, arsenical solutions may be given to the mother; and *mercury may be combined with the arsenic, as in Donovan's solution, of which ten minims may be given thrice daily.* Mr. Hunt lays down certain conditions for the administration of arsenic; and amongst them he forbids its commencement while signs of active cutaneous inflammation are present.

The use of the *natural mineral waters* which contain *sulphur* is the best method of securing the full effects of the alterative action of sulphur in chronic cases of eczema where there is an absence of inflammatory action. These waters are especially, Harrowgate and Moffat in this country; Aix, Barèges, St. Sauveur, Cauterets, on the Continent. *Alkalies* are most beneficial when the patient has been in the habit of taking stimulants; and when there is a tendency to acidity of the stomach, and to the deposit of *lithates* in the urine, or to gout, or to rheumatism, *aqua potassæ may be given, largely diluted with water, in doses of twenty minims three times a day.* Dr. Anderson recommends *sesquicarbonate of ammonia* (now called the *carbonate of ammonia* in the British Pharmacopœia), in doses gradually increasing from ten to thirty grains. If gout prevails, *colchicum* ought to be given with the other remedies; and in rheumatism the *acetate* or *bicarbonate of potash* in half-drachm doses should be added to each dose. All these alkaline remedies should be largely diluted with water. *For the relief of pain and procuring sleep* in acute cases, *opiates* must be administered; and when they fail tincture of *cannibis sativa,* in doses of ♏xxv., has been found sometimes to procure sleep, or at all events comparative ease (CHRISTISON). With regard to *local treatment,* the first point is to remove all sources of irritation, and especially the crusts. A poultice composed of *crumbs of bread* and *hot almond oil* applied to the eruption at night will usually bring away the crusts in the morning. If they fail to be detached by this application, they must be again lubricated with fresh almond oil, and forcibly removed when they are thoroughly softened. If, after the crusts are removed, the surface is seen to be acutely inflamed, and if there is a *burning heat* in place of *itching,* local sedatives must be applied. For this purpose Dr. Anderson recommends a *cold potato-starch poultice,* a small quantity of powder containing *camphor* being sprinkled over the inflamed surface before the poultice is applied. The camphor powder may be composed of the following ingredients:—

℞. *Camphoræ*, ʒss.; *Alcoholis*, q. s.; *Pulv. Oxydi Zinci*; *Pulv. Amyli*, ā ā ʒiii. This powder must be kept in a stoppered bottle (ANDERSON).

Instead of poultices, a mixture of powdered *oxide of zinc* and *glycerine*, in the proportion of half an ounce of the one to two ounces of the other, will be found to be a soothing application; and to it a little *camphor* may also be added, as in the following:—

℞. *Camphoræ*, ʒi.; *Pulv. Oxydi Zinci*, ℥ii.; *Glycerinæ et Adipis Benzoati*, ā ā ʒiv.; *Cochinillini*, gr. i.; *Spt. Rosmar.*, ʒi.; *vel Ol. Rosar.*, ♏ii.; *misce.* This mixture must be stirred before using it, a thin layer being then rubbed over the inflamed part twice or thrice daily (ANDERSON).

Carron oil (*liniment aqua calcis*) I have found a soothing remedy. When the disease passes into a chronic stage, as indicated by the disappearance of the burning heat and the supervention of itching, the local applications must vary with the condition of the parts. If there be much *infiltration* of the tissues, treatment by *potash* applications is the most efficient (HEBRA, ANDERSON). The more extensive the disease the *weaker* the solution ought to be for application over a large surface. If the infiltration is slight, common *potash soap* (soft soap, black soap, *sapo mollis, sapo viridis*), or a solution of *one part of it in two of boiling water, a little oil of rosemary or citronella being used to conceal the odour*, may be used. A piece of flannel dipped in this liniment should be rubbed as firmly as possible over the affected parts night and morning, the solution being allowed to dry upon them. It should, however, be washed off after each application. If the patient can bear it, a piece of flannel, saturated with the solution, may be left in contact with the part all night. Another method of treatment is to paint over the eruption night and morning with a large brush, charged with the *aqua potassæ* of the Edinburgh Pharmacopœia, its irritant action being neutralised by means of cold water when the smarting becomes excessive. A solution of *potassa fusa* may also be employed of various strengths. If the case be mild, *two grains* to an *ounce* of water; if more severe, then *five, ten, twenty, thirty*, or *even more grains to the ounce of water* may be used. *But all these stronger solutions must be washed off very speedily, and they ought not* to be employed more than once daily. Such remedies the physician ought to apply himself, and not entrust the control of their action to the patient. As infiltration subsides, the solution ought to be gradually diluted; and great caution is necessary in using such local applications upon infants, delicate females, and old or infirm persons. The affected parts should be bathed repeatedly during the day in cold water, during the use of *potash* applications. The *cold douche* may be employed where it is practicable. If the itching is very intolerable, *prussic acid* may be mingled with the potash applications, as in the following:—

℞. *Potassæ Fusæ*, gr. x.; *Acid Hydrocyan. Dil.* (Ed. Ph.) ʒss.; *Aq. Rosarum*, ʒii.; *misce.* A little of this solution is to be rubbed firmly over the eruption night and morning, and at any time when the itching sensation is severe.

If there is a tendency to the formation of fissures, which are apt to result from the use of potash, *cod-liver oil, glycerine*, or *carron oil* should be applied to the parts every night. *Glycerine of aloes* is also recommended as a healing agent by M. Chausit and Dr. Waring. It is prepared by evaporating four to eight parts of the *tincture of aloes*, and incorporating the residuum with thirty parts of *glycerine*. *Tarry applications* are of great value in the local treatment of the *declining stages*, when infiltration and itching are subdued. Common tar (*pix liquida*) or oil of cade (*oleum cadini*, as manufactured at Aix-la-Chapelle) should be rubbed firmly over the eruption by means of a flannel cloth, and allowed to dry upon it. It should be applied thrice daily, and washed of with *soft soap* or *petroleum soap*. Dr. Anderson recommends that common tar should be combined with one of the potash solutions—for example, a mixture of *equal parts of common tar, methylated spirit, and soft soap*, used as above directed, will be found useful, or the following preparation, as less offensive:—

℞. *Saponis Mollis, Spt. Rectificati, Olei Cadini*, ā ā ʒi. ; *Olei Lavandulæ*, ℥iss.; *misce*. A little quantity is to be firmly rubbed over the eruption night and morning; and it must be washed off before each reapplication.

Of *mercurial applications* the *citrine ointment* (*Ung.-hyd. nit.*) is the best, or the *ointment of the iodide of mercury*. They may be used of their full strength, or diluted with lard, according to the indications of the case, care being taken that *mercurialism* is not induced. When eczema has reached the dry stage, Mr. Milton regards the *dilute ointment of the nitrate of mercury* as the most effectual remedy we possess. In the itching of *pruritus*—*pruritus scroti* and *prurigo pudendi muliebris*—especially the *prurigo senilis*, this dilute ointment is highly spoken of by Dr. Bowling, United States, and by Mr. Balmanno Squire in this country, as one of the best remedies for subduing the irritation and itchiness. Dr. Bowling advises that the parts be first sponged with vinegar or lemon-juice previous to the application of the ointment; and states that for fifteen years he has not failed to effect a permanent cure (*Med. Times*, June 6, 1868). In using ointments, a small quantity should be melted on the finger, and rubbed firmly into the affected part. None should be allowed to lie undissolved upon the skin; and the part should always be cleansed with soap and water before reapplication. Epilation should be resorted to in cases of eczema of the hairy parts of the face. In very mild attacks, or with a view to prevent the recurrence of the disease, the skin may be washed occasionally with soft soap and water. Hendrie's "Dispensary Petroleum Soap" is recommended by Dr. Anderson, to whose admirable lectures on "Eczema," in the *Medical Times and Gazette* for May, June, and July, 1863 (since published in a separate volume), the reader is referred for more detailed information.

ECTHYMA.

LATIN EQ., *Ecthyma;* FRENCH EQ., *Ecthyma;* GERMAN EQ., *Ecthyma;*
ITALIAN EQ., *Ectima.*

Definition.—*An eruption of large round pustules, generally distinct, and seated upon a hard inflamed base. The pustules are succeeded by dark-coloured* *scabs, which leave superficial cicatrices behind them, or red stains, which disappear after a time.*

Pathology.—The eruption of ecthyma is common on the neck, shoulders, buttocks, extremities, and chest; and is seldom developed on the face or scalp. The typical appearance of ecthyma may be seen in the pustules produced by friction with *tartar emetic* ointment. The pustules continue for several days, and are succeeded by scabs, which begin to form in the centre. The disease may occur at any age or season, but it most frequently appears during spring and summer in young adults. The *ecthyma cachecticum* of Willan prevails in old, irritable persons much addicted to alcoholic fluids. It is classed among *furuncular affections* by Mr. Erasmus Wilson; and the great majority of cases appear to be either of traumatic or syphilitic origin, or due to scabies.

Treatment is chiefly by diluents, simple and emollient baths, and regulation of diet. Moderate exercise should be taken, combined with the use of alkaline or salt-water baths. Mild laxatives are beneficial, and spirits, wine, and beer are to be refrained from. The food should be as nourishing as can be digested. Tonics, such as *quinine* and *iron*, are also indicated. A stimulating lotion, composed of *muriatic acid* diluted with water, is of use to brush over the parts and promote cicatrisation. Specific treatment must be adopted when the disease is associated with syphilis or scabies.

ACNE.

Latin Eq., *Acne;* French Eq., *Acne;* German Eq., *Acne*—Syn., *Finnenausschlag;* Italian Eq., *Acne.*

Definition.—*Inflammation of the sebaceous glands and hair follicles, with retention of the gland secretion.*

Pathology.—This lesion affects the sebaceous glands and hair follicles of the skin generally. The disease generally appears as pimples; but suppuration usually very slowly follows, and there may be persistent redness after suppuration is at an end, giving a blotchy appearance to the part. The parts most frequently affected are the temples, cheeks, nose, and forehead; but it also appears on the neck, shoulders, and front of the chest, where the sebaceous glands are the largest and most active. *Acne* is indicated by the collection of retained material in the sebaceous follicles, which open on the skin by a blackish point (*comedo*), to which the vulgar give the name of "*grubs*" or "*worms*" *in the skin.* The tubercles on which the classification of this disease is sometimes based are merely the terminations of the pustules, and not an elementary lesion. The accumulation of the matter secreted by the sebaceous follicles tends to keep up an eczematous inflammation in them. The lesion has been mistaken for small-pox.

The lesions of *acne* recognised by the College of Physicians are—(*a.*) *Acne punctata;* (*b.*) *Acne indurata;* (*c.*) *Acne rosacea;* (*d.*) *Acne strophulosa*—Syn., *Strophulus albidus.*

Acne rosacea should be separated from true acne, with which it has nothing in common, except occasional coincidence; as well as from the other forms, which it does not even resemble in appearance. It consists in hypertrophy of the cutis and subcutaneous tissues, with enlargement of their blood-vessels, as a consequence of chronic congestion of the skin of the nose and other parts. It is thus analogous to the *œdema durum* of continued venous congestion, the hypertrophy seen in a long prolapsed rectum or uterus; or to the effects of blood-stasis in chilblains (*Med.-Chir. Rev.*, July, 1870, p. 33). If a *demodex follicularum* should be discovered and caught in a follicle of *acne*, make a note of its presence as a probable source of irritation.

Dysidrosis is an inflammation of the sweat glands and follicles of the skin, but especially of the hands and feet, where sweating is excessive, mixed with more or less serum, which is effused into the sweat glands, and distends them. These glands appear as sago-grain-like bodies imbedded in the skin, along the sides of the fingers and palm of the hands, which are hot, painful, and stiff, and more or less swollen. The cuticle afterwards drys and peels off, leaving a dry, reddened, shining surface, which never discharges fluid, as in eczema. The late Dr. Tilbury Fox first described the disease as occurring chiefly in weak, cachectic or debilitated subjects.

Treatment.—Local and general measures require to be combined. Diet, in the first instance, should be restricted. Wine, spirits, and coffee are to be refrained from. Milk is to be used as a drink, and as an article of diet, combined with light food, fresh vegetables, and succulent ripe fruits. Emollient applications, such as an emulsion of bitter almonds, a decoction of bran or quince seeds, and tepid milk, are useful. Dr. Anderson has found *sulphur* (ʒi.), mixed with rectified spirit (℥i.), to be shaken before using it; or a mixture of *hyd. corrosiv. sublim.* (gr. ix.-xii.); *hydrochl. ammoniœ* (ʒss.); *cochinillini* (gr. i.); *aq. rosœ* (℥vi.), particularly serviceable. Generally, if one fails, the other succeeds.

In that form of *acne* common to young women at the commencement of their menstrual period, Dr. Ringer recommends *sulphur* (ʒi.), *glycerine* (*f* ʒi.), *water* (*f* ℥x.), to be applied twice or thrice daily.

Whichever of these lotions is used, let it be applied by dipping a piece of flannel into the lotion, and rubbing *very firmly* over the eruption night and morning. *Flowers of sulphur* used dry to dust the parts with at bed-time is also of service. To promote resolution of the induration, *iodide of sulphur*, in the proportion of *fifteen* or *twenty-four grains to an ounce of lard*, is of great service in dispelling the tumors. Drastic purgation ought to be avoided. Simple baths at a temperature of 88° or 90° Fahr. are of service. *Calomel ointment* (ʒi. of calomel to ℥ii. of lard) has been found useful, care being taken against salivation. If there be much thickening of skin, Mr. Milton recommends the internal use of *liquor potassœ*, although inferior to *arsenic*, which ought to be used. In *dysidrosis*, the treatment is mainly by carron oil to the inflamed parts, with diuretics and tonics internally.

ICHTHYOSIS.

Latin Eq., *Ichthyosis;* French Eq., *Ichthyosis;* German Eq., *Ichthyosis;* Italian Eq., *Ittiosi.*

Definition.—*A lesion which involves the whole tissue of the skin, and is characterised by the growth of thick, dry, imbricated scales of a dirty grey colour, resting upon a perfectly uninflamed surface, and never accompanied by pain, heat, or itching.*

Pathology.—(*a.*) *Morbid Anatomy.*—The microscopic anatomy of *ichthyosis* shows that its true congenital form does not depend on collections of dried up *sebum,* but on the excessive development of the horny layer of cuticle, combined with considerable thickening of the whole skin (Neumann and Rindfleisch). At the same time there is increased secretion from the sebaceous glands, which accounts for the greasy character of the scales, for their frequently dirty colour, and for the masses adhering so strongly to each other. An alcoholic extract of the scrapings of the skin deposited, on evaporation, crystals of *stearin* and of (apparently) *hippuric acid;* while earthy phosphates, oil globules, and cholesterine were left behind. A further treatment of the scales with *ether* showed a quantity of solid and liquid fat, as well as more cholesterine. After incineration there remained chlorides of potash and soda, with phosphates of lime and magnesia, and a certain amount of iron (Schlossberger). On removing and breaking through a crust of *ichthyosis,* vertical streaks, or even actual fibrillæ, may be observed on the fresh surface. When it has been macerated in a weak alkaline solution, and carefully torn to pieces with needles, it may sometimes be entirely divided into prismatic rods, which might be equally well described as short and thick fibres. Each of them consists of a number of horny laminæ, arranged concentrically round an axis like the rings of a tree (Rindfleisch, quoted in *Med.-Chir. Rev.*, July, 1870, p. 43).

Symptoms.—It appears principally on the external parts of the limbs, round the joints, on the knee and elbow, on the upper part of the back, and generally on those regions where the skin is thick and coarse. The disease is usually congenital, and lasts during life. It may not be strongly marked at the period of birth; but the skin appears dull, thick, and fretted, and the lesion is developed as the infant grows older.

Treatment.—Remedial measures are only palliative, consisting mainly of mucilaginous and glycerine lotions, with vapour baths to mollify the roughness. Potash solutions, or other preparations of potash, are contra-indicated as local applications.

CHELOID.

Latin Eq., *Tumor Cheloides;* French Eq., *Chéloïde;* German Eq., *Keloid;* Italian Eq., *Cheloide.*

Definition.—*A tuberculated growth or swelling of the true skin occurring spontaneously or upon a cicatrix.*

Pathology.—(*a.*) *History.*—This disease was originally described by Alibert, subsequently by the late Dr. Addison, of Guy's Hospital, and Mr. Thomas Longmore, Professor of Military Surgery at the Army Medical School. The cheloid of Alibert is a fibrous tumor of the skin, often developed on a cicatrix; whereas true cheloid, as described by Dr. Addison and Mr. Longmore, is a much more extensive lesion; but anatomically the lesions do not appear to differ, whether occurring spontaneously in the skin or limited to a cicatrix, although called genuine and spurious varieties—true and false cheloid.

(*b*.) *Morbid Anatomy.*—According to the recent investigations of Dr. Neumann, "the epidermis and papillary layer are, at least, primarily unaffected; the deep layer of the cutis is thickened by a multitude of fibres running parallel with the surface, which begin in bundles of spindle-shaped cells, first observed surrounding the external coat (*tunica adventitia*) of the arterioles of the skin. The cutaneous glands are at first unchanged, but afterwards become pressed upon so as gradually to atrophy and disappear. The same cellular growth is found in the adventitia of the arteries, spreading to the deeper layers of the corium, in the form of dense horizontal fibres running parallel to the long axis of the cheloid nodule. The epidermis and papillary layer are unaffected, as are the cutaneous glands, until they gradually disappear in the later stages of the disease (Dr. WARREN of Boston). There is thus no difference anatomically between true and false cheloid. Both consist of a 'fibro-plastic' tumor of the corium, and the 'malignity' or tendency to recur after removal, which is so marked a feature of the disease, simply depends on the fact that the primary affection of the blood-vessels can be traced some distance beyond the circumference of the tumor, and can therefore reproduce a fresh nodule after the original one has been apparently completely extirpated. The chief anatomical difference between cheloid and an ordinary cicatrix appears to be, that the fibres of connective-tissue of which the latter is composed form a felted network, like that of normal cutis, with no definite parallel arrangement. Virchow regards the most typical forms of cheloid, as sarcomata (a group which, it will be remembered, corresponds in great part with Lebert's fibro-plastic tumors), and distinguishes from these the fibrous growths described by Addison as true cheloid, and others which he believes to be of cancerous or syphilitic origin. Rindfleisch agrees in regarding cheloid as a sarcoma, but places its seat in the papillary layer of the cutis—a supposition disproved by microscopic investigations, which were wanting when he formed this opinion" (*Med.-Chir. Rev.*, July, 1870).

Symptoms.—It begins as a few prominent red tubercles, generally over the sternum, whence it may extend gradually to the sides of the body and the back. The red tubercles gradually increase in size, and coalesce into larger growths. These growths then send forth spur-like processes on every side; a slight puckering of the healthy skin surrounds the marginal limits of the bases of the tumors, and marks the advance of the disease. The spread of the disease is very slow and gradual, extending over months and even years. Intense itching and irritation accompany its advance, more particularly when warm in bed at night, or during hot weather. Continued cold causes the growths partially to disappear, leaving an old cicatrix-like condition of the skin (*Med.-Chir. Trans.*, 1863).

Treatment is not yet defined; but the history of the disease points to cold applications and tonic remedies as the basis of a rational treatment.

SECTION III.—PARASITIC DISEASES OF THE SKIN.

TINEA TONSURANS—*Syn.*, RINGWORM.

LATIN EQ., *Tinea Tondens;* FRENCH EQ., *Tinea Tonsurans;* GERMAN EQ., *Tinea Tonsurans*—Syn., *Ringwurm;* ITALIAN EQ., *Teigne Tonsurante.*

Definition.—*An affection implicating the hairs of the skin, scalp, or chin, and usually assuming a circular form. The hair becomes dry and brittle, having a tendency to crack or break across. Itching accompanies the primary eruption, which is generally at first erythematous, with slight swelling, and a parasitic fungus ultimately appears* (Achorion Lebertii—Trichophyton tonsurans), *which had been developing between the epidermis and the true skin. The fungus has a pure white colour and powdery aspect. It covers the epidermis between the hairs, and forms around them a complete white sheath. Inflammation of the hair follicles and of the surrounding tissues occurs; and when pus forms, the fungus is destroyed at the expense of obliteration of the roots of the hair, when perfect baldness ensues* (BAZIN, ANDERSON).

Pathology.—The nature of this disease is to be studied in the botany of the cryptogamic parasite called the *Achorion Lebertii* (*Trichophyton*), discovered by Malmsten in 1845. It consists of oval transparent spores or globules, about the $\frac{1}{7000}$ part of an inch in diameter. Many of these are isolated; others constitute, by their juxtaposition, articulated filaments. Comparatively few cryptogamic tubes are visible—a character which distinguishes the ringworm affections from the vegetable structures seen in the other parasitic diseases of the skin (Fig. 75). Its anatomical seat is in the *interior of the roots of the hair.* The *hairs* and *fungi* simultaneously increase; the former seem larger than usual, are paler in colour, lose their elasticity, soften, and break off when they have risen some one or two lines above the surface of the scalp. In the short cylinder of hair left, the fungus grows still more rapidly, so that the normal structure of the small stump soon becomes indistinguishable. Sometimes the hair breaks off before emerging from the skin, and the fungus, epidermis, and sebaceous matter fill the ends of the piliferous conduits, and form the little prominences which can be seen by the naked eye in this disease, and which gives to the skin a rough anserine appearance. The sporules and mycelium of the plants can sometimes be seen in the form of a white

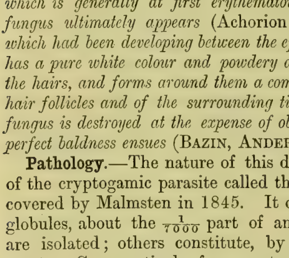

Fig 75.*

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* Parasitic fungus from a case of *Tinea tonsurans.*—(A, A.) Isolated spores; (B.) spores united at their ends; (C, C, C.) empty tubes; (D.) sporular tube (after BAZIN).

powder on the roots of the broken hairs; sometimes the cutis becomes congested and thickened, and then the plant becomes mixed up with the scales of epidermis, with fatty and albuminoid granules, with pus and serous exudation, and so crusts are formed of greater or less thickness, in which the growth of the fungus goes on. It exists in the *Herpes tonsurans* of Cazenave, which is the *Porrigo scutulata* of Willan, the *Tinea tonsurans* of Bazin, and the *Trichosis furfuracea* of Erasmus Wilson and Dr. Wood. The disease commonly called *Ringworm* is what is intended to be described by all those names now mentioned, on which dermatologists are not agreed.

There are three varieties of this disease, described under the following names:—(1.) *Ringworm of the Body* (*Tinea circinatus*), commences by a little rose-coloured and slightly elevated spot, about the size of a fourpenny piece, which shortly becomes the seat of slight furfuraceous desquamation, accompanied by tingling or itching. The spot gradually increases in size, but retains its circular form; and as it extends, the healing process commences in the centre; so that in a short time the red spot is transformed into a large, prominent, erythematous ring, inclosing a portion of sound skin. This process goes on for an indefinite period, the ring gradually increasing till it may have a diameter of four or five inches. When the extent of surface affected is large, the circle is apt to become incomplete, so that various segments of circles appear. The disease may terminate spontaneously, the parasitic fungus being very superficial, the hairs small and rudimentary, so that the parasite dies for want of nourishment. It is apt to affect the face, the neck, the back, and outside of the wrist and hand (Bazin, Anderson). (2.) *Ringworm of the Beard* (*Tinea sycosis*) is met with on the upper lip and hairy parts of the cheeks, as well as on the chin, when it affects men; but the hairs of the axillæ or genital organs of females are not exempt from this disease. It commences exactly like *T. circinatus*; but it is not till the deeper structures are involved, and when small indurations occur, surmounted by pustules resembling *acne*, and when the hairs can be pulled out with ease, that the attention of the patient is attracted to the affection.

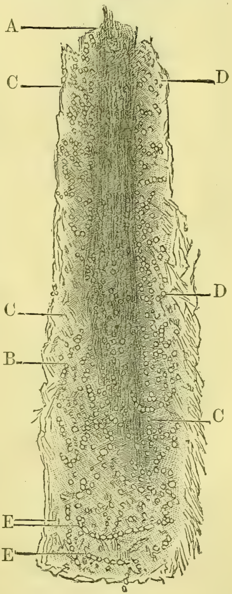

Fig. 76. *

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* Hair from a case of *Sycosis*—(A.) Broken condition of the superior extremity of the hair; (B.) rupture of the inferior extremity of the hair; (C, C, C.) epidermic tunic of the hair; (D, D.) isolated spores; (E, E.) chains of spores (after Bazin).

The hairs are thickened, the bulbs flattened and more or less disorganised. The longitudinal fibres of the hair are separated by masses of sporules imbedded between them; and where the fungus accumulates, nodes on the hair indicate the site of such accumulation. The medullary part of the hair is quite disorganised (Figs. 76 and 77), and may disappear altogether. In the advanced stage of *sycosis*, when inflammatory and suppurative phenomena prevail, the fungus is difficult to find. (3.) *Ringworm of the scalp* (*Tinea tonsurans*) is for the most part met with in children. It generally makes its appearance first in the form of rounded patches on different parts of the head, of a scaly or pityriasis-like inflammation. From the irritation induced small vesicles may form. The hairs in the first instance are dull, dry, and twisted, and easily extracted; but as the disease advances they become very brittle, and break on attempting to extract them; and as they become more and more friable they break of themselves within a line or two of the skin (Bazin, Anderson). The twisting of the hairs, so frequently observed, is due to—(*a.*) Plugging up of the follicular orifice by secretion, and detention of the upper part of the shaft of the hair, while its growth at the papilla still continues. Half an inch in length of hair may sometimes be pulled from beneath the false operculum. (*b.*) It may be due to the presence of *mycelium* in the follicle, clinging to the hair on one side and to the follicle on the other. It thus blocks up the follicle, and holds the hair (while still growing) to the diseased spot (Fox). The epidermis and stumps of the hair become covered over with a characteristic greyish-white powder, which ensheathes the hair. This powder consists of the *sporules* of the fungus. There is slight elevation and puffiness of the skin of the diseased parts, while its colour is bluish or slate-coloured in dark subjects and greyish-red or yellow in fair persons (Bazin, Anderson). This elevation and puffiness of the skin is due to a granular layer or *stroma*, which, on the addition of *liquor potassæ*, is seen under the microscope to be due to *sporules* of the fungus closely packed together. The

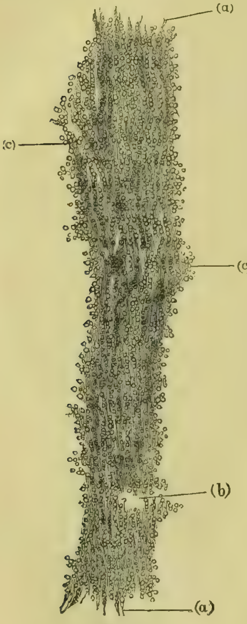

Fig. 77. *

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* Hair from a case of *Tinea tonsurans* loaded with *sporules*—(*a, a.*) Broken ends of the hair; (*b.*) ruptures of the longitudinal fibres; (*c, c.*) ragged edges of ruptured hair (Dr. T. M. Anderson).

amount of fluid influences materially the size of the *sporules* (Robin, Fox). When pustules and yellow crusts form, the detection of the fungus is more difficult. The inflammation will last as long as the growth of *fungi* continues, but sometimes they die out spontaneously, and a perfect recovery takes place; but the hair follicles may be obliterated, when permanent *alopecia* of the affected parts is the result, with more or less atrophy of that portion of the scalp (Bazin, Anderson).

The **Treatment** of ringworm has been long one of the most difficult points in dermatology. Its principles, however, are now well understood, and few cases resist the proper measures. The essential point is to apply to *the roots of the hairs* a preparation which may destroy the fungus: if this can be done, the disease is cured. It is first of all necessary to remove the hair. This is in part generally accomplished before the case comes under treatment, by the course of the disease; if it has not been sufficiently done, "epilation" can be accomplished by a chemical agent, or by extraction with pincers (Fig. 78). The forceps most suitable for this little operation are those about three inches long, having a weak spring, so that the hand may not be fatigued in using them. They should be made so that the two extremities come together very exactly, and do not slide the one upon the other. Each extremity should be a couple of lines broad, so that a fasciculus of hair may be caught up at one time when required; and should be furnished on the inside with very fine, but at the same time blunt, transverse denticulations, so that they may not cut across the brittle hairs. M. Bazin recommends an ointment composed of *lime* and *carbonate of soda*, of each one part, and thirty parts of *lard*, as an agent to remove the hair. The *oil of cade*, however, appears to be the best depilatory known, and with this mode of treatment epilation with the pincers may be combined. If the hairs are pulled out in the proper direction, there is very little pain, especially after the sensibility of the skin has been blunted by the use of the *oil of cade.* The removal of the hairs permits a "parasiticide" solution to be applied to the hair follicles, within which are the prolific spores of the fungus. For this purpose M. Bazin recommends either a solution of *bichloride of mercury* (1 part to 250 of water), or an ointment of the *acetate of copper* (1 part to 500 of lard), about *two* grains to an ounce of water; and a little *alcohol* or *muriate of ammonia* should be used to facilitate the solution of the mineral. The *oil of cade* should be mixed with *glycerine* in the proportion of half a drachm to a drachm of the oil to an ounce of *glycerine.* Kuchenmeister's experience shows that the alcoholic solutions act most powerfully. Dr. Parkes used, with excellent effect, a solution of the *pernitrate of mercury*, about one part to thirty or forty of water. This is, however, a very powerful remedy, and is to be cautiously

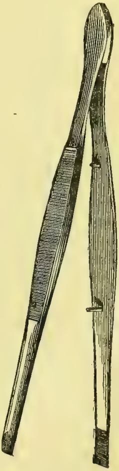
Fig. 78.*

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* Forceps for epilation (Dr. Anderson).

used, as it easily blisters the scalp; also, an ointment composed of *sulphate of copper* (one part), *alum* (three parts), and *lard* (twenty to thirty parts, according to the age of the patient). Probably a better and safer parasiticide than any of these is *sulphurous acid* (Sir WILLIAM JENNER). Diluted with equal parts of *glycerine*, or with two or three parts of water, it is to be applied on a piece of lint to the affected part, and covered with oil-silk to prevent evaporation (*Med. Times*, Aug. 20, 1853). The following formula for sulphur may also be used:—

℞. *Sulphur*, ℥iv. ; *Hydrarg. Ammon.-chloridi, Hydrarg. Sulphuret*, ā ā ʒii. ; *Ol. Olivæ*, ℥i. ; *Adipis*, ℥iv. ; *Creasoti*, ♏v. It must be well rubbed into the patches of tinea.

Or, a lotion of one ounce of *hyposulphite of soda* in twelve ounces of water (Dr. TILBURY FOX). The internal use of *cod-liver oil* at the same time that *sulphurous* applications are used, has also been recommended (J. H. BENNETT). My friend, Staff-Surgeon Dr. Davidson, has found the following method of treatment to succeed:—namely, to apply *tincture of iodine* to the affected parts twice a day for fourteen days, and afterwards ointment of the *bichloride* of mercury (*Coros. sublimat.*) After the third or fourth application of the iodine, the disease ceases to spread, and the hair (which may have been thinning rapidly) ceases to fall off. A kind of crust is formed by the application of the iodine, which scales off in the form of a scurf when the ointment is applied. *Soft soap* (*black soap*) applied to the patches of disease at bedtime, and washed off in the morning, fits the parts for the better reception of other local remedies. Washing the head before medical treatment is begun, sometimes tends to spread the disease to parts of the head which had been sound before. *Goa powder*, called also *Bahia powder* and *Araroba powder*, first introduced to European medical practice by Sir Joseph Fayrer, has also become a remedy, made into an ointment composed of 20 grains of the powder, 10 drops of *acetic acid* or *common vinegar*, and an ounce of *benzoin ointment*. It is applied by means of a hair pencil (Dr. DA SILVA LIMA). Its active principle, *chrysophanic acid*, has the objectionable property of staining; but it forms an efficient ointment in the treatment of some forms of ringworm, 10 to 60 grains to an ounce of lard being melted together in an oil-bath.

The *iodide of arsenic* in doses of one-tenth of a grain, gradually increased to one-fourth of a grain for an adult; one-fifteenth of a grain for a child six years old; and from one-eighteenth to one-twentieth for younger children, improves the general health (NELIGAN, *On Diseases of the Scalp*).

TINEA DECALVANS—*Syn.*, ALOPECIA AREATA, PORRIGO DECALVANS.

LATIN EQ., *Tinea Decalvans;* FRENCH EQ., *Teigne Pelade;* GERMAN EQ., *Tinea Decalvans*—Syn., *Alopecia Areata;* ITALIAN EQ., *Tigna Decalvante.*

Definition.—*A fungous disease causing the formation of rounded or oval* *patches of baldness, sometimes solitary, more generally multiple. It affects the hairy scalp principally; but the beard, the genital organs, and hairy portions of the skin may also suffer.*

Pathology.—The fungus to which such circumscribed patches of baldness are owing, has been named the *Microsporon Audouini*, detected by Gruby in 1843. It is present in the disease commonly called, after Willan, *Porrigo decalvans*, or *Alopecia circumscripta;* or, after Bateman, *Tinea decalvans;* by Bazin, *Tinea achromatosa;* by Anderson, *Alopecia.* It differs from the *Trichophyton* of *Tinea tondens* by its numerous waved filaments, and by the extremely small size of its sporules. It is not found, like the *Trichophyton,* in the interior of the root, but forms round each hair a little tube; the hair then becomes opaque, softens, and breaks off (Fig. 79). The *alopecia* is rapid, with or without previous *vitiligo* of the skin; the dermis is not congested, and the epidermis is thin and smooth. In the early stage of the disease the hairs appear dull and lustreless, and more easily extracted than healthy hairs. The skin is reddened, swollen, and slightly itchy. A whitish matter may be seen on the diseased skin and hairs, which is due to the sporules of the fungus. The hairs suddenly fall off from the affected parts, and a round bald patch is left, which is perfectly white, contrasting in its whiteness with the parts of the scalp or skin provided with hairs. The fungus may also be developed in the nail, like favus (Bazin). There is an affection which should be distinguished from the *Porrigo decalvans* (or *Alopecia circumscripta*), characterised by a rapid disappearance of pigment from both skin and hair, with or without *alopecia.* M. Bazin includes it in his *Tinea achromatosa* (*Teigne achromateuse*), but does not mention the fact that *alopecia* is not constant; and

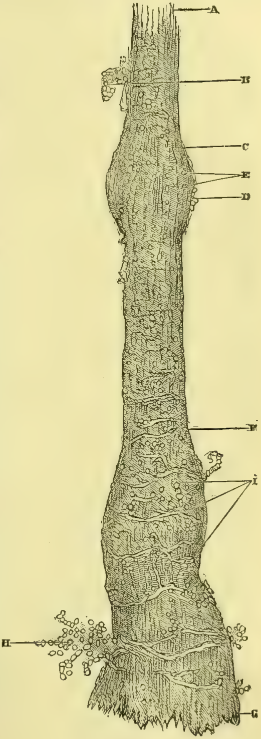

Fig. 79.*

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* Fungus of the hairs resulting in *Alopecia.*—(A, F.) Lower part of the hair; (F, G.) root of the hair without the capsule; (C.) spheroidal swelling of the hair, due to the accumulation of sporules (E.) between the longitudinal fibres of the hair; (D.) rupture of the longitudinal fibres; (I.) sporules and tubes of the parasite; (H.) a group of sporules proceeding from (G) the ruptured root (Anderson).

states that a parasitic plant is present. It is probable, however, that something more than a fungus exists, to cause the total disappearance of pigment from a considerable portion of dermis. Besides, when the hairs return, they are at first quite white and downy, like those on children, and only gradually regain colour; whereas, if the *vitiligo* were owing to a plant, they would most likely not grow at all. The disease appears to be allied to those obscure pigmentary changes which have a much deeper seat than the surface of the body (PARKES). *Vitiligo* is sometimes a congenital affection, and seems to consist in an abnormal distribution of the pigment of the skin; so that there are irregular patches which are quite white, and altogether wanting in pigment, but which are surrounded by skin provided with an excess of colouring matter. The hairs proceeding from the portion of the skin deprived of pigment are similarly colourless. The disease is not quite so readily transmitted as *Tinea tonsurans,* but still it is capable of being transmitted from one person to another, so that children so affected should be separated from their companions (ANDERSON).

Treatment consists—(1.) In preventing the spread of the disease circumferentially. All the hairs within a quarter of an inch of the circumference of the patch ought to be carefully extracted. The head should be washed daily with soft or black soap. All the *downy hairs* within the patch must be similarly removed till healthy hairs begin to grow; and some of the parasiticide lotions or ointments must be industriously used. (2.) Stimulants, or even blisters, must be applied to the surface of the bald patch after the fungus has been destroyed. A mixture of equal parts of *collodium* and of *ether cantharidalis* (*collodium vesicans*) is the most useful stimulant. The following lotions may be found advantageous alternately with the *collodium* stimulant, namely:—

℞. *Liquor Ammoniæ,* ʒiss; *Ol. Olivæ,* ʒii.; *Ol. Macidis,* ʒss.; *Spiritus Rosmarini,* ʒiv.; *Aq. Rosæ,* ℥ii.; *misce bene.* To be used night and morning, applied over the bald patches.

Mr. Startin recommends a lotion nearly similar, namely:—

℞. *Spt. Ammon. co., f*ʒi.; *Glycerine, f*℥ss.; *Tinct. Cantharidis, f*ʒi. to *f*ʒii.; *Aq. Rosmar.,* ℥viii.

Mr. Erasmus Wilson recommends the following:—

℞. *Ol. Amygdal.; Liq. Ammoniæ,* ā ā ʒi.; *Spt. Rosmarini; Aq. Mellis,* ā ā ℥ii.; *misce.* To use as a lotion.

Of course, none of these agents have any good result if there are no bulbs or roots of hair left whose growth can be stimulated by such application; but when hair falls off, or breaks away after debilitating illnesses, or from dryness of the scalp, they are often useful.

TINEA FAVOSA—*Syn.*, FAVUS, PORRIGO FAVOSA.

LATIN EQ., *Tinea Favosa*—Idem valet, *Favus;* FRENCH EQ., *Teigne Favosa;* GERMAN EQ., *Tinea Favosa*—Syn., *Favus;* ITALIAN EQ., *Tigna Favosa.*

Definition.—*A fungous parasitic disease, composed of cup-shaped scabs, sometimes distinct and separate, at other times indistinct or confluent. The fungi* (Achorion Schönleinii, Puccinia favi) *are capable of being implanted by transference from one person to another. The hairy scalp is its most common site, but the disease may be developed on the face, neck, or limbs.*

Pathology.—The disease depends on a cryptogamic fungus, which has been named the *Achorion Schönleinii*, after Schönlein, who was the first to suggest that the *yellow favus* crusts in *Porrigo lupinosa* and *P. scutulata* were composed of a vegetable parasite. The disease with which it is associated is now called indifferently *Favus*, *Tinea favosa*, or *Porrigo scutulata.* The primary seat of the parasite is in the depth of the hair follicle, outside the layer of the epithelium covering the root of the hair, which forms the "inner root-sheath" of Kölliker. By using a concentrated solution of *liquor potassæ*, to make the parts transparent, the fungus may be observed with the microscope in the follicle round the hair at the place where it passes through the epidermis. A second form of the disease is that in which the plant is found in depressions on the surface of the skin, forming the yellow honeycomb-like masses which gave the specific name *favus* to the disease, and which, from their frequent buckler-like shape, suggested the term "scutulata." A cuticular elevation is seen, beneath which is a small favus. When the cuticle is raised, a drop of pus sometimes issues; hence the error of those who have considered this disease always pustular. Generally, however, there is no pus or liquid of any kind: the plant grows, and the cuticle over it (supposing it has not been forcibly detached) finally separates, leaving the favus exposed to the air. A third form of the disease is that in which the fungus attacks the nails, and occurs for the most part in those who have been long affected with the favus of the hair follicles, the fungus taking root and germinating beneath the nail (Fig. 80). After the spores have commenced to germinate between the superficial and deep epidermic layers, the nail becomes thickened over the affected part, and its colour becomes gradually more and more yellow, owing to the favus matter shining through it. As the fungus increases in growth, it gradually presses on the nail, rendering its longitudinal striæ very evident, and ultimately leading to the formation of fissures in it. As the pressure on the nail increases, its substance gets thinner and thinner, till perforation occurs; and then a favus cup

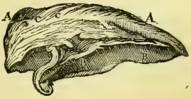

Fig. 80.*

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* (A, A.) Upper surface of nail; (B, B.) lower surface of nail; (C, C.) *favus* matter (white in the woodcut, yellow in the original), running upwards and forwards between the laminæ of the nail (ANDERSON).

makes its appearance externally, but more or less deformed (ANDERSON). At first there is, at the point where the favus is about to form, only an increased secretion of epidermis; and sometimes the under surface of the favus is coated by cuticle, which separates it from the compressed and attenuated derma. As it increases in size, and becomes more prominent, the epidermic covering is ruptured. Each favus crust is also enveloped in a capsule of amorphous structure, within which is inclosed the true favus matter (Fig. 81). The *favus* consists of the *mycelium*, the *spores*, and the *receptacles* of the *Achorion*, together with a finely granular amorphous layer, which forms the external coat of the favus, and is the representative of the amorphous "stroma" which accompanies the mycelium of *fungi*. In the favus another and distinct fungus can sometimes be found—namely, the *Puccinia favi*, which is easily recognised; it has one extremity (the body) rounded and composed of two cells of unequal size, a superior and an inferior; the other extremity is prolonged into a jointed stem or trunk. When a little of the favus matter is broken up and examined micro-

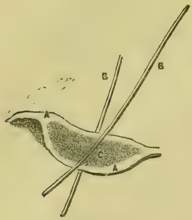

Fig. 81.*

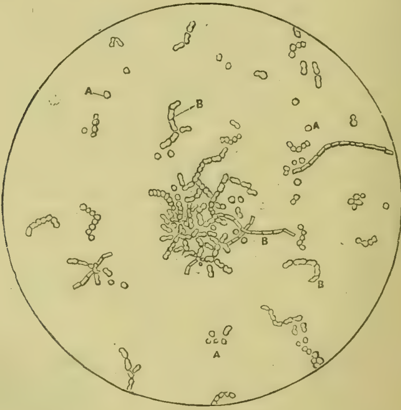

Fig. 82.†

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* Favus cup.—(A, A.) Amorphous envelope; (C.) favus matter; (B, B.) hairs traversing the favus cup (ROBIN).

† Fungous matter from a favus crust.—(A, A, A.) The isolated sporules; (B, B, B.) chains of sporules (Dr. ANDERSON).

scopically, after being acted upon by solution of potash, it is seen to consist of numerous little oval or rounded bodies, the sporules of the fungus having a diameter of about $\frac{1}{3000}$ part of an inch (Fig. 82). A number of cells united end to end form simple or jointed and branching tubes (Figs. 83 and 84), developed from the sporules (Fig. 82). Little granules or nuclei may be seen in the interior of the spores. The tubes vary in diameter, and hairs in the vicinity of the favus crusts are impregnated with the fungus. The disease has been communicated by inoculation from man to man, and from mice to cats, and thence to man (Bazin, Draper, Fox, Anderson).

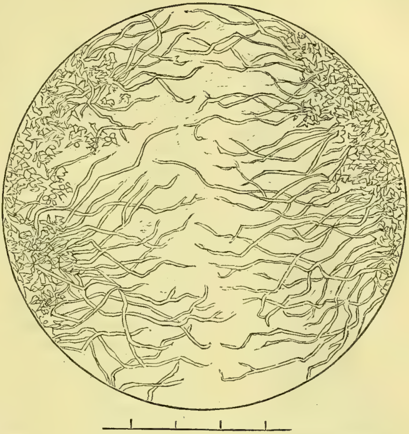

Fig. 83.*—$\frac{1}{1000}$ of an inch × 340 diameters (Dr. Anderson).

Symptoms.—Favus is the most common and the most inveterate form of scald head. The disease commences with a slight pruritus or itching of a few hours' duration, followed by an eruption of small red *vari,* sensible to the touch and to the sight. These augment in size, and before twelve hours have passed, a yellowish point forms on each of their apices, at first so small as to be only visible under a glass of considerable power. The surface appears now as if covered with specks of a sulphur-yellow colour, and each *varus* appears

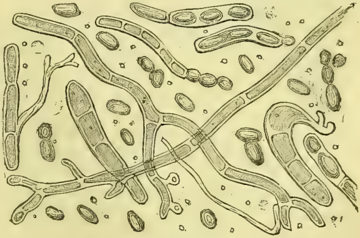

Fig. 84.†

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* Fungous matter from a favus crust, showing branching tubes running inwards to the centre of the figure from the epithelial scabs and sporules at the edges (Anderson).

† Showing the mode of reproduction of the *Achorion,* or fungus of the favus (after Bennett).

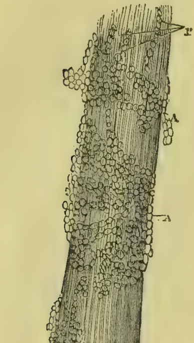

Fig. 85.*

as if set in the skin, with an umbilicated or depressed centre. Any fluid exudation concretes into a dry, brittle, candied, honeycombed-looking scab or crust, which retains the form of the pustule, is similarly cupped or depressed in the centre, covered by the epidermis, while its under surface is marked by a small mammary process which corresponds to the depression of the pustule. The honeycombed appearance of the scab gives the peculiar character of the disease, and hence the term "favus." The crust continues to increase, still preserving its circular form and depressed centre, till it occasionally attains a magnitude of five to six lines in diameter. When the crust is recent, it is of a yellow or fawn colour; as it becomes older its hue becomes lighter; and, as it is easily reduced to a powder, it has been compared to pulverised sulphur. The number of favi is considerable, and they commonly appear in crops, affecting the same or different parts of the head at distant intervals. They may be either distinct or confluent. When very numerous they are confluent, but the cupped form of the individual crusts may still frequently be recognised; and, according to Rayer, should this peculiar form be lost through the copiousness of the secretion, still, by removing the superficial layers, each particular favus, with its central depression, may in general be made out. At a more advanced stage of the disease the epidermis disappears, and a viscid fluid is secreted in such abundance as to form one entire encrustation over the whole head; hence the *Porrigo larvalis*—mask or vizor-like scald head. The smell of the scab is peculiar, and has been compared to that of the urine of a cat, or of a cage in which mice have been kept. It is probably due to a species of alcoholic fermentation (*methylamine*) in connection with the vegetable growth (LOWE). When a crust of recent formation is removed, a circular depression, wider and deeper than the favus is seen. At a more advanced stage the ulceration penetrates below the dermoid tissue. Indeed, it may not be possible to remove a crust without deeply wounding the scalp, and producing considerable hæmorrhage. In severe cases a deep and extensive ulceration takes place, which has penetrated even to the bones of the cranium (ALIBERT).

The *Porrigo lupinosa* and *Porrigo scutulata* are accidental and rare varieties, in which the scab resembles a lupine rather than the cell of the honeycomb. Or the appearance of the scab is shield-like; and when of some extent and well-marked, the patch is soft, doughy, and painful when pressed upon. Some of the hairs appear to be removed with their roots, while other hairs are broken off near the scalp, the roots remaining, which are readily removed by friction, and if pulled, have scarcely any hold of the scalp.

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* Hair with favus fungus.—(A, A.) Chains of sporules projecting beyond the edges of the hair; (B.) sporules between the fibres of the hair; (C, D.) broken-up root-end of the hair, with masses of sporules between the *laminæ* (after KUCHENMEISTER).

Treatment.—The treatment of the various forms of *favus* is now very strictly determined. Some practitioners, however, still rely entirely on a constitutional treatment, such as small doses of *rhubarb* and *soda*, small doses of *mercury*, some preparation of *iron;* or on vegetable tonics, as the *infusion of cascarilla* or *compound infusion of gentian.* With such treatment, if the health improves, it is believed the fungus will spontaneously disappear. Others, again, as entirely rely on a local treatment, attempting to exterminate the disease by cauterisation, or by applying some favourite ointment. As in the last-described disease, the cryptogamous parasite must be destroyed, and its germs eradicated. The best method to accomplish this is, in the first instance, to shave the head and apply a poultice till all the scabs, or nearly so, are removed; and this being affected, the whole hairy scalp, or site of the favus fungus, should be anointed with some one of the following applications:—The *tar ointment* (*ungt. picis liquidæ*) has hitherto been the orthodox application. This ointment should be washed off night and morning with soft soap and water, and be as often re-applied. The head should be shaved twice or thrice a week, and where there are other children, the affected child should be isolated as much as possible, to prevent the disease from spreading. This form of porrigo, in the early stages, will sometimes yield by washing the part with the *oleum terebinthinæ* night and morning, and cutting the hair close. Two parts of *carbolic acid* to three parts of *glycerine* and *water* used twice a day, with the daily use of carbolic acid soap, have been also found of service.

But *favus* is more often rebellious to every mode of treatment. It has been known to yield to fomentations, or to bread poultices. The application of the lunar caustic round the patches, about a line from their outer margin, is another favourite method of treatment. In the later periods of the disease, Dr. Willis recommends—

> A solution of *sulphate of copper*, in the proportion of seven grains to ten ounces of water; or of the *nitrate of silver* in the same proportions. The mild ointment of the *nitrate of mercury*, a salve of the black sulphuret of the same metal (*sulphuretum hydrargyri nigr.*, ʒj. ad. ʒij., *adipis* ʒj.); the *unguentum picis*, an unguent of the *cocculus Indicus* (ʒj. to ʒij., *adipis* ℥j.), may be tried one after the other; and in different instances each may have the merit of the cure.

The most effectual remedy is unquestionably the eradication of the affected hairs, and the use of such parasiticides as have been already mentioned under the different forms of *Tinea.* Dr. Anderson finds that when *favus* affects the head, all treatment is absolutely useless except *epilation.* The other methods are merely palliative, the disease reappearing whenever the treatment is stopped. Treatment with olive oil is very useful in many cases before proceeding to epilation. After the hair is cut short, and the favus crusts removed with poultices, the parts affected should be smeared thoroughly night and morning with fresh almond oil; and once or twice a week the head should be washed thoroughly with soft soap and warm water. After a few weeks of this treatment the hair becomes less friable, and *epilation* is much more easily and efficiently performed, and does not cause nearly so much local irritation. The hairs are to be removed *singly* with the forceps, not pulled out along with all the healthy growth in their neighbourhood, as used formerly to be done by the barbarous application of the pitch-cap. When favus occurs on surfaces not particularly covered with hair it yields at once to the application of a solution of *sulphate of copper*, or of the *nitrate of silver in water*, or to the *solution of sulphuric acid*, as recommended by Sir William Jenner. The treatment of favus recommended by Robin and Bazin is epilation, and the application of the *corrosive sublimate solution*, or of *acetate of copper ointment* (1 part to 500 of lard), to kill the plant still remaining adherent to the hair follicle.

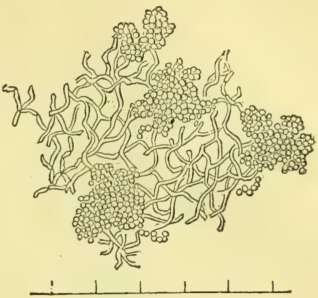
TINEA VERSICOLOR—*Syn.*, PITYRIASIS VERSICOLOR.

LATIN EQ., *Tinea Versicolor;* FRENCH EQ., *Tinea Versicolor;* GERMAN EQ., *Tinea Versicolor*—Syn., *Pityriasis Versicolor;* ITALIAN EQ., *Tigna Versicolore.*

Definition.—*A fungous affection of the skin, characterised by one or more broad irregularly-shaped patches of a yellow or yellowish-brown colour, occurring most frequently on the front of the neck, breast, abdomen, and groins, having a predilection for those parts of the body covered by clothing. The patches do not generally rise above the surface of the skin; and there is usually some degree of itching.*

Pathology.—On passing the hand gently over the diseased surface, it may be less smooth than the surrounding skin. It may be seen to be the seat also of a very fine desquamation, or at least of an abnormal condition of the epidermis. Thus the surface of the affected parts may have a *dusty-like* appearance, *like bran*, and so may merit the name of *Pityriasis versicolor;* but in no other respect has it anything in common with ordinary *pityriasis*—a disease altogether unconnected with parasitic *fungi*. The scales which desquamate from chloasma have a yellowish colour when contrasted with the white scales of such scaly diseases as *Pityriasis vulgaris* or *Psoriasis*. Hence the term Chloasma—from χλοος, "a greenish-yellow colour"—appears more suitable than any of the other names by which the disease has been described.

It commences by little spots about the size of a pin-head, which extend circumferentially; circular spots form and unite so as to produce large irregular patches, which may extend till the greater portion of the skin of the trunk is affected. The skin of the diseased parts has a peculiar brownish colour; but the depth of tint varies from the slightest increase of colour to a shade almost black. The colour has been said to resemble diluted bile. A microscopic fungus, to which the name of *Microsporon* *furfur* was given by Robin, is the parasite associated with the disease. It was discovered by Eichstädt in 1846. Soon afterwards it was described by Sluyter and by Sprengler, who gave a drawing of it. On putting a little of the dust from the desquamating surface under the field of the microscope, and adding a drop of *liquor potassæ*, scales of epidermis are seen mingled with the sporules and tubules of the fungus. The sporules are oval or rounded, and usually collected into large clusters like bunches of grapes, and are so characteristic as almost to be pathognomonic (ANDERSON). The tubes are short and branching. Dr. Anderson and Mr. Startin give numerous instances which prove that chloasma is a disease capable of being propagated from one person to another. It is a common affection with scrofulous persons especially, and may not unlikely be favoured by wearing the same flannel day and night, neglecting to wash the body for fear of catching cold (ANDERSON). It is not uncommon for such people to wear the same flannel next to the skin for a week, a fortnight, three weeks, and among the poor even for a month. And it is by no means an uncommon thing for them to wear the same flannel night and day, not once removing it from the moment it is put on till the time it is considered desirable to have it washed. The consequences of such habits are an accumulation on the surface of the skin of its secretion, and of undetached epithelium, and the consequent formation of a nidus favourable to the growth of the *Microsporon furfurans* (Dr. JENNER, *Med. Times and Gazette*, 1857, p. 651).



1,000th of an inch × 340 diameters.

Fig. 86.*

Treatment.—Local applications constitute the principal part of the treatment. *A solution of the bichloride of mercury, in the proportion of two grains to an ounce of water*, applied over the affected parts once or twice daily, is generally effectual in destroying the progress of the fungus. Mercurial or sulphur baths have a similar effect, either singly or combined, care being taken to avoid salivation. The use of black soap night and morning is recommended by Dr. Anderson, or the use of the following mixture :—

℞. *Bichloridi. Hydrarg.*, ℈i. ; *Alcoholis*, ℥ss. ; *Saponis Viridis, et Aquæ distillatæ*, ā ā ℥iiss. ; *Ol. Lavandulæ*, ℈i. ; *misce.* To be used night and morning in the same way as the black soap ; but if the gums get tender, its use must be suspended.

Great attention must be paid to cleanliness. The patient should change his flannel clothing very often, and should not sleep in the same flannels that are worn during the day.

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* Shows the grape-like arrangement of the sporules and the short branching tubes of the *Microsporon furfur* in *Chloasma* (after Dr. ANDERSON).

SCABIES—*Syn.*, ITCH.

LATIN EQ., *Scabies*—Idem valet, *Psora;* FRENCH EQ., *Gale;* GERMAN EQ., *Scabies*—Syn., *Krätze;* ITALIAN EQ., *Rogna.*

Definition.—*Lesions consisting of an eruption of distinct slightly acuminated papules or vesicles, accompanied with constant itching, due to the irritation caused by the burrowing underneath the epidermis of a female Acarus* ("Sarcoptes scabei"), *for the purpose of depositing her eggs.*

Pathology.—The nature of this parasite has been described in Vol. I., under "Parasites."

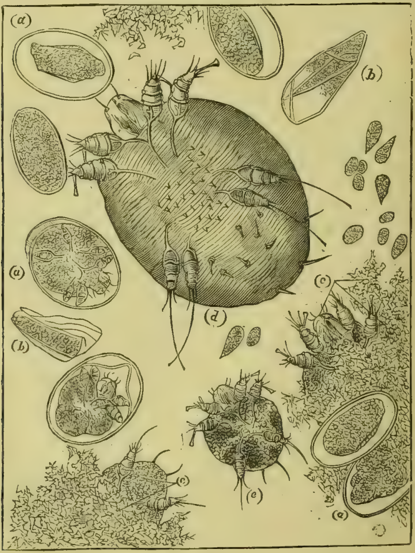

Fig 87.*

Symptoms.—The phenomena of the eruption of scabies are more often papular than vesicular; and the markedness of these phenomena depend partly on the length of time that the person has been affected, the number of *Acari* developed, and the degree of sensibility of the skin. It is known to infest sheep and dogs (YOUATT), and therefore hair does not preclude its existence. It seems, however, to prefer delicate parts of the skin; for example, the inner surfaces of the fingers, and folds of the skin between the fingers, the wrists and palms of the hands, the penis in the male and nipples in the female, as well as the hips, the feet, the umbilicus, and axillæ. Itching, increasing at night, first attracts attention, and is a characteristic symptom. It becomes general all over the body, and the scratching aggravates the eruption. The *prurigo* of itch is generally most expressed on the forearm, lower part of the abdomen, and the upper and inner part of the thighs. Vesicular eruption is most usual on or about the fingers and nipples of females; and pustules may be met with in children whose skin is delicate, especially on the hands, feet, and hips.

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* Crust from a case of the so-called *Scabies Norvegica* which occurred in Würtzburg.—(*a, a, a.*) Eggs of the *Acarus* in various stages of development; (*b, b.*) egg-shells; (*c, c.*) fragments of *Acari;* (*d, d.*) female *Acarus;* (*e.*) larva. The little oval irregular-shaped masses are presumed to be the excrement (after ANDERSON).

There is a severe form of scabies common in Norway and some parts of continental Europe, in which the greater portion of the skin of the body becomes thickened, the natural furrows increase in depth, the pigmentary deposit is greatly augmented, and a fine white desquamation covers the surface. Here and there papules may be seen, either with dots of coagulated blood, or with whitish crusts on their summits. In these crusts portions of *Acari* and their exuviæ, excrement, and eggs may be found. The hairy scalp, covered with a crust which adheres firmly, of a bark-like consistence and yellow colour, is studded with fine openings for the hairs, which are glued together. On the under surface, and in the furrows of this crust, multitudes of *Acari* may be seen. Such are the main features of a case recorded by Bergh, of Copenhagen, and related by Dr. Anderson in his excellent little treatise already referred to. The itching was intolerable. The crusts were principally composed of the *Acarus,* its exuviæ, excrement, eggs, and egg-husks (Fig. 87). A piece of the most superficial and dense part of the crust, less than half a line square, contained two female *Acari,* eight six-legged young, twenty-one pieces *Acari,* six eggs, fifty-three egg-shells, and about 1,030 pieces of excrement. "In the deepest and softest parts of the crusts, amongst the remains of deceased generations—partly in holes and passages, partly between particles of the crusts, partly on their free surfaces—masses of living *Acari* wallowed and tumbled about." The cases in which such a severe form of *scabies* has occurred have been characterised by extreme filth and neglect of treatment, and the irritation and course of the disease has tended to a fatal termination by pneumonia and hyperæmia of the brain.

Treatment.—Such applications are to be made to the skin that, while tending to kill the *Acari,* they will not increase the irritation of the dermis; and if the *Acari* are thus destroyed, the eruptions will in general subside in due course. If much irritation of the skin prevails, *warm baths* are to be prescribed, and *opium* may be given internally. In healthy adults the whole body of the patient ought first of all to be thoroughly scrubbed over with good black (soft) soap, and the process continued for at least half an hour. The patient should then get into a warm bath, in which he should remain for another half hour. Having thus washed and dried himself thoroughly, he is to rub himself over with the following ointment:—

℞. *Subcarbonatis Potassæ*, ʒi.; *Sulphuris*, ʒii.; *Axungiæ*, ℥xii.; *misce.*

Next morning a warm bath is to be taken, to clean the surface of the body from the remains of the anointing of the previous night. The cure ought now to be complete, so far as the destruction of the *Acari* is concerned (Hardy, Helmerich, Anderson). The genuine pomade of Helmerich is one-third stronger than that which has been just quoted from Dr. Anderson. The potash in the black soap and ointment acts as a solvent of the epidermis, and thus allows the sulphur to come into more immediate contact with the *Acarus.* A warm bath and plenty of hot water ablution completes the cleansing process. The treatment may, however, be inapplicable to children, females, and men with delicate skins or constitutional affections of the skin. In them, although the principle of treatment is the same, the process of cure must be more slowly conducted by less powerfully irritant substances. The patient having cleansed himself thoroughly in a warm bath, with ordinary yellow soap, the following lotion may be applied:—

℞. *Calcis*, ℥ss.; *Sulphuris*, ℥i.; *Aquæ*, ℥viii. These ingredients are to be boiled and stirred constantly till a homogeneous mixture is produced, which is to be strained through a sieve.

These ingredients ought to produce a quantity more than sufficient for one person, and should be rubbed into the skin, not too roughly, every night for several evenings. The cases of scabies in the Belgian army are treated by this lotion (Velminskz, Anderson). When the person affected is predisposed to *eczematous* eruptions, the following application is recommended:—

℞. *Sulphuris, Olei Fagi*, ā ā ʒvi.; *Saponis Viridis, Axungiæ*, ā ā lbj.; *Cretæ*, ʒiv.; *misce.*

This ointment should be well rubbed in, after the skin has been prepared for it by the use of the warm bath and cleansing the body with common yellow soap. The potash in the black soap of the ointment acts as already stated, the chalk tends to remove the epidermis mechanically, the tar counteracts the tendency to eczema, and the sulphur destroys the *Acari.* The ointment ought to be left on overnight (if the skin is not too irritable), and should be washed off in the morning (Wilkinson, Hebra, Anderson). Specific printed directions should be given to each patient; and cards are useful for this purpose, similar to those in use in the Dispensary for Skin Diseases in Glasgow. The following are the directions *printed* on each card, which is given to each patient along with the quantity of ointment required:—

"1st. Scrub the *whole* of your body (except the head) as firmly as possible, without hurting yourself, with black soap and water.

"2nd. Sit in a hot bath for twenty minutes, or if you cannot get a bath, wash yourself with hot water thoroughly.

"3rd. Rub some of the ointment firmly into the skin of the whole body (except the head) for twenty minutes. Let the ointment remain on the body all night.

"Repeat these processes every night for three nights, and then return to the dispensary.

"Besides, put all your washing clothes into boiling water, and iron all your other clothes *thoroughly* with a hot iron."

The ordinary compound sulphur ointment of the pharmacopœia is also an efficient remedy. The cure being complete so far as the person is concerned, care must be taken to destroy the *Acarus* and its eggs which may be amongst the clothes of the patient. For this purpose they should be exposed to hot air at the temperature of at least 150° Fahr., or boiled in water, or exposed to the action of sulphur by steaming them amongst sulphur vapours. If such methods are systematically carried out, itch cases ought never to occupy hospital beds, either in civil or in military life.

CHAPTER XXIII.

THE PERNICIOUS INFLUENCE OF SOME POISONS.

THE College of Physicians indicates a consideration of poisons and their effects under the following heads:—(1.) Metals and their Salts; (2.) Caustic Alkalies; (3.) Metalloids; (4.) Acids; (5.) Vegetable Poisons; (6.) Animal Poisons; (7.) Gaseous Poisons; (8.) Mechanical Irritants; (9.) Poisoned Wounds. Time and space do not permit of such a detailed consideration, so that a few poisonous agents and their effects can now only be noticed here.

LEAD PALSY:—SATURNISM.

LATIN EQ., *Paralysis ex Plumbo;* FRENCH EQ., *Paralysie Saturniné;* GERMAN EQ., *Beilähmung;* ITALIAN EQ., *Paralisi Litargirosa.*

Definition.—*A series of morbid phenomena induced by the absorption of the salts of lead contained in solution in drinking-waters, or in various foods and drinks, or conveyed into the system through the integuments of those who are in the habit of handling the soluble salts of lead; or through the pulmonary mucous membrane of those exposed to the influence of vapours containing lead.*

Pathology.—(*a.*) *Causation.*—The theory of lead-poisoning is that, being absorbed, it produces a painful affection of the alimentary canal, termed *lead colic* or *painters' colic* (eq. *colum ex plumbo*). It may also affect the muscles, producing palsy. The introduction of lead into the system has taken place in a variety of ways. In France the pernicious effects were wont to occur from putting a lump of litharge into the *vin gâtée,* to cover its acidity and render it saleable; and from this custom having been practised to a great extent by the Pictones, or inhabitants of Poictou, the disease has been named *Colica Pictonum.* In the cider counties of Great Britain lead colic formerly existed to a great extent. It was termed Devonshire colic, or *Colica Damnoniensis.* But such impregnation of cider with lead was usually the effect of accident, from the troughs in which the apples were crushed having the different pieces of stone of which they are composed clamped together with iron, and fixed by melted lead. In some districts it was the practice to line the entire press with lead, or to tip the presses with that metal. It was also a custom, almost universal, to make the upper part of the boiling vessel of lead; while some brewers, in managing weak ciders, put a leaden weight in the cask to sweeten the liquor. From these and other causes, Sir George Baker found the cider to contain four and a half grains of lead in eighteen bottles, or a quarter of a grain in each bottle. In the West Indies, lead poisoning was produced by using leaden worms to the stills, so that the rum became impregnated with lead. There are many other minor sources of poisoning by lead; such as keeping pickles or preserves in glazed earthen vessels, and colouring confections with preparations of lead. The still and other machinery used in the distillation of fermented liquors being now constructed of metals so combined as not to be acted upon by acid fruits or sugar, diseases from the action of lead are no longer so common as they were wont to be. They are confined principally to labourers in the lead manufactories and to painters. The use of paint where lead exists is the most common source of its absorption in this country, especially in the manufacture of white lead; and hence house painters and those who are employed in the manufacture of *white lead* are most frequently affected. In the east-end of London there are several factories in which *white lead* is made, and many persons suffer from lead-poisoning contracted in those factories. The paint called "flatting" (or that which is mixed with a large amount of turpentine, so as to give a *flat, dead,* or *non-glistening* surface) is the most injurious to the workman. The turpentine, readily passing off by evaporation, carries with it a small supply of lead, which is constantly and gradually inhaled, or it is left on the skin to be absorbed, or mixing with saliva it gets into the stomach. The use of lead ointment, as in the treatment of piles or prolapsus ani, may also induce the specific effects of lead poison. By some one of these ways the system becomes affected, first through the circulation of the blood, and subsequently by the constituent tissues of the organ combining, in some form or other, with lead, which is thus deposited in them. The quantity of lead necessary to produce its specific results, or the time it takes to accumulate in the system when introduced, is not determined; and both the dose and the time vary greatly in different individuals. Sometimes all its most pernicious effects are produced by one dose taken by the mouth. If introduced by the skin, months, and even years, may elapse before the system is under its influence. As a general rule, however, a much smaller dose will produce colic than is necessary to produce palsy. Dr. J. S. Ramskill's averages, in 1875, founded on the statement of twenty-five patients who applied at the London Hospital from the *white lead* factories, showed that women were attacked after working about four months, and the men two years; the period varying according to the individual occupation. The earlier suffering of the women was due to the fact that nearly all of them were employed in the more injurious occupations of carrying and otherwise moving the dry, powdery, white lead. Some men, suffering from lead-poisoning, said that they had been employed for many years, one even twenty-seven years, without suffering greatly; but they had worked amongst the blue, that is, the metallic lead (*Brit. Med. Journ.*, May 8, 1875, p. 599). All ages, both sexes, and all classes are liable to the poisonous action of lead, but the workers in this metal have been at all times the greatest sufferers. Dr. Ramskill's analysis gave the largest number attacked as men under forty years of age. The stove operations and drying white lead seem to be most influential in producing lead poisoning. Those so employed suffer soonest, and usually have to discontinue their work.

(*b.*) *Morbid Anatomy.*—The fact of lead being absorbed and mingled with the blood is demonstrated by the circumstance that it has been obtained from the coats of the stomach of a dog poisoned by lead as late as a month after poisoning. It has been detected in the brain of the human subject (DUVERGIE and GUIBOURT). It has also been found in the muscles (BUDD). Dr. Henry Burton, of St. Thomas's Hospital, first pointed out that, in persons under the influence of lead, a peculiar blue line could be seen on the borders of the gums; and Mr. Tomes subsequently explained its production as due to a sulphuret of lead. Dr. C. Hilton Fagge has recently described the microscopical appearance of the gingival structures when thus affected, and the conditions under which the lead line is developed. He shows (as originally pointed out to him by Sir W. Gull) that the line is not continuous, but consists of a series of dots arranged side by side at pretty uniform distances. These dots correspond with vascular points which exist in the normal mucous membrane of the gum. The discoloration is not uniform, but is distributed in the form of rounded loops, which are either portions of blood-vessels, or minute papillary processes of the deeper layer of the mucous membrane. The pigmentation is caused by the presence of granules scattered at a little distance from one another, or collected into dense masses. They are sometimes situated in the interior of the smallest blood-vessels, and sometimes outside in the tissue adjacent to them. When the line is well marked it may surround the bases of all the teeth; or the margins of the gums may be uniformly blackened; or may extend over the membrane covering the whole alveolar process, and upon that which lines the lips and interior of the cheeks. This blue line is itself really *in* the gum, and not *on* its surface. When examined with a lens during life the dots which make up the line are seen to be black, and only look blue from being seen through a comparatively thick layer of translucent tissue. Wherever there is a gap in the row of teeth the line is wanting. It is to be regarded as a *precipitate* of the sulphide of lead from the blood, or even in some sense as an excretion. It may exist at one time and not at another (*Med.-Chir. Trans.*, Vol. LIX., May 9, 1876). From the observation of the late Dr. George Wilson of Edinburgh, it appears that there are various tissues of the body for which lead has an affinity, and that it is more apt to be found in some organs than in others. The stomach and cœcum of a pony that died a fortnight after being removed from the sources whence lead was received into its body through the ingesta, having been carefully analysed by this able chemist, it was found that while the contents of these viscera did not contain the metal, the substance of their tissues yielded it in small but manifest quantity. In a mare dying under similar conditions, the following organs were examined—(1.) A part of the lungs; (2.) a part of the heart; (3.) a part of the large intestine and contents; (4.) a part of the stomach and duodenum; (5.) the spleen; (6.) one kidney; (7.) a portion of the liver. The spleen yielded the most abundant and most deeply coloured precipitates, while the intestinal canal gave the faintest indications. Next to the spleen the liver yielded most, afterwards the lungs, then the kidney, next the heart, and least the intestines. The general result of these analyses show,—*First*, That in cases of slow poisoning with lead, the metal comes to be diffused through the entire body, and exerts its poisonous action, though in an unequal degree, on every organ; and, *Second*, that lead having once entered the body, leaves it again very slowly, so that long after an animal has ceased to receive lead in its food or drink, or by any other medium, we may expect to find the metal in its tissues. Constrictions of the colon and of the cœcum and intussusception of the bowels have been recorded (De Haen, Mérat, Dubois de Rochfort).

In the paralysis arising from the poison of lead the tissues of the muscles and nerves are early affected, and at a subsequent period the nervous centres become implicated. The muscles seem to be first affected, and through them the nerves participate in the contamination, which gradually advances to the nervous centres in the severer forms of the disease. This is shown by the fact that local paralysis always precedes, and generally for some considerable time, those phenomena which indicate disease of the nervous centres. The evidence is also abundant which proves that it is lead existing in the affected tissues which thus contaminates and impairs their function. The morbid appearances in the brain and spinal cord are generally such as denote imperfect or depraved nutrition of those centres. The brain is pale and soft, and its convolutions wasted, the *sulci* between them wide, and sometimes patches of white softening are to be seen in the hemispheres; and this seems to be more particularly the case in those who have had paroxysms of an epileptic nature, and in whose brains lead has been detected (Todd).

Symptoms.—When the dose is of such intensity as to produce *colica Pictonum*, the symptoms do not differ, except in their greater intensity, from those which mark ordinary colic. There is the dragging and twisting pain, the relief by pressure, the absence of fever, the unhurried pulse, the constipation (only more obstinate), and, in the worst cases, vomiting. Andral, who treated upwards of 500 cases at La Charitié in the course of eight years, says it is not strictly true that the pain in lead colic is always diminished on pressure. In the greater number of cases pressure neither augments nor diminishes the pain. In some cases the sufferings of the patient are increased by it. It is also as common to find the abdomen distended with gas as to find it drawn in, and the *rectus* muscle strongly contracted on both sides. Occasionally there may be an attack of epilepsy. The duration of the constipation varies. Three or four days may elapse before a stool is procured, and fifteen days have been known to elapse without a stool. As soon as the bowels act, the severity of the colic is mitigated; every symptom is gradually relieved, and the colic generally terminates within a week.

In slow poisons (as in the course of time from working in lead) Dr. Ramskill's results show that the patients, almost at the outset, become troubled with pain in the limbs and abdomen especially. Weakness with a feeling of great lassitude was an early and very common symptom. The patients frequently felt sick and vomited. Constipation was usually an early trouble, for which they commonly took some aperient medicine, seemingly with relief. Not a few had an appearance which at once suggested lead-poisoning—the face had a dirty yellow or sallow colour. The patients were generally a good deal wasted and walked feebly, more or less bent forwards. Some were so prostrate that they moved with difficulty, and availed themselves of any opportunity to sit down. Pain and weakness seemed to be the most common and most distressing symptoms. In some the pain was confined, for the most part, to the abdomen; and although a few had, at the time they applied for relief, severe paroxysmal pain, so called "lead-colic," in the majority it was not so urgent, but rather of a dull, wearing, constant, aching character. With the abdominal disturbance the patients usually complained of pains in their limbs, and, in some of them, it was confined to these parts, chiefly the joints, or disseminated through the muscles as well. Some complained of pain in the head, and occasionally this symptom was very distressing. The skin may be exceedingly pale. Nausea and vomiting were frequent symptoms, and tremors were common.

When palsy is the result of the absorption of lead, a painful state of the arms precedes it, and it is in general limited to the upper extremities. The nerves of the forearm and hand become first affected, the extensor muscles of the hands and fingers become paralysed, so that when the arms are stretched out, the hands hang down by their own weight, causing what the patients term "*wrist-drop.*" Palsy may be limited to one finger. More commonly it affects the whole arm, and so completely that the patient can execute no movement with it. When lifted up it falls like an inert mass. Sometimes the extensor muscles of the limb are alone affected. In this case the hand may be strongly closed by the powerful and unresisted action of the flexors. When the case continues for a few weeks, the posterior surface of the forearms (where the extensor muscles are situated) is rendered concave, from atrophy and consequent shrinking of the muscles. The arm loses its plumpness, and the interosseous membrane may be felt. The muscles of the ball of the thumb are in a similarly wasted state. In general both arms are palsied, but not equally so, one being slightly more affected than the other. The pernicious influence of lead is also shown by emaciation. The skin is dry, harsh, and unperspirable. The complexion is sallow, or the skin is pale or yellow, with a yellow conjunctiva. The breath is offensive, and a sweetish taste is experienced in the mouth. The pulse may be slower than normal. Disorder of sensation prevails in various forms, such as *numbness, formication,* neuralgic pains, headache, with aching in the limbs and joints, or there may be *hyperæsthesia.* There may be also partial blindness, caused by—(1.) Amblyopia, usually transient, without ophthalmoscopic changes; (2). atrophy of the optic nerve; (3.) optic neuritis. The transient amblyopia has been seen in acute cases of *saturnism* after but a short exposure to the influence of lead. In amblyopia of longer duration there are signs of atrophy of the optic nerves. The discs are sharp-edged, pale, often greyish, and the arteries small. In the early stages there is simple congestion of the discs. The tint is uniformly red with softened edges, with little or no swelling. Gradually the redness fades, and a reddish-grey atrophy results, often with distinct white lines along the narrowed vessels. Sight has been much affected in all the recorded cases, the acuity of vision impaired, and the field presenting a central or peripheral defect. The loss commonly progresses till perception of light may be lost. This congestive atrophy is usually double, but the affection of one eye may precede that of the other. Chronic cases may present considerable *papillitis* with swelling, obscuration of the margins of the disc, concealment of vessels, and hæmorrhages. The arteries beyond the swelling are commonly narrowed, and the veins distended or of normal size. The affection is almost always double, and entails considerable amblyopia. At the same time slight degrees of neuritis are not uncommon in lead-poisoning without affection of sight. The conditions are analogous to the affections in *uræmia* and *diabetes;* and the occurrence of neuritis may coincide with symptoms of cerebral disturbance, headache, convulsions, or delirium (GOWERS, *Medical Ophthalmoscopy*, p. 207).

Diagnosis.—The colic of lead-poisoning can only be distinguished from ordinary colic by the history of the case, and by the blue line in the dental edge of the gums, which is present only where the teeth or their stumps are in the alveoli, and ceases where a tooth is completely wanting. The *palsy* is to be distinguished from *cerebral paraplegia* by the history of the case, by the integrity of the intellect, and by the blue line in the gum. The excitability of the muscles to the electric current is always much diminished in paralysis from lead, and often it is entirely lost (Dr. ALTHAUS). Such is the case, not only when the muscles are atrophied, but when the bulk of the muscles is only slightly diminished; and even after the voluntary movements have regained their former power, the excitability of the muscles to the electric current still remains impaired. Thus the relation of the muscles to the stimulus of Faradisation helps in doubtful cases to establish the diagnosis, as the excitability of the muscles is always either lost or diminished in lead palsy, whilst it is normal in spontaneous paralysis. *Therefore, when the muscles of a paralytic limb move well under the influence of the electric current, we may fairly conclude that there is no lead in the system* (ALTHAUS, *On Paralysis, Neuralgia, &c.*, p. 72). The diagnosis of saturnine atrophy and neuritis rests especially on the recognition of the signs of lead-poisoning, especially the line in the gums, the occurrence of gout, of colic, of wrist-drop, and of the presence of anæmia. It is by these symptoms that the neuritis is distinguished from that of primary encephalic affections. The possibility of a renal neuritis in cases of lead-poisoning must also be remembered (GOWERS, l. c.)

Prognosis.—The termination of lead colic, except where the dose has been in such excess as to produce death in a few hours, is always favourable; and those cases which prove fatal are generally such as have been exposed to the cumulative influence of lead for a long time, and who have been intemperate. The *palsy* does not appear greatly to affect the health of the patient; but in some cases it has hitherto not been cured or relieved. In general, he recovers, although perhaps not completely. Supposing both sets of muscles to be equally palsied, the patient usually recovers the use of the *flexors* before that of the *extensors*, so that he can carry a weight hanging in his hand before he can shave himself. This restoration of the lost power is usually accompanied by more or less pain. The duration of the palsy under any treatment is always long, and often lasts many months, and in some cases years. Both colic and palsy may occur an indefinite number of times. When epilepsy is produced, the fit does not differ from epilepsy due to other causes. Drs. Garrod and J. W. Begbie have satisfactorily demonstrated that lead-poisoning exerts a remarkable influence as a predisposing cause of gout; and my friend Dr. W. England, of Winchester, as the result of his experience, spontaneously volunteered to me a similar remark. Inveterate forms of dyspepsia may be traced in many cases to the influence of salts of lead in the drinking-water, its pernicious influence expressing itself differently in different constitutions, although never amounting either to colic or to paralysis. The restoration to health is always protracted. The lead passes off by the urine sometimes in large quantities, but very slowly (PARKES). Fletcher found in the urine of a man with lead colic no less than 4·8 grains of metallic lead in 100 grains of solid matter of the urine (*Dublin Med. Press,* 1848); and Dr. Parkes records a case in which the last exposure to the influence of lead was on the 20th December, 1852, and lead was found in the urine before treatment commenced on the 16th June, 1853, and the blue line was still perceptible below the edge of the lower gum.

In all forms of change in the optic disc, the prognosis must be guarded. It is least grave in toxic amblyopia, next in cases of pronounced neuritis, especially of acute course; less so in cases of chronic congestive change. In pronounced atrophy it is very unfavourable (GOWERS, l. c.)

Treatment.—The objects to be obtained in the treatment of *lead colic* are to procure stools, a copious discharge of urine, and perspiration, with a view of eliminating the poison from the body. To allay pain is also an urgent necessity. For these purposes five grains of *calomel,* fifteen grains of *jalap,* and one grain of *opium,* should be administered as soon as the patient is seen; and at the end of two hours about two ounces of *camphor mixture,* combined with a drachm or two drachms of *sulphate of magnesia,* and twenty minims of *tincture of hyoscyamus* may be given, and repeated every two or every four hours, till the bowels are freely evacuated, when relief more or less complete is obtained. The mixture should be continued at proper intervals for three, four, or five days, when the patient, though greatly weakened, has in general recovered. In a few cases, however, the pain continues, and with considerable severity, after the bowels have been freely evacuated. The practice in these instances is to apply a blister to the epigastrium, and to keep the blister open for a few hours. This additional application will generally complete the cure. The patient is also relieved if placed in a warm bath, and at the same time directed to inject repeated enemata of hot water, that stools may be readily obtained. In the absence of the warm bath, a large linseed or mustard poultice should be applied over the abdomen.

With respect to the treatment of *lead palsy,* it has been believed that sulphur has the power of neutralising the effects of lead by forming some innocuous compound with it. It is not known, however, whether any such compound is really formed; but *lead poisoning* in its chronic and constitutional forms is more successfully treated by *sulphur baths* than by any other agent except *iodide of potassium,* to which they form a most important adjuvant. The ingredients of these baths consist of from *two* to *four* ounces of the *sulphuret of potassium,* mixed with from twenty to thirty gallons of water. The *ergot of rye* (*secale cornutum*) has been said to produce a considerable increase in the power of the flexor muscles of the arm in about a fortnight, and the improvement gradually extends to the flexors, till at the end of about three months the patient has recovered. This may have been the natural result of elimination of the lead. The experiments of Orfila long ago rendered it probable that lead is removed from the body by the kidneys; and *iodide of potassium* promotes the elimination of lead in this way. It may be used with advantage combined or not with the *citrate of iron,* the use of iron, in some form or other, having been found of benefit in cases of palsy from lead contamination. Dr. Parkes has chemically proved that lead can be made to pass off by the urine by the action of *iodide of potassium,* in the same way as mercury is known to be eliminated. The principle upon which the *iodide of potassium* acts is that the lead is in actual union with the affected tissues, being retained among them as an *insoluble* compound; and the *iodide of potassium,* after its absorption into the blood, combines with the lead, and forms with it a new and soluble salt. The poison is thus liberated from its union with the injured part, dissolved out from the damaged fibre, and once more set afloat in the circulation. Thus the poison and the remedy are cast out together by the urine (MELSENS, WILLIAM BUDD). It is necessary, however, to notice the dangerous phenomena which may at first supervene on the administration of *iodide of potassium* in cases of lead poisoning; and great caution is necessary in the employment of this remedy in man for the first few days. At the moment when the metallic compounds fixed in the body become dissolved or transformed, the phenomena of acute poisoning may occur, caused by their liberation. So much is this the case, that the treatment may be supposed to be at first hurtful rather than beneficial. The patient should have beside him a graduated solution of the *iodide of potassium;* and should begin with a small dose (fifteen grains during the twenty-four hours), and afterwards increase or diminish it according to his pains and sensations (MELSENS). Dr. Fagge has also noticed that the administration of *iodide of potassium* to a person poisoned by lead, is sometimes followed by the development of a line in the gums which had previously been absent, and this when the patient was removed from the influence of lead.

Galvanism, in the form of Faradisation, ought to be used as a local stimulant to the nerves, with the precaution that its application is not to be continued too long each time. Ten or fifteen minutes, at three different periods of the day, or of every second day, and persevered in for not less than four weeks, will be found of great service (TODD, ALTHAUS). The beneficial influence will follow, although, in the commencement of the treatment, even a current of very high tension does not cause any movement whatever in the paralysed muscles. In such cases the beneficial influence seems attributable to the restoration of mobility to the molecules of nerve and muscle by an induced current, and which is necessary to enable them to be physiologically active. Severe shocks, especially in the commencement of the treatment, should therefore be carefully avoided, as by such the weakened excitability of nerve and muscle may be reduced, in place of being fostered and developed (ALTHAUS, l. c., pp. 112 and 119). It would be rational, however, to defer the application of galvanism till the lead has been completely eliminated.

MERCURIAL POISONING; TREMOR MERCURIALIS.

Definition.—*A series of morbid phenomena, especially peculiar tremors, with cachectic and cerebral symptoms, induced by the absorption of mercurial inhalation of vapours from mercury or its salts, or from the medicinal administration of the salts of mercury in continuous small doses.*

Pathology.—(*a.*) *Causation.*—Workers in preparing mercurial compounds, gilders and looking-glass makers, are those most liable to be affected.

Symptoms.—Various forms of cachexia, vertigo, sleeplessness, loss of memory, imperfect and unsteady action of the muscles, slight atonic convulsions, and delirium, epilepsy or apoplexy, may supervene. Salivation, ulceration of the mouth and gums, hæmorrhages and emaciation, are also the product of mercurial vapours. Excessive salivation and *mercurial stomatitis* may also result from repeated doses of the mercurial salts. There may also be fever with tenderness of the parotids and salivary glands. The tremors usually begin in the upper limbs, with slight movements at first, which increase to spasms or convulsions, increased by mental excitement. At last they become constant, the patient becomes helpless, with the speech and breathing affected.

The **Treatment** is similar to that already just described for the elimination of lead.

ERGOTISM.

LATIN EQ., *Morbus Cerealis;* FRENCH EQ., *Ergotisme;* GERMAN EQ., *Mutterkornbrand;* ITALIAN EQ., *Ergotismo.*

Definition.—*A train of morbid phenomena produced by the slow and cumulative action of a specific poison in a fungus peculiar to wheat and rye, and which gives rise to convulsions, gangrene of the extremities, and death, or to symptoms of ill-health.*

Pathology.—(*a.*) *Historical Notice.*—*Ergotism* is a disease very little known in this country; but it is not unfrequent that diseased, unripe, or damaged grain of any kind, and especially *rye*, has been observed to be injurious to the animal economy. *Wheat, rice,* and such like grains, are equally injurious when similarly unsound. The most frequent form of this unsoundness consists in the development of a fungus (the *Ergotœtia abortifaciens* or *Cockspur*) upon the grain, to which the name of *ergot* has been given. In this ergot fungus there is a large proportion of fixed oil (WIGGER). A morbid state is produced by various other poisonous fungi, such as the *Amanita muscaria* and *citruria*, the *Hypophyllum sanguineam;* and by plants, such as the *Lolium temulentum*, being mingled with the grain. A characteristic and specific result of the poison follows as a form of gangrene in parts remote from the source of circulation. It is a species of mortification not much seen in this country, but well-known and frequently observed in different parts of Europe, particularly in France, in some districts of which it has been repeatedly known to prevail as an endemic disease, where *rye* forms the principal food of the inhabitants. The *ergot* or *cockspur* in *rye* is apt to occur after rainy or moist seasons. The grains grow to a large size, acquire a black colour, and have a compact, horny consistence. The attention of the public was first called to the peculiar mortification which follows the use of diseased rye by M. Dodard, in the *Journal des Savans*, for 1676. He described the appearance of the affected parts, and mentions that fowls fed with the grain soon died. In 1694 its frequent occurrence is noticed at Sologne, and it prevailed amongst the patients of the Hôtel Dieu at Orleans. The upper and lower extremities of those suffering from the disease "grew as dry as touchwood, and as emaciated as Egyptian mummies." In 1710 it was again the subject of treatment in the Hôtel Dieu at Orleans, from its extreme prevalence in the neighbourhood. About fifty people, men and children, were admitted that season into the hospital. During thirty-three years the endemic appeared three or four times, and always in those rainy or moist seasons in which the rye contained a large proportion of the cockspur—more than a fourth. The mortification always began in the toes, and extended gradually along the foot and leg, till it might rise to the upper part of the thigh. In some patients the gangrened part came away of its own accord; in others it became necessary to assist nature by amputation. In some instances death succeeded to amputation, the disease having continued to extend to the trunk of the body. The rye of Sologne, in the year 1709, contained fully *one-fourth* part of the cockspur (*Royal Academy of Sciences at Paris*). Gangrene usually began in one or both feet, with pain, redness, and a sensation of heat, as burning as the fire. At the end of some days these symptoms ceased as quickly as they had come on, when the sensation of extreme heat was changed to cold. The part affected became black, like a piece of charcoal, and as dry as if it had been passed through the fire. A line of separation tended to form between the dead and the living parts, like that which appears in the separation of a slough produced by the application of the cautery. The disease prevailed in Switzerland in 1709 and 1716, and is described by Langius, of Lucerne. M. Garroud, a physician of Dauphiny, where the disease prevailed in 1709, makes some very important observations on the different symptoms apt to predominate in different individuals. The experiments and observations of Tessier show that a certain quantity is required to produce the specific effects, and that the action of the poison is cumulative.

The history of some cases of mortification of the limbs related by Dr. Charlton, Woolaston, in the *Philosophical Transactions* for 1762, shows that it may occur in this country from eating *wheat* diseased similarly to the *rye;* and the late Sir William Wilde, of Dublin, has recorded the occasional occurrence of such gangrene in Ireland.

Symptoms.—The gangrenous form of ergotism is ushered in by excessive lassitude, more or less protracted, accompanied with fever. The extremities become painful, cold, rigid, benumbed, almost insensible, and are with difficulty capable of movement. Severe internal pains of the limbs prevail (*acrodynia*), greatly aggravated by heat. It extends by degrees from the toes to the legs and thighs, and from the fingers to the arms and shoulders, when *sphacelus* supervenes. With the exception of slight febrile heat, the constitutional disturbance appears to be slight, and in this respect resembles scurvy. The early symptoms of a case of poisoning by bread containing *ergot* have been given in a family of six persons who partook of the bread. All of them suffered from the same symptoms —namely, dryness of the throat, epigastric oppression, nauseous taste, mucous and biliary vomiting, vertigo, stupor, and diarrhœa (*New Syden. Society Year-Book*, 1861). Seeing that symptoms vary greatly in severity, probably in proportion to the amount of diseased food taken, and the poisonous nature of the particular fungus which affects it, we may have the expression of some phenomena more fully than others—for example, *acrodynia*. Under this name Chomel described a painful affection of the wrists and ankles which prevailed in Paris in 1827, 1828, and 1829. It was then so prevalent among the soldiers in the barracks of Lourcine, that 560 men were affected out of 700; and in that of Comtille, 200 men out of 500. Since 1828 no case has been recorded in the barracks or military hospitals (PARKES). In 1859 M. Barudel observed three cases in the military prison at Lyons; and in spite of negative results of the examination of the quality of the food, Dr. Parkes is inclined to believe that the cause lay there, and probably in ergot of the flour (*Army Medical Department—Sanitary Report* for 1860, p. 358). The general train of symptoms produced by the use of diseased grain assumes two forms, the spasmodic or the gangrenous. The spasmodic form commences with a sense of tingling or itching in the feet, followed by cardialgia and similar tingling sensations in the hands and head. Violent contractions of the hands and feet follow, which affect each particular joint. The pain is said to resemble that of a dislocation. The sensations are of a bruising kind; and the body is bathed in copious sweats. The symptoms intermit during intervals of two or three days of a remission at one time. Drowsiness, giddiness, indistinctness of vision, and an irregular gait are constant phenomena. Coma and epileptic convulsions are apt to supervene, which generally indicate a fatal result. An enormous appetite accompanies this train of evils. Spots like those of purpura appear on the face, and the disease rarely abates before the third week.

Treatment.—Considerable differences of opinion prevail regarding the treatment of this *dietetic* disease. The cause, in the first instance, must be ascertained and removed; and to obviate the effects it has already produced, the constitutional treatment must be directed to improve the state of the blood. Tonics and stimulants are to be administered, after a free employment of evacuant remedies, to clear out the alimentary canal completely. The chlorates of potash and of soda, with antispasmodics, tonics, and narcotics, are especially indicated. *Camphor*, *musk*, *ammonia*, *capsicum*, may be particularly mentioned; and the strength of the patient is to be supported by light, nourishing, and wholesome food.

ALCOHOLISM—DELIRIUM TREMENS.

LATIN EQ., *Delirium Alcoholicum;* FRENCH EQ., *Delirium Tremens;* GERMAN EQ., *Delirium Tremens*—Syn., *Säuferwahn Sinn;* ITALIAN EQ., *Delirium Tremens.*

Definition.—*A train of morbid phenomena produced by the slow and cumulative action of alcohol in the various forms in which it is used as a drink. Delirium is one of the most prominent features of the morbid state, which is otherwise characterised by hallucinations, dread, tremors of the tendons and muscles of the hands and limbs, watchfulness, absence of sleep, great frequency of pulse. A thick, creamy fur loads the tongue, and a cool, humid, or perspiring surface prevails; while the patient gives forth a peculiar odour, of a saccharo-alcoholic description, more or less strong.*

Pathology.—(*a.*) *History.*—*Alcoholism,* in the form of *delirium tremens,* has only been recognised and described since the beginning of this century. The *Experimental Inquiry* of Dr. John Percy, in 1839, illustrating the physiological action of alcohol; an inquiry into the *Physiology of Temperance,* by Dr. Carpenter; the recent *Pathological Observations on the Bodies of Known Drunkards,* by Drs. Roesch and Francis Ogston (1855); and, lastly, an interesting review on the "Treatment of Delirium Tremens," in the *Brit. and For. Med.-Chir. Review* for October, 1859, are contributions which have placed on a more sure foundation our previous theoretical information regarding morbid states which follow the persistent use of alcohol. (See also p. 1137, Vol. I.)

(*b.*) *Causation.*—The term *alcoholism* is used to denote various symptoms of disease attending morbid processes of various kinds which are capable of being traced to the use of stimulants containing alcohol. The immediate effects of *intemperance* in the use of alcoholic fluids, the nature of *delirium tremens,* and of *spontaneous combustion,* may be embraced under the general designation of *alcoholism.* This term is used in the sense analogous to that in which we use the terms *mercurialism, ergotism, narcotism,* and the like—the agents inducing these specific states acting after the manner of a cumulative poison. The progress of modern science has distinctly demonstrated the poisonous action of alcohol; and an account of the nature of delirium tremens, as well as the grounds on which its treatment must be based, are now alike founded on this knowledge. Tiedemann and Gmelin, in 1820, and Majendie, in 1823, detected alcohol by its odour in the blood. The fluid found in the ventricles of the brain had also been observed to have the smell, the taste, and the inflammability of gin (Sir A. CARLISLE). In 1828 it was theoretically advanced by Leovèille that *delirium tremens* consisted in an exalted state of the vital powers of the brain, excited by molecules saturated with alcohol absorbed from the surface of the stomach and bowels, and carried into the current of the circulation. Now it is a matter of fact, determined by direct experiment, as well as by casual observation, that alcohol is absorbed directly into the circulation, and is capable of acting as a direct poison upon the nervous tissue through which the infected blood circulates. Alcohol has been found in the blood, in the urine, in the bile, in the fluid of the serous membranes, in the brain matter, and in the liver (PERCY, OGSTON). Its odours can be easily detected in the breath, and the habitual immoderate drinker exhales a distinct alcoholic and saccharine odour more or less strong. His clothes at last acquire a spirituous aroma, every part of his body being long thoroughly imbued with alcohol (CRAIGIE). This odour is generally so well expressed in cases of *delirium tremens* that a place has been given to a statement of the fact amongst the characters embodied in the definition. Dr. Percy's experiments directly support these statements, and prove at the same time the great rapidity with which alcohol passes into the current of the circulation. He injected strong alcohol into the stomachs of dogs, and within two minutes after completing the injection, their respiratory and cardiac movements ceased; the stomach was found nearly empty after death, whilst the blood was highly charged with alcohol. I once had an opportunity of examining the body of a person who had for many years been in the habit of drinking daily a large quantity of brandy. He died of *typhoid asthenia*, with characteristic degeneration of nearly every important organ of the body and of the blood-vessels. The fluid collected from the cavities of the brain, consisting of serum and some blood, contained 2·6 per cent. by volume, and 2·1 per cent. by weight, of alcohol. This quantitative analysis was made for me by my friend Professor De Chaumont, with Giessler's vaporimeter.

(*c.*) *Morbid Anatomy.*—The pernicious effects of the continuous use of alcoholic stimuli on the organs and tissues of the body have been deduced from a careful study of the morbid appearances, of *a chronic kind*, met with in the bodies of individuals known to have lived intemperate lives, and who *had perished suddenly* from the effects of accident, suicide, or homicide, and while apparently in ordinary health and activity. The extent of the chronic changes in the various organs of these individuals is found to have been far in excess of what could have been reasonably looked for in a like number of persons of the same age, and of temperate habits, suddenly cut off while apparently in average health and vigour. The *cumulative* effects of long-continued intemperance have been clearly proved by Dr. Ogston's observations. The following results of his *post-mortem* inspections, on the whole, support the conclusions which have been arrived at on *theoretical grounds* as to the injurious effects of alcohol in excess:—(1.) *The nervous centres* present the greatest amount of morbid change, the morbid appearances within the head extending over 92 per cent. of those examined. By this observation the theoretical remarks of Leovèille, Craigie, and Carpenter are clearly established. (2.) *The changes in the respiratory organs* succeed in frequency those of the nervous centres, yielding a percentage of 63·24 of those examined. (3.) *Morbid changes* in the liver are next in order of frequency, and are due to enlargement, granular degeneration, the nutmeg-like congestion, and, lastly, the fatty state. (4.) Next to the changes in the liver come those *in the heart and large arteries.* (5.) Next are those of the kidneys. (6.) Least frequent of all are morbid changes in the *alimentary canal.*

Two orders of changes may be observed to result from intemperance in the use of alcoholic fluids—namely, one which must have taken some considerable time before they could be completed; another of shorter duration, which probably are more closely connected with the immediate symptoms which precede the fatal event. The abnormal changes in the cranium, the substance of the brain, its convolutions and cerebral ventricles, all indicate the prolonged action of a morbid poison. The prolonged action of the alcoholic poison on the cranial contents is to produce induration of the cerebral, cerebellar, and spinal substance in by far the largest number of cases, coincident with an increased amount of subarachnoid serum; while the fatty degeneration of the small arteries leads to *atrophy* of the convolutions and *œdema* of the brain.

When spirituous liquors are introduced into the stomach they tend to coagulate, in the first instance, all albuminous articles of food or fluid with which they come in contact. The mere coagulation of the albuminous articles of food and fluid is very different from that effected by the gastric fluids, and tends to render the articles more difficult of solution by the gastric juice. As an irritant, they stimulate the glandular secretions from the mucous membrane, and ultimately lead to permanent congestion of the vessels, to spurious melanotic deposit in the mucous tissue, and to thickening of the gastric substance. By the veins and absorbents of the stomach the alcohol mixes with the blood, and immediately acts as a stimulant to all the viscera with which it is brought in contact. The functions of the brain are at once stimulated, and ideas follow in more rapid succession; the liver is excited to secrete an excess of sugar, by the immediate action of the stimulant on its tissue (Drs. HARLEY and BERNARD). The flow of urine is excited in a similar manner. In these effects it is impossible not to recognise the operation of an agent most pernicious in its ultimate results, when taken in habitual excess; but most valuable as a remedial agent, when its action is understood and appreciated (consult Dr. ANSTIE'S valuable work on *Stimulants and Narcotics: their Mutual Relations*).

In these facts it is impossible not to recognise that alcohol being absorbed, a double series of morbid results ensue. It acts as a depressant and narcotic. On the one hand, a train of phenomena is induced, partly of a *chemical* nature and partly *physiological* or *vital*. The general nutrition of the body suffers; and a bad state of health is at last induced, of a peculiar kind, sometimes described as the "*drunkard's cachexia* or *dyscrasia*." This state of the system is characterised by positive irritation and depression, which soon succeed to the intemperate use of alcohol, and which is manifested in a variety of ways, sometimes by an unnatural voracious appetite; but those who over-indulge in the use of alcohol subsequently suffer a total disrelish for food. They become unable to eat, and dyspeptic symptoms of various kinds betray the irritable state of the alimentary canal, such as stomach-ache, the frequent generation of gases, water-brash, heart-burn, squeamishness, diarrhœa, vomiting, sometimes streaked with blood, and palpitations of the heart. A constipated condition of the intestines, attended with deficiency in the power to expel their contents, is very soon established, and sometimes ascribed to the deficient secretion of bile, which is known not to be secreted in due quantity; and there is every reason to believe, although the fact is not proved, that its quality is deteriorated. Its functional agency on the food and fluids in the intestines is therefore diminished.

If we follow the course of alcoholic absorption through the vascular and pulmonary system, it is found unquestionably to *retard* the motion of the blood, while it produces a temporary increase in the action of the heart, and a congestion of the whole system of the pulmonary capillary vessels. Respiration is thus, in the first instance, rendered from four to six times more frequent per minute than it otherwise is; and various symptoms of accumulation of blood within the chest, and pulmonary congestion especially, are apt to occur. A short, tickling cough is a most constant phenomenon. Dr. Craigie remarks that all the spirit-drinkers whom he has ever seen or known have been either subject to chronic cough or dyspnœa, or have laboured under chronic, dry bronchial disorder, with asthma. That the use of spirituous liquors ultimately retards the motion of the blood in the vessels is known by experiments on the lower animals, and by pathological observations. Thus far we have a morbid condition induced which is highly favourable to the accumulation of fat in the blood, and such an accumulation has been proved to take place. It has been shown by Becquerel and Rodier that fat increases in the blood, in most acute diseases, when the biliary secretion is retarded, and when a scanty amount of food is taken. These are the very conditions which obtain in alcoholism. It is in cases of undoubted drunkenness that fats have occurred in the blood in such obvious quantity as to leave no doubt of its presence. In such cases a milky character is imparted to the serum—a condition which may be recognised by simple inspection; while a microscopic examination and treatment with ether will establish the diagnosis, and distinguish the fat from colourless blood-corpuscles, or from molecules of albumen. Slighter degrees of this condition are altogether physiological, and are met with during the period of digestion, after eating substances rich in fat (A. BUCHANAN, VOGEL). But the extreme degrees of this condition have been specially met with in drunkards. J. Frank regards the white and fatty blood as having its origin in the abuse of alcoholic drinks (*Hannoversche Annalen*, 1847, p. 283, quoted by VOGEL). Dr. Adams (*Trans. Med. and Phys. Society of Calcutta*) mentions the case of a sergeant at Fort-William who went to bed drunk, and was found dead in the morning. The vessels of the brain were greatly distended with blood, and oil was seen floating in it. Rayer relates the case of a man who, after drinking largely of punch, destroyed himself by the fumes of charcoal. The blood and the urine contained globules of oil. Serules, of Strasbourg, records similar phenomena. Thus, it is shown by abundant testimony that the blood becomes surcharged with unchanged and unused material, and contains at least thirty per cent. more of carbon than in the normal state. The order of events by which this state comes about is somewhat as follows :—Alcohol is directly absorbed by the blood-vessels without undergoing any change or decomposition. Part of it is eliminated very slowly as alcohol by the lungs, by the liver, and by the kidneys; but appears to tarry in largest amount in the liver and in the brain (PARKES). Another portion is decomposed. Its hydrogen enters into combination with oxygen to form water, which, with acetic acid, having been produced, carbonic acid and water are formed. Oxygen is thus diverted from its proper function, the exhalation of carbonic acid at the lungs is diminished both absolutely and relatively, and less urea is excreted by the kidneys than is consistent with health; but the pulmonary aqueous vapour is not lessened (BOCKER and HAMMOND, quoted by PARKES). The water of the urine is diminished, the chlorine is greatly lessened, as well as the acids and bases. All the evidence, therefore, points to the effect of alcohol as causing the retention of substances which ought to be eliminated; and this retention of the effete matter is still more intensified by the action of alcohol increasing for a limited time the frequency of functional acts, followed by a corresponding depression. In this way impaired health is soon brought about, tending to wasting of the tissues generally; and so long as any alcohol remains in the blood *as alcohol,* a certain toxic or poisonous effect continues to be produced upon the nervous system, through which the poisoned blood circulates. If a constant supply of the alcohol is kept up, the phenomena of *alcoholism* become chronic or persistent; and acute paroxysms, generally in the form of *delirium tremens,* supervene, which is at once the most common and the most prominent evidence of *alcoholism.* In other instances the degeneration of several vital organs generally may become so excessive that death follows by *asthenia,* or with typhoid phenomena ending in coma. When mixed with blood out of the body, spirituous liquors cause more or less coagulation according to their strength and concentration; and when applied to the blood-vessels in the transparent parts of animals, they can be seen to produce the same effects. The congestion that constantly exists in the mucous membranes of the lungs and stomach is evidence of the retarded motion of the blood. The fact that hæmorrhoidal swellings are always aggravated by the use of alcoholic fluids is the result of retarded motion of the blood in the hæmorrhoidal vessels. When death occurs from poisonous doses, either in animals or in man, although the dose is at first followed by increased frequency of the pulse, yet in a short time the pulse becomes rapid and small, while the extremities become cold, and the power of generating heat is suspended in proportion as the blood progresses slowly and more slowly through the pulmonary capillaries. These effects upon the lungs must be regarded as of a toxic kind; and this specific toxic action is not less obvious on the brain. Its nerve-substance becomes poisonously affected—a condition which seems to constitute one of the most necessary antecedents in the causation of *delirium tremens.* The effects produced on the *medulla oblongata* tend to sustain this toxic effect upon the lungs. The brain and the lungs in this respect act and react on each other. Death may ensue in various ways, but generally by coma, asphyxia, syncope, exhaustion, or epilepsy.

Symptoms.—In the case of habitual spirit-drinkers there is thus constantly going on a temporary quickened motion of the blood through the vessels, especially manifested by cerebral, thoracic, and hæmorrhoidal phenomena, followed by a corresponding depression and tendency to stagnation of the blood in the capillaries of all the internal organs, especially in the membranes and the lax areolar tissues of dependent parts. The essential nature of *delirium tremens* is associated with the loss of cerebral power, evinced especially in the want of control over thoughts, emotions, and muscular action, consequent on the direct influence of the alcoholic poison. Disturbances of function, depression, and debility are the attendant phenomena. The feeble but rapid action of the heart, the tremulous undecided action of the muscles, and the terror-stricken and agitated mental state, betoken the depressed condition of the living functions. The amount of phosphates in the urine, as determined by Dr. Bence Jones, is diminished, while the proportion of the sulphates and of the urea is greatly increased. The disease has been variously named the *brain fever of drunkards, dipsomania,* or *delirium tremens,* by which latter name it is more frequently known and described in this country. The disease has been said to become developed under two sets of circumstances—namely, according as the patient has been continuing his potations, or after he has suddenly abandoned them. But its occurrence under this latter circumstance is now known to be nothing more than a coincidence; and of all the errors in popular acceptation connected with the malady, none is greater than that which affirms the exciting cause of a paroxysm of *delirium tremens* to be a sudden stopping or withdrawal of the accustomed quantity of stimulants. The ceasing to drink depends on the commencement of the disease (GAIRDNER). There are some who are constantly taking small quantities of spirits, and who, although they never get completely intoxicated, yet sometimes exceed considerably their accustomed allowance, and continue to do so for some time. The symptoms of *delirium tremens* generally appear from the second to the eighth or ninth day after a protracted debauch, and are by some pathologists divided into three stages. The first stage is marked by a peculiar slowness of the pulse, by coldness and clamminess of the hands and feet, by general debility, by nausea and vomiting in the morning, and by frightful dreams at night. Very moderate exertion of body causes the patient to perspire profusely, and anything which suddenly affects his mind throws him into a tremulous agitation. The tongue is tremulous and furred, the hands shake, there is great depression of spirits, he sighs frequently, is anxious about his affairs, and is either restless or watchful. These symptoms last from twenty-four to forty-eight hours. The second stage is indicated by a hurried and anxious manner, by great excitability of temper, by a small, accelerated pulse; some heat, perhaps, of the surface of the trunk, but accompanied with coldness and clamminess of the extremities. The tongue may be clean, but is often brown and dry, and the patient delirious, suffering from various mental illusions and alienations. In general the delirium is melancholy, and has reference to his usual occupation and habits, or to some difficulty in his domestic affairs. He sometimes sees flames or hears voices talking to him, or as soon as he shuts his eyes he sees people passing under the bed-clothes. He sees objects and sights in situations in which they are not, and which have no real existence; or betrays the most dreadful alarm at hideous objects which he imagines are threatening him with immediate destruction. Restless and sleepless, he moves his trembling hands horizontally over the bed-clothes, as if feeling for something. In general he is harmless and easily controlled; but in some instances he is violent, mischievous, and requires to be restrained. This stage lasts from three or four days to a week, when the third stage commences by the patient falling into a sound sleep and gradually recovering, or by a fatal collapse coming on, which finally and shortly closes the scene. Without reference to stages of the disease, there is always more or less derangement in several other functions besides the brain. The patient is generally void of appetite; or he may be squeamish, and vomit at intervals. Sometimes he is thirsty, and calls loudly for liquor of various kinds; but he may be indifferent to the sensation of thirst. In several instances great aversion, and even dread, of all food and drink has been evinced; and it has been impossible to persuade the patient to partake of either. The tongue is at first covered with moist white, grey, or slate-coloured fur, and when protruded it is tremulous. The bowels are constipated, and less sensible to the action of medicine than in the state of health. When they are acted upon by remedies, the discharges are very dark-coloured, the first generally consistent, the later liquid, dark, and offensive. There are generally fulness and distension, and not unfrequently tenderness and pain in the epigastric, umbilical, and right hypochondriac regions. *The pulse* varies from 96 to 110 or 120, sometimes 130; and though sometimes small and oppressed, is often full, voluminous, and throbbing. The carotid and temporal arteries beat most violently; those of the wrist less forcibly; and the anterior and posterior tibial arteries pulsate feebly enough. The action of the heart is in general unusually violent, and the cardiac beat is diffused over the whole chest. The respiration is occasionally panting and irregular, but not otherwise morbid. Restlessness is extreme. The patient is in constant agitation of mind and body; speaks almost incessantly, yet seldom adheres above a minute to one subject, and is constantly changing place and looking for some new object. He cannot sleep, and dreads to be left alone, from the apprehension of spectral visitations. With this restlessness the upper extremities, and especially the hands, are in constant tremulous motion, such that they cannot be kept for two seconds in the same position, nor can the pulse in many cases be accurately numbered at the wrist. Though this tremulous motion of the arms, wrists, and hands is very general, it is not constant; and instances of the mental disorder, agitation, spectral illusions, and sleeplessness have been observed to take place without any such kind of tremors. They are very rarely, almost never, seen in the young or middle-aged to any very great extent, or those whose muscular motions are not otherwise unsteady; and are seldom well marked in first attacks of the disease. Such tremors are principally observed in the cases of confirmed dram-drinkers, whose movements are always unsteady in the morning and early part of the day, until they take a certain proportion of their habitual stimulus (CRAIGIE). After symptoms of restlessness and sleeplessness have continued for three or four days, the patient may fall into a sound, unbroken slumber, which lasts for some hours. The paroxysm thus works itself out in a definite time in uncomplicated cases; and sleep occurs simply as the natural and spontaneously favourable termination of the disorder. It occurs as the *result* of the paroxysm having run its course, and of the nervous system having lapsed into an improved condition: it must not be regarded as the *cause* of those favourable conditions (Reviewer in *Brit. and For. Med.-Chir. Review*, l. c.) On the other hand, the symptoms may pass into a state of *coma vigil*, with constant muttering delirium, *subsultus tendinum*, and picking of the bed-clothes, the pupils become contracted, the muscles of the face and jaw are moved incessantly, and death may ensue from prolonged coma or convulsions.

Diagnosis.—*Delirium tremens* is to be distinguished from typhus fever, and from paralysis agitans, by the previous history of the case, and by the symptoms.

Prognosis.—The duration of the paroxysm varies from three, four, or seven days, and a favourable or fatal termination may be looked for in from three to five days. It is hardly determined what is the proportion of recoveries to deaths; but unquestionably three persons out of four do well. A paper in *The Indian Annals of Medical Science* for 1855, by Dr. Macpherson, notices the great discrepancy in the statistics of writers on *delirium tremens,* with regard to its frequency in both sexes, and to the mortality of the disease. Calmeil states the rate of mortality at 5 per cent., Bougard at 19 per cent. The most accurate records to be got are those regarding the British troops at different stations. The late Sir Alexander Tulloch, in his report for 1853, gives the following percentages, stated in the order of the greatest mortality, namely—Great Britain, Infantry, 17·6; Cavalry, 13·8; Bermuda, 15·0; Gibraltar, 13·6; Nova Scotia, 9·1; Malta, 8·8; Canada, 7·94.

A return of admissions and deaths from *delirium tremens* and *ebrietas* in the General Hospital in Calcutta, from 1848 to 1852, and another of admissions and deaths from the same causes in the Medical College Hospital, during 1851, 1852, and 1853, give some important results. *Delirium tremens* occurs in women and men in the proportion of one to twenty-five; but this difference is due to the difference of habits rather than of sex. In regard to age the ratio is as follows:—

	Cases.	Deaths.	Per cent. of Deaths.
Ages from 20 to 25, ...	34	4	9·1
,, 25 to 30, ...	66	16	24·2
,, 30 to 35, ...	48	11	22·9
,, 35 to 40, ...	76	7	9·2
,, 40 to 45, ...	62	6	9·6
,, 45 to 50, ...	23	4	17·3
,, 50 to 60, ...	7	—	—
,, 60 to 65, ...	5	1	—

The greatest mortality is between the ages of twenty-five and forty. There is no evidence to show that the season of the year exerts a definite influence on the occurrence of the disease, whereas the mortality varies with the temperature, it being more than double in the eight hot months as compared with the four cold months.

The cause of death is by exhaustion (often with coma); or by coma only; or by fits, sometimes called epileptic, but probably apoplectic.

Treatment.—The indications are,—(1.) The elimination of the poison; (2.) the sustenance of the patient during this period. The two most fatal errors which can be committed in the treatment of *delirium tremens* are to *bleed* the patient or to give him *opiates.* The greatest number of cases of those treated by opiates are apt to terminate by convulsions and coma (MOREHEAD, PEDDIE, LAW, CAHILL, LAYCOCK). If it be true, also, that opium and alcoholic stimulants singly are to be deprecated in the treatment of *delirium tremens, à fortiori* in their combination there is a twofold danger. Alike in tropical as in temperate regions it is a course of treatment attended with much hazard, and if systematically followed, is certain of leading to unfortunate results (see MOREHEAD'S *Researches on*

Diseases in India; also, "Notes on Treatment of Delirium Tremens," by W. Hanbury, 33rd Regiment, in *Madras Quarterly Journal,* July, 1863). The strength must be supported by diet of the most nutritious kind, in a fluid and mild form, such as yolk of eggs, soups, and the like. Food should be given in small quantities and often. Beef-tea, spiced soup, and egg-flip, are each to be commended at different periods of the day. If the patient continues to digest food, the danger is much diminished, which in the first instance is from exhaustion. Careful nursing is above all things necessary, so that protection from all sources of danger may be adequate and the food adapted to the state of the digestion, which is always feeble. The disease must be treated as one spontaneously curable; not by withholding remedies, but by using them in strict subordination to good nursing and carefully adjusted diet and regimen (Ware, Hood, Peddie, Laycock, W. T. Gairdner). Active specifics for *delirium tremens* appear to be founded on the idea that the disease is one nominally of high mortality. Hence the enormous doses of *digitalis* (Jones) and of *Cayenne pepper* (Kinnear and Lyons of Dublin); but before resorting to the use of such remedies as *digitalis,* it will at least be judicious practice to adopt such means as are calculated to restore the powers of nature—namely, nutrients and rest; while the stimulus of such a spice as *Cayenne pepper,* given in the soup, on the atonic stomach, will have a favourable influence on absorption. Under the care of my friend, Dr. Lyons, of Dublin, numerous cases of *delirium tremens* have rapidly yielded to *capsicum,* in doses of xx. to xxx. grains, in the form of a bolus. I have given it in numerous cases and in all forms of chronic alcoholism. Its influence in soothing the patient and in securing quiet sleep has been certainly remarkable. By some reviewers this influence has been questioned; but the evidence of its good effects are daily accumulating. Dr. Ringer, in an able article in the *Brit. Med. Journ.,* in 1871, advocates the use of capsicum, "given in doses of the tincture (five to ten drops) in a little syrup of orange peel. Or the powder may be formed into five-grain pills with *extract of gentian*—one to be taken every four hours till quietude is obtained; or, twenty grains in a single dose may be taken before meals, or whenever depression or craving for alcohol arises." It induces sleep in the early stages of delirium tremens. It obviates the morning vomiting, removes the sinking at the pit of the stomach, the intense craving for stimulants, and promotes appetite and digestion. Dr. Wilks prescribes it with *nitric acid* and *nux vomica.* Dr. Lauder Brunton has given a great deal of attention to this subject, and, according to his experience, a combination of *iron* and *nux vomica* (say fifteen minims of the tincture of the *perchloride* and ten of *tincture of nux vomica*) is one of the most efficacious remedies for the tremors of chronic alcoholism. If the stomach be deranged, as it very frequently is, the indigestion should be treated previously to the administration of *iron,* by giving ten grains of *subnitrate* or *carbonate of bismuth* with ten of *magnesia,* suspended with *gum tragacanth.* If there be any tendency to sleeplessness, or if the tremors should not rapidly disappear under the use of the iron and nux vomica alone, thirty or forty grains of *bromide of potassium* should be given at night. The chalybeate mixture already mentioned, either alone or with the addition of five or ten minims of *tincture of capsicum,* tends to alleviate the craving for drink. Another mixture recommended for this purpose consists of *sulphate of iron*, *magnesia*, and *oil of cloves;* a third consists of *carbonate of ammonia* in *infusion of gentian*, to be taken whenever the craving is felt. Should the craving come on at intervals of several weeks, with complete intermissions between, the case should be treated like one of *epilepsy*, by the administration of *bromide of potassium.*

Chloral is now a most important and valuable addition to our remedial agents in this disease. It produces sound and refreshing sleep, followed by relief to all the symptoms. It now seems to serve all the purposes intended by opium.

The late Dr. Jones, of Jersey, gave as much as half-ounce doses of the *tincture of digitalis* till three doses had been taken; and then, if excitement were not subdued, nor sleep induced, two fluid drachms were repeated every three or four hours (*Med. Times and Gazette*, Sept. 29, 1860). But Dr. Ringer justly cautions against such excessive doses, and records two instances in which the patient suddenly fell back dead. The disease no doubt proves suddenly fatal, sometimes independently of any remedy; but the powers of *digitalis* in a tincture are much too uncertain to be relied upon as safe in such enormous doses. *Bromide of potassium* has been found of great use in calming the excitement of delirium and procuring sleep, especially in the earlier stages of the disease, before the delirium has become furious. The dose may be xx. or xxx. grains every two hours. Its good effects are, however, very uncertain when given alone; less so when combined as suggested by Dr. Lauder Brunton (RINGER).

In some cases purgative remedies are indicated from the first. "These cases are known by the flushed, bloated appearance, the very foul tongue, the *mawkish*, peculiar odour of the breath, the fetid discharges from the bowels, and the history of a recent surfeit of eating as well as drinking" (W. T. GAIRDNER, *Clinical Medicine*, p. 271).

Opium may be administered with safety and advantage only in protracted cases, provided the quantity given in twenty-four hours is never allowed to exceed the full dose which would be considered safe for a healthy person of the age and sex of the patient. Where it appears to be indicated in protracted cases, it ought to be pushed as rapidly as possible for two or three doses, while its effects are carefully watched. Its use must be discontinued for at least a good many hours as soon as a full maximum amount of ʒiss. to ʒii. of the tincture, in all, has been reached, or even sooner if the pupils have *become contracted during its use.* This remedy should always be given in the fluid form, otherwise it is apt to accumulate in the bowels, owing to the weakened state of the digestion; and a laxative, or even a purgative, should be alternated with opium, followed by a bitter tonic, which always operates favourably in lingering cases of nervous and dyspeptic exhaustion. Narcotics are only safe in *delirium tremens* when they are given with the object of aiding and seconding the natural cure of the disease, employed in moderate doses, and given only at the later stages. The *heroic* use of them, as heretofore too often advocated by most eminent physicians, is now recognised as a treatment which merely substitutes *narcotic* poisoning for *alcoholism* or *delirium tremens.*

Envelopment in a wet sheet, and then a blanket round the wet sheet, is recommended by Dr. Wilks and Niemeyer as a valuable sedative appliance. As soon as hot vapour so generated surrounds the patient, he falls into a quiet sleep (*Med. Times*, Sept. 19, 1868).

PARALYSIS OF THE LOWER LIMBS PRODUCED BY THE USE OF *LATHYRUS SATIVUS.*

LATIN EQ., *Paralysis ex Lathyro;* FRENCH EQ., *Paralysie causée par Lathyrus*; GERMAN EQ., *Lähmung durch Lathyrus;* ITALIAN EQ., *Paralisi da Lathyruz.*

Definition.—*A specific form of paralysis, commencing more or less suddenly by stiffness in the legs about the knees, weakness of the loins, unsteadiness of gait, till at last paralysis becomes confirmed, and the feet are so dragged upon the ground that, with the feet tending to turn inwards, and the knees bent, the great toe scrapes the ground. The disease occurs from the use of the flour of the beans of the* Lathyrus sativus; *and ill-health is apt to occur when the flour of this vetch exceeds one-twelfth part; and if the proportion used as food amounts to one-third, the consequences may be serious.*

Pathology.—Attention has been called to this form of paralysis by Dr. Irving, Civil Surgeon of Allahabad, as extensively prevalent in part of that district. Village after village in Pergunnah Barra, on the right bank of the Jumna, contain many cripples and lame persons, whose paralysis is known by the natives to be due to their having lived too much upon bread made from the flour of the *Lathyrus sativus.* They are well aware that eating the flour of this vetch has a peculiar effect on the lower part of the spine (IRVING). It is found that a proportion of 3·19 per cent. of the population are rendered useless by this disease (COURT and IRVING in *Indian Annals* for 1857); but different villages are affected in different degrees and proportions. The country where it prevails has the appearance of a vast swamp, in which the *L. sativus* is the vetch most extensively cultivated as an article of food. It is common enough in most parts of India, and is frequently sown with wheat or barley, and cut down green as fodder for cattle. The ripe bean is used as food when made into flour, but is generally used with wheat or barley flour; and it is only when it exceeds *one-twelfth* part that it is injurious; and when it exceeds *one-third,* the specific paralysis sets in. Wheat flour will not grow in the district, therefore the natives are in a great measure left to feed upon this deleterious bean, and suffer in consequence. This form of paralysis is also known in Thibet; and even in Europe it has been known to follow the use of the *L. sativus* as an article of food. Other species of the same genus are occasionally known to render bread poisonous (DON, TAYLOR, LOUDON). Cattle, horses, and birds, when fed on the beans, become paralysed (SLEEMAN, IRVING). The use of bread made from the flour of the *L. cicera* has been known to establish complete paralysis of the lower extremities in a young and healthy man in a few weeks. Six or seven individuals of the same family, who had been in the habit of eating such bread, suffered more or less from similar symptoms, and one died (VILMORIN, *Ann. d'Hyg.*, Avril, 1847, p. 469; TAYLOR, *On Poisons*, p. 536). Further accounts of this vetch as a cause of paralysis may be found in *Indian Annals of Medical Science*, Vol. VII., 1861, by the late Dr. Kinloch Kirk, p. 144; also by Dr. Irving, pp. 127 and 501. An incidental reference is also made in Thomson's *Travels in the Western Himalaya and Tibet*, p. 391, footnote.

Symptoms.—The paralysis is observed most frequently during the rainy season in India—cold and wet being exciting causes, so that the first lameness may be a mixture of palsy and rheumatism. Men who had gone to bed quite well awoke in the morning feeling their legs stiff, especially at the knees, their loins weak, and their gait unsteady. Fever does not seem to attend the accession of the more obvious phenomena; but pain gets worse, and eventually the lower limbs become quite paralysed. The patient walks with difficulty, the toes turn inwards, the legs waste, and the great toe-nail scrapes the ground, till, in persons who go barefooted, the nail has been known to get rubbed down to the quick. Males are more often affected than females; and the *Ryots* are more liable to the disease than the *Zemindars*.

Treatment.—Some cases seem to have been benefited by generous diet, tonics, the use of strychnine, and of blisters to the loins; but nothing is known definitely on the subject; nor have we any records of the morbid state of the spinal marrow in cases that have proved fatal. Of course, the food must be obtained free from all poisonous elements.

THE

SCIENCE AND PRACTICE OF MEDICINE.

PART IV.

MEDICAL GEOGRAPHY; OR, THE GEOGRAPHICAL DISTRIBUTION OF HEALTH AND DISEASE OVER THE GLOBE.*

CHAPTER I.

SCOPE AND AIM OF THIS BRANCH OF SCIENCE.

This department of the Science of Medicine treats of the manner in which diseases are distributed over the world, or are confined to certain districts, and endeavours to investigate the conditions under which diseases are so distributed or limited. The subject embraces a consideration of topics which constitute the basis of *Hygiology*, which are of the greatest importance to Practical Medicine, of the utmost interest to Science, and of inestimable value in Political Economy. It embraces the medical application of the facts of *physical geography*, combined with those of *vital statistics;* and it has been variously named Medical Geography, or Noso-Geography.

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* Our knowledge on this subject is as yet only beginning to assume a shape; and the limits of this text-book merely permit the most faint outline to be given. To Alexander Keith Johnston, F.R.S.E., the medical profession in this country is indebted for bringing the subject prominently forward, in a communication to the Epidemiological Society of London, published in their *Transactions* for 1856, p. 25, and also in his *Physical Atlas of Natural Phenomena*, where his observations at p. 117 are illustrated by a map. "No scholar out of the domain of medicine has contributed documents more valuable than these to medical literature." That map Mr. Johnston has reduced to a scale suited to this handbook, and thus liberally permits me to use it in illustration. The most important works or monographs which have been published on this subject are Mühry's *Outlines of Noso-Geography*, in two volumes; and Boudin's *Traité de Géographie et de Statistique Médicales, et des Maladies Endémiques*, Paris, 1857, 2 vols. 8vo. A paper on Acclimation by Dr. J. C. Nott, in a work entitled *The Indigenous Races of the Earth;* Sir Alexander Tulloch's *Army Statistics;* Marshall's "Sketch of the Geographical Distribution of Diseases," in the *Edinburgh Medical and Surgical Journal*, Vol. XXXVIII., p. 330, and Vol. XLIV., p. 28; Dr. A. S. Thomson's *Thesis on the Influence of Climate on the Health and Mortality of the Inhabitants of the different Regions of the Globe*, Edinburgh, 1835; and Sir Ranald Martin's work on *The Influence of Tropical Climate*, are the sources from which the outline here given has been compiled.

Geographical Distribution of Disease-Realms.—As the physiological conditions of plants and animals vary according to different degrees of latitude (or rather with the different lines of equal temperature and moisture north and south of the equator), so do the pathological characters of diseases differ; and races of men are influenced as to health in proportion as they migrate from the land of their birth. It is the ascertained facts in meteorology and climatology of our globe which will help to explain the geographical limits of particular diseases, and their regulated distribution according to atmospheric temperature and moisture, the density and electricity of the air, and the vegetation with which they are surrounded. Such causes determine some of the laws by which diseases may be geographically distributed; but other concurrent causes must be taken into account in considering the special diseases of countries. For example, topographical situation, geological nature and elevation of the soil, and state of the vegetation; in short, *physical climate*, generally and properly so called, combined with the habits of the people, their attention to personal hygiene and general sanitary arrangements, all concur to stamp the diseases of certain countries with a special character, and to facilitate or retard their propagation. There are facts which show that certain diseases are so completely under the influence of temperature that they are susceptible of being arranged systematically in zones of geographical distribution. The diseases susceptible of being thus classified are those of a communicable type, which require a certain concurrence of physical conditions for their prevalence and propagation. They are chiefly *yellow fever*, *plague*, *typhus fever*, *typhoid fever*, and *cholera*. The geographical distribution of these diseases into zones, north and south of the equator, appears to be reglated in a great measure by relative degrees of temperature and humidity in the several places where they prevail, in America, Asia, Africa, and Europe. Such diseases, whose realms are bounded in a great measure by isothermal lines, are not only associated with locality, with characteristic vegetation, with heat and humidity, but they follow the physiological habits of the several animal inhabitants of the different latitudes. *Malarious fevers*, *yellow fever*, *plague*, *typhus and typhoid fevers*, have particular climates or zones where each predominates, and beyond the limits of which the disease is rarely propagated when imported, unless change of seasons gives rise to a state of climate analogous to that in which the specific disease is known to flourish.

Isothermic Zones bear a prominent part in relation to the geographical distribution of diseases. They connect the different places on the earth which have the same mean temperature, which Humboldt was the first to indicate. *Insect-realms* have been similarly indicated by Latreille. The late Professor Edward Forbes described *homozoic belts* of marine life. Cuvier, Blumenbach, Morton, Latham, Prichard, and others, have indicated *homoicephalic zones* or *realms of men;* and, following out these ideas, we have *realms of disease* defined by Dr. Mühry and Mr. Keith Johnston.

Realms of Disease and Description of the Map.—These zones, belts, or realms of particular types of disease thus marked out on the globe, are intimately associated with temperature, and may be generally indicated by the regions of the *tropical*, *temperate*, and *polar* zones, distinguished on the map by the respective colours of *brown*, *green*, and *blue*.

I. The northern limit of the tropical zone unites with the southern limits of the temperate zones, and the lines of union of the two colours on the map (green and brown) indicate Humboldt's mean annual isothermal line of 77° Fahr. or 19° Reaumur. It passes through Cuba and Florida in America; skirts the Cape de Verd Islands to Africa, where, extending beyond the usual limits of the tropics, it passes the northern part of the great desert (Sahara) below Algiers, runs through Egypt, Northern Arabia, and Persia, into China, where it is lost in the Pacific Ocean, below the limits of the northern tropic. The limiting line of this zone ascends somewhat in summer, when the sun is north of the equator; and descends again in winter, when the sun is to the south of it. To the south of the equator the same *isothermal line* (77°) marks the southern limits of the *tropical disease-realm*, where it joins with the northern limits of the *south temperate* zone. It crosses South America near the Amazon district, and approaching southwards in Africa towards the Cape, crosses over and embraces the northern half of Australia. This is the REALM OF TROPICAL DISEASES, and is coloured *brown* on the map. The class of diseases which characterise this realm are the worst forms of *malarious* (*intermittent* and *remittent*) *fevers*, associated more especially with *dysentery, diarrhœa, malignant cholera, specific yellow fever, hepatic affections and their results.* Our summer and autumnal affections characterised by *biliousness, diarrhœa, and bilious, gastric, or typhoid fevers*, approach, by the phenomena they express, the type of the *tropical* diseases. The paludal fevers of this *tropical disease-realm* prevail in their greatest intensity in flat, low-lying countries in the vicinity of marshes, the borders of lakes, shores of rivers and of the sea, and especially where the soil is damp underneath, and of certain geological formation. Sir Ranald Martin has clearly shown how the various soils affect powerfully the temperature and humidity of a place. Argillaceous and ferruginous soils appear in this realm to be especially insalubrious. The malarious fevers of this region make their appearance soon after the setting in of the rainy season, or when overflowed grounds, such as rice-fields, the partially dried-up beds and mouths of rivers, or irrigated plains, begin to dry up and leave portions of the surface of the land, whose subsoil is constantly wet, exposed to the rays of a tropical sun. From such a surface the belief is now universal that a miasm (recently reasserted to be of the specific nature of a fungus, but of which we know nothing) emanates, and acts as a poison upon the blood; and is apt to be developed under similarity of climate, season, and soil, and to produce diseases whose symptoms and course express a constancy and similarity of type. The great centres of these malarious diseases in the different continents are—(1.) In America—the shores of the Gulf of Mexico, the West India Islands, and the northern portion of South America; (2.) in Asia—India, China, Borneo, Ceylon; (3.) in Africa—the countries round the Gulf of Guinea on the west, Madagascar and Mozambique on the east, Algeria and the shores and islands of the Mediterranean on the north.

The whole mortality within this realm of disease amongst native troops was estimated by Mr. Johnston to be about 18 per 1,000 for all India, and about 75 per 1,000 for the more unhealthy districts. An average mortality was stated to be about 46 per 1,000 for the realm of disease now under consideration, or 4·6 per cent. annually. Active disease and premature death have been such common occurrences in India, that people have been led to believe these results to be the price which we must pay for the tenure of that country. Because disease and premature death were so common, they ceased to arrest public attention; and the causes of them, so frequently pointed out by medical authorities in India, were generally disbelieved by the home authorities when reported to them. At last the *Report of the Royal Commission on the Sanitary State of the Army in India* disclosed *to the public* the real state of the case, so long known to the intelligent but helpless medical officer. The state of affairs had been unceasingly represented by such men as Jackson, Sir Ranald Martin, Norman Chevers, Morehead, W. C. Maclean, Macpherson, and many others. It has been promised by the Home Government that the sanitary reforms and improvements indicated by the Commission in their published Report shall be carried out. Several great measures advocated in the Report, and most ably urged by Miss Nightingale in her excellent pamphlet, *How to live and not die in India*, have already been carried out. A Commission of Health has been appointed for each Presidency, and that for Bengal has already given public evidence of its zeal. These Commissions have been put into communication with the Barrack and Hospital Improvement Commission at the War Office, under the active sanitary direction of Dr. Sutherland, and which contains members representing the Indian Government. They furnish the Indian Commissions at the several Presidencies with all the more recent results of sanitary works and measures which have been of use at home. The condition of the soldier in India is now greatly improved. The worst personal causes of ill-health to which he was in former times exposed have been, or are being, removed. The soldiers are allowed and encouraged to cultivate gardens and to work at trades. The regulation *two drams* of alcoholic spirits have been reduced to one, and that *one dram* is to be diluted with water. An attempt is being made to abolish the spirit allowance altogether, and substitute malt-liquor instead. But the main cause of disease in India—want of drainage, want of water-supply for stations and towns, want of proper barracks and hospitals—remains still to a large extent to be supplied. In 1877, the rate of mortality for the whole army of India was only 12·71 per 1,000—a result more favourable than in any previous year, even although famine prevailed in a great part of the country, with an unusual amount of disease more or less consequent on it. In continuation of previous statistics, Dr. Bryden reports the history of the army up to 1876, and he shows that whereas the Royal Commission on the Sanitary Condition of the Indian Army stated in 1863, that the death-rate of the European Army in India ought eventually to fall to 20 per 1,000, the average rate for the five years, 1871 to 1875, was actually so low as 15·62. During 1877 it was 12·71, and in 1876 it was 15·32. In the Army of Bengal the rate was lower still, namely, 11·64 against 16·02 in 1876. Among the troops of the Bengal Presidency, the average rates for three quinquennial periods were as follows:—From 1861 to 1865 it was 29·30; from 1866 to 1870 it was 27·48; from 1871 to 1875 it was 18·50. On the other hand the average rate for invaliding during five years (1866 to 1870) was 49·66; while the rate for Bombay (11·84) exceeds any recorded since 1868. In all the three Presidencies the same diseases are the ten chief forms of sickness, with slight variation in the order in which they come. Stated in the order of frequency they are especially—(1.) For BENGAL:—*Malarial fever, venereal diseases, wounds and accidents, abscess and ulcer, respiratory diseases, rheumatism, diarrhœa, hepatitis, dysentery, eye-diseases;* (2.) For MADRAS:—*Venereal diseases, malarial fevers, abscess and ulcer, wounds and accidents, dysentery, diarrhœa, hepatitis, respiratory diseases, rheumatism, eye diseases;* (3.) For BOMBAY:—It is the same as for Bengal, except that *diarrhœa* comes before rheumatism.

The principal causes of death in each Presidency are as follow:—(1.) BENGAL—*Enteric fever, hepatitis, injuries, apoplexy, heart disease, phthisis pulmonalis, malarial fevers, respiratory diseases, dysentery, suicidal deaths;* (2.) MADRAS—*Hepatitis, dysentery, enteric fever, cholera, injuries, heart diseases, apoplexy, phthisis pulmonalis, suicidal deaths, malarial fevers;* and, (3.) BOMBAY—*Injuries, apoplexy, enteric fevers, cholera, hepatitis, suicidal deaths, phthisis pulmonalis, heart diseases, respiratory diseases, dysentery.*

As a whole the results of the statistics of sickness and mortality among European troops in India during 1877 are most satisfactory and encouraging, especially as representing the culmination of what is for the most part a gradual and steady improvement. Reduced sickness and mortality among women and children is also shown, so that ample evidence is afforded that the measures which have been taken since the Report of the Royal Commission to improve the sanitary condition of the British soldier in India have been attended with great success; and the results afford good ground for believing that when the many other improvements which are still required shall have been carried out, the sickness and mortality will be still further reduced (*Report on Sanitary Measures in India in* 1877–1878–1879, Vol. XI.).

The maximum intensity of *dysentery, yellow fever, diarrhœa, malarial fevers,* and *affections of the liver,* is observed in those countries which are situated under the line of greatest annual mean temperature—namely, 82° Fahr., which is the assumed equator of heat of the globe. This line of greatest heat also intersects the region of the earth where the greatest amount of water is deposited. Northward to 23° of north latitude, 53 per cent. of all the deaths are attributable to these diseases. During the six years, 1871 to 1876, *hepatitis* and *dysentery* have caused 24 per cent. of the total mortality in Bengal, 26 per cent. in Bombay, and 40 per cent. in Madras. In latitude 35° north, where the greatest annual temperature is only 77° Fahr., and just where the zone of the tropical disease-realm merges into that of the temperate realm of disease, these diseases cause about 14 per cent. of the whole mortality. To the southward, again, in the same latitude—namely, 35°, at the Cape of Good Hope—these diseases yield a loss of only 3 per cent. of the whole deaths.

II. The next well-marked *realm* is that in which varied forms of *continued febrile* disease take the place of the *malarious* or *paludal* fever of the torrid zone. The region where diseases of this type prevail embraces *realms* to the north and south of the equatorial regions, which may be generally described as in the north and south temperate zones. The southern boundary of the northern realm corresponds to the northern boundary of the tropical *disease-realm,* as already indicated by the isothermal line of 77°. In a northern direction it extends to latitude 60° north, and includes the British Isles, Norway, and Sweden; while in America its northern limit includes part of Nova Scotia and Newfoundland. Its boundary line to the north passes from 60° north latitude in a south-eastern direction on both continents, till it gradually declines towards the borders of Asiatic Russia in the Old World, and to the district between Boston and Philadelphia on the eastern shores of the New, and corresponds nearly with the annual isotherm of 41°. In the southern hemisphere it embraces the southern two-thirds of South America, the district of the Cape of Good Hope, and the southern half of Australia. It embraces the most healthy regions of the world, in which the prevailing causes of ill-health are mostly due to the *condensation of people in towns,* and the insalubrious and depressing conditions which necessarily arise from that cause. Emanations from nuisances tend to accumulate where mechanical and chemical arrangements of a sanitary kind do not remove the concentrated impurities. But where proper sanitary measures exist for the supply of pure water and free air, the artificial disadvantages inseparable from town-life may be greatly modified in their influences, so much so, that in our country (the least unhealthy of all) the mortality may range from 1·7 to 3·6 per cent. annually (*Reg.-Gen. Rep.* for 1853, p. 15). Nearly every type of disease has a representative in this realm; but, generally speaking, the general and mixed diseases are the classes which furnish the greatest mortality. *Typhus* and *typhoid fever* prevail between the parallels of 44° and 60° in Western Europe; *yellow fever* has prevailed on the southern shores of Spain, the north-west coast of France, and Northern Italy; *intermittent fever* in the Netherlands, Sweden, and Central Italy, and generally where marshy undrained lands exist. *Small-pox* especially prevails where vaccination has not checked its ravages; *leprosy* and *elephantiasis* prevail in Scandinavia; *pellagra* in Italy, France, and Spain; *plica polonica* in Poland and Tartary; and *consumption* and *rheumatism* everywhere. While, therefore, this temperate zone embraces, on the one hand, the extremes of temperature of the torrid and the frigid zones in the seasons of summer and winter, it is observed to have representatives of the types of disease which prevail in both these realms; and according as we approach its northern or its southern limits we find that the characters of disease become so modified, and their types are found so to mingle together on the confines of these disease-realms, that the *continued* type of febrile diseases peculiar to the temperate zones tend, as we approach the tropics, to merge into and to participate in those irregular intermittent characters which are peculiar to the type of the tropical malarious districts. It is only to a limited extent, however, that such modifications are found to occur; and there are some diseases which rarely tend to pass their geographical boundaries, unless especially favoured by *tropical* identity of climate on the one hand, or *temperate* identity on the other. Under the former circumstance yellow fever has been met with at Gibraltar, Cadiz, Lisbon, St. Nazaire, and even so far north as Plymouth Sound, Swansea, and Southampton; and it has also extended in America southwards beyond its usual limits; but so soon as the temperature falls below 55° Fahr., the importation of yellow fever into *this zone,* and its propagation or persistent existence in it becomes impossible. Diarrhœa, also, and dysentery are apt to prevail during the summer and autumn months; while under bad diet, defective ventilation, and generally imperfect sanitary measures, *contagious typhus fever* is apt to be propagated in crowded localities of towns, and in huts, hospitals, and barracks. Typhus and typhoid fevers seem to have their special habitation in this zone, especially between 30° and 40° of north latitude, and likewise the true glandular plague. They may be occasionally propagated beyond these limits by their specific poisons by human intercourse. The zone, therefore, which we inhabit, while it may be said to exhibit the greatest variety of disease-processes and types of disease, may, nevertheless, be considered a highly favoured district of the globe, inasmuch as the development and progress of disease and of epidemics are much more manageable, less intense, and on the whole less fatal than similar types of disease in the tropical realm, or even than they were centuries ago. Under proper sanitary regulations *typhus fever* and many other diseases in this country are greatly under control. *Cholera* may be said to be the only scourge which occasionally, as an epidemic, becomes *rapidly* destructive to life. But, by scientifically directed sanitary measures, and the increasing experience of an enlightened age, the spread of the *plague* in this country has ceased. In this realm "history records the existence of diseases of former days now happily unknown; while on the other hand, scourges unknown to ancient times devastate modern populations" (PLINY, SYDENHAM, BOUDIN). In the sixteenth century the Oriental plague, "like the destroying angel, spread its wings on the blast," and added to the miseries of that stormy and bloody time. Our physician-poet tells us that when the fate of England was about to be decided on "Bosworth's purple field," that plague, of most gigantic arm,

> "Rushed as a storm o'er half the astonished isle,
> And strewed with sudden carcases the land."

Then, as now, our standing armies and bodies of men congregated together especially suffered from the pestilential disease. It so thinned the ranks of Henry's victorious army, that few were left to see the conqueror crowned with Richard's diadem. Asiatic cholera may now be said to have taken the place of the Oriental plague, and it is often no less destructive and important in its results—sternly demonstrating "how one dread year performs the work of ages, when the pestilence mocks in his fury the slow hand of time."

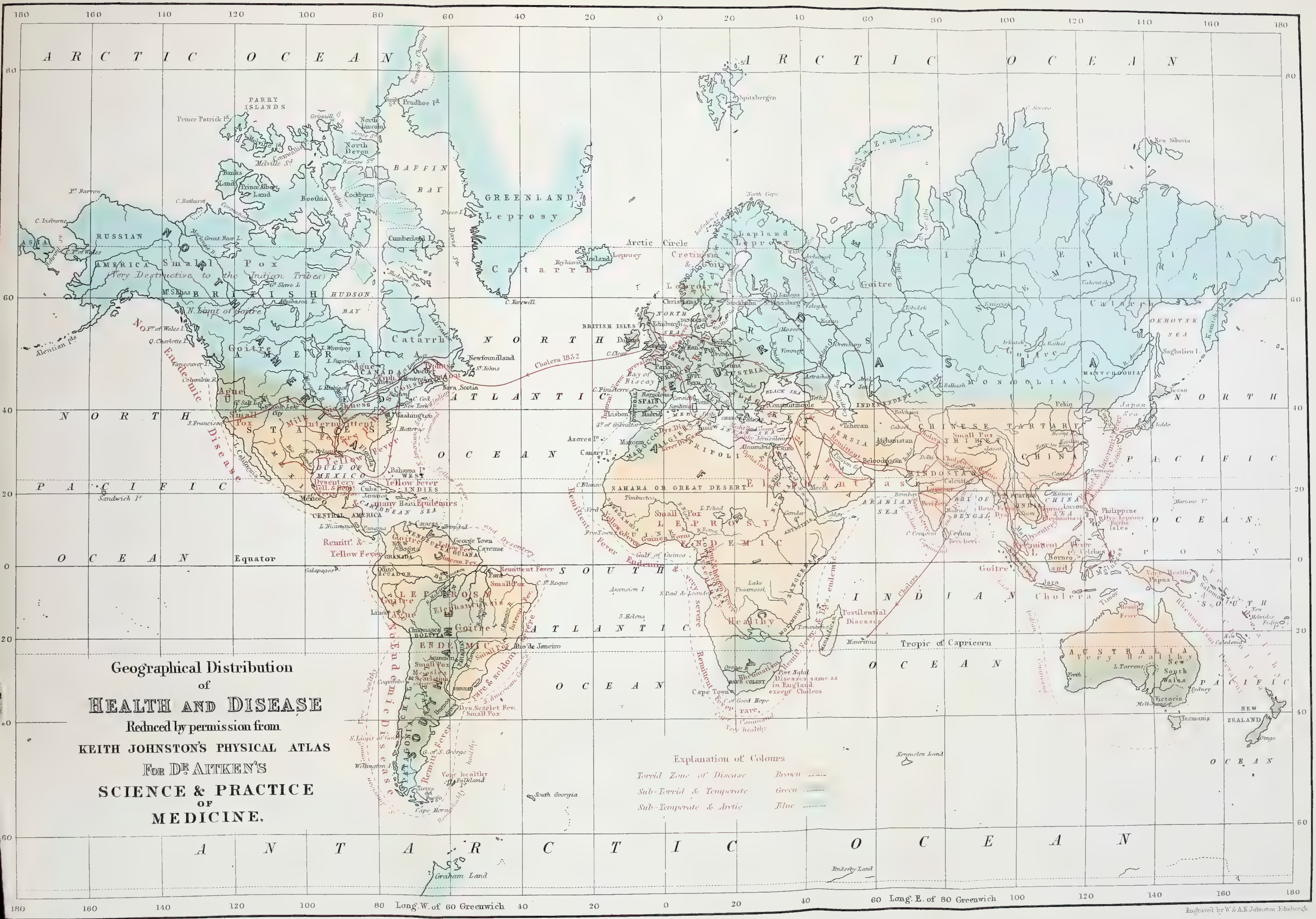
III. To the northward of this temperate zone, in the northern hemisphere, there is another disease-realm, where *catarrhal affections*, *influenza*, *scurvy*, *erysipelas*, *diseases of the skin* and *digestive organs*, and *various* CONSTITUTIONAL *affections* more especially prevail, to the exclusion of *malarious febrile diseases*, except on rare occasions in summer, and when *cholera* and *dysentery* may prevail. This *disease-realm*, in the polar isothermal zone, rejoices in a climate directly opposite to that of the tropical zone. Its southern limits are the northern boundaries of the previously defined region—namely, the isothermal line of 41° Fahr., or 2° or 3° of Reaumur. Commencing on the western coasts of North America above Sitka, it extends southward across the district of the

Canadian lakes, sinking south and east into Canada and Newfoundland to Boston and New York or Philadelphia. Thence it continues north-west nearly on 41° Fahr. annual temperature, when it crosses to Europe, and ascends till near the borders of Iceland, whence it sinks towards Norway and Sweden, and, running above St. Petersburg and Moscow, crosses to Siberia. Iceland being the best-known locality of this district, Mr. Johnston takes its peculiar diseases as the representatives of this realm. Every year, in spring or in early summer, it is visited by *catarrh;* and at short intervals it is visited by *catarrhal fever*—a *true influenza*, which has usually a great effect on the mortality. The majority of Icelanders are said to die before the age of fifty, from *asthmatic* or *catarrhal affections*, which are also prevalent in Greenland and Labrador.

CHAPTER II.

PLACES KNOWN AS MALARIOUS, AND NATURE OF MALARIA.

GEOGRAPHICAL facts, collected by medical writers from Hippocrates downwards, show that every country is unhealthy in proportion to the quantity of marsh, or of undrained alluvial soil, it contains, the inhabitants of such districts dying often in the ratio of 1 in 20, instead of in the average mortality in healthy countries; and the connection of a peculiar and specific class of disease—represented by remittent and intermittent fevers—with such marshy districts is an established and generally recognised fact. Ancient Rome was once the seat of so many fatal epidemics that the Romans erected a temple to the goddess Febris. These epidemics were known to arise from the great masses of water poured down from the Palatine, Aventine, and Tarpeian hills becoming stagnant in the plains below, and converting them into swamps and marshes. The elder Tarquin ordered them to be drained, and led their waters by means of sewers to the Tiber. These subterraneous conduits ramified in every direction under the city, and were of such considerable height and breadth that Pliny terms them "*operum omnium dictu maximum suffossis montibus atque urbe pensili subterque navigata.*" This system of drainage, which was continued as late as the Cæsars, rendered Rome proportionably healthy. On the invasion of the Goths, however, the public buildings were destroyed, the embankments of the Tiber broken down, the aqueducts laid in ruins, the sewers obstructed and filled up; and the whole country being now again overflowed, Rome has once more become the seat of an almost annual paludal fever, as in the times of her earliest foundation. The insalubrity of the Pontine marshes, past or present, is notorious. Three hundred years, however, before the Christian era, Appius Claudius drained them, by making canals, building bridges, and by constructing that magnificent road, portions of which still remain, and still bear his name. On the invasion of Italy by Theodoric, Cæcilius Decius gave a free course to the waters in the neigh-



Geographical Distribution of HEALTH AND DISEASE

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FOR D^R. AITKEN'S
SCIENCE & PRACTICE
OF
MEDICINE.

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bourhood of Rome, and the re-establishment of these immense marshes was one of the many disasters which resulted from the attacks of the Goths on Italy. Their present state is such that the Tuscan portion of Maremme, and indeed the whole of that district, may be said in summer to be absolutely depopulated, not a house retaining an inhabitant, except the guard-houses, with a few soldiers and custom-house officers; and these are relieved twice or thrice during the summer, with the Maremme fever almost invariably upon them.

Many districts in the East and West Indies, in Pennsylvania, and Continental Europe, are known to be active in the evolution of *malarious influences.* Such places are generally the deltas, marshy banks, and embouchures of rivers, in the plains extending from the bases of mountain ranges; partially inundated and irrigated lands, or such as are traversed by percolating streams or canals in wooded districts, termed *jungles;* the seaboard, especially where there is jungle or salt marsh; and in the Bengal district the stations of Calcutta, Chinsurah, and Berhampore are highly malarious (MARTIN). The woods and marshes of the Sunderbunds, covering a superficies of more than 20,000 miles, and extending 180 miles south and east of Calcutta, composed of marshy land covered with forest and underwood, together with the numerous embouchures of the Ganges, are well-known unhealthy districts. The partially dried-up marshes and beds of rivers have too often been fatal to our armies when imprudently and ignorantly encamped in their vicinity. In 1810 the plains of Spain, along the course of the Gaudiano, with its "lines of detached pools" and its ravines always "half-dried," could tell of a fever-stricken army. The pages of history remind us how our British soldiers perished on the low, dry-looking, sandy plains of Walcheren and of Rosendaal in 1794 and 1809-10. Our last war with Russia, during the campaign in Bulgaria, and especially at Varna in 1854, furnishes a no less melancholy record of the sufferings of British troops, and the persistent pernicious influence of a residence in malarious districts (*Medico-Chir. Trans.*, Vol. XL.)

In China we know of the miasmatic nature of the deltas of the Blue and the Yellow Rivers. In Africa we know of the Zais, the Orange, and the Zambesa as unhealthy rivers. In America the Amazon, the Orinoco, and the Rio-del-Norte are similarly deleterious. In England we have the fens of Norfolk and Lincolnshire still a source of malarial disease. In short, there is hardly a country which has not its marshy lands, so that abundance of work exists for health officers and chief commissioners of sewers to direct for good, where such "*heads*" of "*bodies*" exist; and the extent of disease proceeding from marshes has been shown in many places in Italy, Sicily, and Greece to be so great as to occasion more than two-thirds of the average mortality (See Vol. I., under "Ague" and "Remittent Fever").

Experiments have recently (1879) been made in the vicinity of Rome (Agro-Romano) by Professor Klebs and Signor Thommasi-Crudeli as to the etiology and nature of malaria. They were conducted in the following way:—(1.) The efficiency of various sorts of soils, of the air, and of the water in regions where malaria exists was first tested. (2.) The solid and liquid parts of these substances were separated, and examined after three different methods:—(*a.*) Starting from the supposition that *organisms* represent the germ of the disease, it appeared feasible to promote the development of these germs in certain substances which contain them in great abundance, by exposing the substances to such conditions as have been found to be most favourable to their development (a high temperature, 86° to 104° Cent., 106·8° to 219·2° Fahr.; moisture of the profound layer of the soil; rapid evaporation on the surface). (*b.*) After the substances had been thus prepared, minute particles were placed in different fluids and cultivated. This process was repeated successively, with the object of ascertaining whether the particles had retained the virulence of the primary substance. If so, it is clear that only those particles of the substance which are capable of development can be regarded as the germs of the disease, because few, if any, of the particles that are not capable of reproduction can have survived the successive cultivations. (*c.*) In every case the liquid was separated from the fine microscopic particles by filtration through plaster and other materials. The virulence of the *residuum* and of the *filtrate* was tested separately, by injecting each under the skin of rabbits, with the following results:—(1.) The *malaria* poison is contained in great quantity in the soil of malaria countries at seasons of the year when no cases of fever have occurred. (2.) At such times it can be obtained in favourably situated localities from the lower strata of air, which are immediately above the soil. This is done by means of a ventilator, which throws about 300 litres of air, with great force and rapidity, against a sheet of glass covered with glue, to which the particles contained in the air adhere. (3.) Water which stagnates in malaria countries does not appear to contain the poison, even if it should be rich in lower organisms. The experiments show that much water prevents the development of the malarial poison, and renders the germs harmless which may already exist. The experiments on animals gave the following results:—(*a.*) By inoculating rabbits with *liquids obtained directly from the soil,* or *prepared from the cultivated germs,* fever was produced which had a typical course. The intervals between the attacks lasted in some cases as long as sixty hours. The temperature rose during the attacks sometimes as high as 107°. (*b.*) If the *filtrates of the liquids* were used, the changes of temperature were less considerable, even if five times the original quantity was used. The fever was either of an intermittent type, or the temperature only rose once, immediately after the injection. The same results could be obtained by filtering the substances through a double paper filter. This proves (the experimenters hold) that the active organisms which convey the malaria poison can be more easily separated from the fluid in which they are contained, than is the case in many other infectious processes, such as *anthrax, septicæmia, pyæmia.* (*c.*) Some of the animals which had not been inoculated with the malarial fluids, but had accidentally been wounded, and had *septicæmia,* exhibited very different curves of temperature. In some cases there was a continuous rise of temperature; in other cases the fever assumed an irregular character. (*d.*) All the animals affected with malaria liquid exhibited considerable enlargement of the spleen. In normal animals, killed for the purpose of comparing the size of healthy with diseased spleens, it measured 4 centimetres long by 0·8 centimetre broad, by 0·3 centimetre thick. In an animal which died in thirty-six hours after two injections, the spleen measured 8·3 centimetres long, 2·3 centimetres broad, and 0·9 centimetre thick, so that the organ had increased by nine or ten times its normal size in thirty-six hours. (*e.*) A large number of these spleens (especially in the graver cases) contained a black pigment, like that found in individuals suffering from malaria. (*f.*) The *organisms* which the experimenters considered to be the real cause of malaria, as they were found not only in the virulent substances which were obtained from the soil and the water, but also by cultivation, and in the bodies of the sick animals, belong to the genus *bacillus* (see p. 152, Vol. I.) They exist in the soil of malarious countries in the form of numerous shining ovoid spores, which are in perpetual motion. These spores develop to long threads, both in the animal body and in the cultivation apparatus. The threads are homogeneous at first, but later on they divide into several sections, and new threads are formed from each of them. The experimenters consider them to be a special form of *bacillus*, and propose to call it the *Bacillus malariæ*, as they have been found in the bodies of infected animals. (*g.*) It belongs to the class *ærobii* (Pasteur)—*i.e.*, it must inhale oxygen in order to live and develop. It can only exist in liquids which are rich in nitrogen (*e.g.*, solutions of glutine, albumen, lime, and other liquids of the body, but not in water). The most favourable soil for their development in the animal body seems to be the spleen and marrow of the bones (see p. 368, Vol. I.; *London Med. Record*, Vol. VII., 1879, p. 395 : also No. 28 of the *Allg. Wien. Med. Zeit.*, 1879).

CHAPTER III.

ENTERIC FEVER AND OTHER FEVERS IN INDIA.

1. Enteric Fever.—This subject has attracted much attention of late, but not more than its importance demands. By some enteric (*i.e.*, typhoid) fever in India has been regarded as the one disease of all others from which our young soldiers die in that country (Bryden). By others the existence of enteric fever in India is altogether denied. Such extreme views can only arise from one of two causes—either, (1.) The profession are not agreed as to what enteric fever is. But this can hardly be the case after the exhaustive expositions and practical teachings on the subject since the beginning of the present century, and more especially since the elaborate researches of Drs. A. P. Stewart, Sir William Jenner, and Murchison, in this country, since 1848 (see p. 583, Vol. I.) Or, (2.) That sufficient opportunities are not afforded to our students of medicine for learning to distinguish enteric fever from other diseases, and of seeing and recognising the anatomical signs, after death, of its having existed during life. But this can hardly be the cause, for now much better knowledge is acquired, more extensive opportunities are afforded for acquiring special knowledge, and much better diagnoses are made in this direction than were wont to be.

(*a.*) *History.*—Evidence of the existence of enteric fever in India goes as far back as the experience of Clark, Annesley, and Twining. In their classic works they have pointed out that a fever often prevailed in Bengal which proved fatal under typhoid symptoms, and in which the small intestines were found ulcerated after death. Similar observations were made in Madras by Mouat and Shanks. But to Surgeon-Major Scriven, of the Bengal Medical Service (now—1880—Civil Surgeon of Lahore), belongs exclusively the distinguished honour of identifying enteric fever in its home in our Eastern Empire on the lines laid down by Gerhard, Stewart, and Jenner (see EWART, in *Trans. Epidem. Soc.*, London, Feb. 4, 1880). The prevailing opinions had erroneously excluded the affection entirely from India and the tropics; and even up to 1856 one of the greatest authorities on diseases in India, Dr. Charles Morehead, writes that typhus, typhoid, and relapsing fevers, are unknown in India (*Researches on Diseases of India*, Vol. I., p. 307). In 1861, when he again published his work, he found it necessary to modify his opinion; for cases had come under his notice of undoubted enteric fever, confirmed by reports of cases by Drs. Joseph Ewart, Goodeve, and Surgeon-Major Scriven (*Ind. Annals of Med. Science*, Vol. IV., pp. 65, 511; also No. XI., p. 141). Scriven had learned the error of the prevailing opinions from a *post-mortem* examination he made at Meerut, in November, 1851, in which the ulceration in Peyer's patches and other appearances characteristic of enteric (typhoid) fever, were unmistakably demonstrated; and the life-history of the case in its previous symptoms agreed fully with the view entertained of its true nature (*Indian Annals of Med. Science*, No. 8, p. 512, 1857). In the *Med. Times and Gazette* (January 28, 1854, p. 70), he published notes of three cases he met with in Burmah. At the European General Hospital at Calcutta, he subsequently saw one or two doubtful cases which recovered, and three other cases regarding the nature of which there never was any doubt, and in which the diagnosis was satisfactorily confirmed by the revelations of the autopsies (*Indian Annals*, No. 8, p. 521, *et seq.*). Thus, from 1854 to 1857 he continued to record cases (l. c., Vol. IV., pp. 65 and 511, No. 11, p. 141). In 1855, Deputy Surgeon-General Dr. Joseph Ewart (late Bengal Medical Service) detected the presence of enteric fever among the native prisoners in the Ajmeer Jail, Rajputana. He recorded and published in the seventh number of the *Indian Annals*, an epitome of the symptoms of three well-marked cases during life, and of the *post-mortem* appearances at the lower end of the ileum. When subsequently attached to the Megwar Bheel Corps, Kherwarrah, Rajputana, 1857 to 1859, he recorded several well-marked cases among the Bheels of the regiment and the aborigines of the district. Enteric fever then prevailed in the regiment and the surrounding villages, more especially in the autumn and early months of the winter season, when the worst forms of malarious fever abounded. Dr. Ewart's varied experiences then induced him to inquire, through the columns of the *Indian Lancet*, whether many of the so-called malarious remittents met with in hot countries were not in reality either typhus, typhoid, or relapsing fevers, modified and often materially aggravated by the presence of malaria in the system.

Drs. Goodeve, Barclay, and W. C. Maclean recorded cases of enteric fever in Madras in 1859 and 1860. Surgeon Hanbury, of the 33rd Regiment, at Dessa, recorded cases of enteric fever in the autumn months of 1859

(Army Medical Department Blue-Book, 1861, p. 335). The morbid anatomy of the cases he dissected struck him most as being different from anything he had before seen in India. The clinical character of the cases are fully given, and the etiological relations of the epidemic are considered and described. The *fact* of the occurrence of the epidemic during the autumn months in India, suggested at first that the cases were the usual "remittent" fever of India of malarious origin, the more so as numerous cases of remittent and intermittent fever were recorded in the regiment at that time. Such concurrent phenomena must often happen in India. There can be no doubt that in the experience of fevers in India, cases of enteric fever have been confounded with remittent fever, especially in the adynamic forms, because remittence is a characteristic feature of both fevers. This is abundantly testified to both by the late Dr. Murchison, and also by Professor W. C. Maclean, and very recently by Surgeons Hannah and O'Farrel (63rd Regiment), in an excellent account of enteric fever in Bengal (*Ind. Med. Gazette*, 1871). Dr. Joseph Ewart also bears testimony to the fact, "that in both natives and Europeans, who have been subjected to the influence of malaria, the fluctuations of temperature in enteric fever are *greater* than in those who have never been exposed to the action of the two poisons. The morning remission being so marked as to delude the observer into the belief that he is dealing with a malarious remittent simply, when he is really called upon to encounter enteric fever modified by the malarious infection of the system" (*Trans. Epidem. Soc.*, Feb. 4, 1880). Drs. Cornish, Ranking, and Peet, recorded cases of enteric fever in 1862. There are also cases described by the late Dr. Charles Murchison at Burmah. He had extensive experience in India and in this country, and his classic work on continued fevers (now in the second edition), is at once the best and most exhaustive that has ever been written on that subject. Enteric fever is distinctly described at Bangalore by Dr. W. Sims Murray (66th Regiment), with *post-mortem* evidence of its characteristic lesions, and a statement of its causes (*Army Medical Department Reports*, 1863, p. 516). In the *sixth* volume of these Reports, that for 1864, p. 547, Surgeon-Major Ranking, in charge of the Royal Artillery at Secunderabad, describes cases of enteric fever amongst the men, also cases of enteric fever combined with dysentery. The lesions seen after death were characteristic of both affections; and there was during life the characteristic rash of enteric fever. Implication of the glands of the lower bowel is by no means unusual in enteric cases in Europe, and examples of such extensive lesions from China are preserved in the Museum of the Army Medical Department. Dr. Ranking's cases show that a similar extent of lesion also occurs in India. The ilio-colic valve does not always constitute a limiting boundary to the lesions, either in dysentery or in enteric fever. In scorbutic dysentery especially, the lesion extends upwards beyond the valve into the ileum; and in enteric fever, especially in the tropics (China and India), implication of the solitary glands of the lower bowel is by no means uncommon in severe cases. Hence the etiological relations subsisting between enteric fever and dysentery are of very great interest and importance, and they are suggestively referred to in Dr. Ranking's excellent report of his cases at Secunderabad (l. c., Vol. VI., 1864, p. 5). I have had sent lately, by Dr. John E. Purdon (Army Medical Department), the intestines of three soldiers who died of enteric fever at Secunderabad in 1878, in which this implication is well marked. These cases he attended himself, and verified his diagnosis by *post-mortem* examination.

It is also important to notice etiological relations subsisting between malaria (as well as dysentery) and enteric fever. The late Dr. Hugh D. Massy describes cases of enteric fever in Ceylon, on a table-land 6,200 feet above the sea-level, amidst the mountains of the central province, encircled by still higher ranges of hills (l. c., Vol. VI., p. 496). In 1865 diarrhœa and dysentery became prevalent there, though fevers hitherto observed had been malarious; but Dr. Massy brings evidence to show a combination in the same patient of enteric fever with malaria. He recognises the two morbid states, and distinguishes between them. The fever was characterised by the usual "rose spots" in crops; and the first access of the fever was typically periodic, accompanying the increasing enteric symptoms. Sometimes, also, not only in India, but in this country, the characteristic enteric symptoms are so obscure (although the lesions may exist to a severe degree in the intestines) that the case is overlooked, until the specific lesions suddenly and unexpectedly become a source of danger. Some of these cases in this country do not ever take to bed, and they have been described as "walking cases" of enteric fever. Dr. Ewart in his practice in the College and General Hospital of Calcutta has seen the disease in all its phases,—so mild that persons have gone about their ordinary business during the greater part of the fever; so severe as to end fatally in a few days, either singly from an overpowering dose of the poison, or when complicated with serious malarious poisoning, or to terminate rapidly by uncontrollable hæmorrhage from ulcerated agminated glands, or by perforation of the gut and general peritonitis.

In our own Registrar-General's reports deaths are weekly ascribed to "simple continued fever"—a disease which (as the late Dr. Murchison observed) has not once been fatal in twenty-five years in the London Fever Hospital. Hence the official nomenclature may in some measure be responsible for cases of enteric fever not being entered as such, but under the head of simple continued fever—a name which may become a refuge for various otherwise undiagnosed affections.

Dr. Ewart records that ever since Scriven directed attention to the subject, enteric fever has been recognised as a constant factor in the sickness and mortality at the General Hospital at Calcutta, to which he was attached for several years, where he watched most carefully the course and conduct of the disease, and where fatal cases when possible were tested by *post-mortem* examination. In Dr. Scriven's discovery of the disease in young Europeans, and in Dr. Ewart's recognition of enteric fever amongst the natives of India, and in the cases dissected by Dr. Hanbury at Dessa, and those of other observers, the first thing that seems to have attracted attention was *the morbid condition of the agminated glands of Peyer, and of the solitary glands at the lower part of the ileum.* The endemic occurrence of enteric fever in India has since been established and acknowledged by many others besides those whose papers have been referred to; and especially by Drs. Norman Chevers, Sir Joseph Fayrer, Furnell, Lyons, Moffit, O'Brien, Cleghorn, and Green. A place has been assigned to the disease in the statistical returns since 1871; and before 1861 no cases of fever in India *were classed* as enteric—a fact which has given rise to the speculation that it is a new disease in India. The new classification has naturally arisen from improved diagnosis, as with the increase of enteric cases those of continued fever (of undefined species) have diminished (*Report on Sanitary Measures in India*, 1877-8-9, p. 34). The first appearance of enteric fever in the statistics of British troops serving in India, was in 1861; and subsequently there has been a continued increase of deaths recorded from this cause. Of late years the proportionate fever mortality has increased nearly a fourth; and the proportion of fatal cases entered as enteric fever to all other fatal fevers has quadrupled (*Report on Sanitary Measures in India*, Vol. XI., p. 204).*

The Indian medical papers for the past ten years have been constantly recording authentic cases of enteric fever by many of the most able and most zealous members of the Indian and British medical services practising in India, often with excellent graphic illustrations of the range of temperature curves, which greatly add to the value of the record. "The general result is, that enteric fever now figures as the most fatal form of pyrexial disease met with among the younger European soldiers sojourning in India" (EWART). Year by year its increasing extensive prevalence has been challenging attention, no doubt in consequence of the greater ability to recognise the disease. The diagnosis and appreciation of the pathological relations of enteric fever are matters of education. There are diagnoses which are superior, and there are others which are inferior. But more mistakes are made, and more cases are lost, from want of examining cases completely and systematically, than from not knowing the nature of the case when the examination of it is complete (SIR WILLIAM JENNER). The opportunities for studying enteric fever in our town and county provincial hospitals are much greater than formerly, because such cases are now taken and treated (under certain precautions) in our general hospitals, when before it was not considered safe for other patients that enteric fever cases should be mixed with them in the same ward. According to the recent statistics of Dr. Bryden for three years ending in

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* The idea that enteric fever is a disease unknown in India till recent years is not supported by evidence. The statistics of fevers in the Bengal Presidency from 1860 to 1877 show that during the eleven years 1860 to 1870 the total annual mortality under this head varied between a minimum of 2·63 in 1867 and a maximum of 4·85 in 1860. Speaking roughly, it averaged 3·6 per 1,000. Since 1871, when enteric fever was first separated, the annual mortality from all fevers has never exceeded 3·96 per 1,000, and in 1877 it stood at a minimum of 2·21. During these last seven years it has averaged 2·9. Not only, therefore, has there been no increase in the total mortality from fevers, but there has been a considerable decrease. But since 1871, enteric fever, which had not been entered before, has accounted for a large and increasing proportion of the fever deaths, and for the last few years it has accounted for the great mass of them. In 1874, for example, it accounted for 2·01 of a total fever mortality of 3·11; in 1875 for 1·59 of 2·77; in 1876 for 1·75 of 2·46; and in 1877 for 1·55 of 2·21. There is therefore good reason to suppose that many cases of enteric fever occurring in the years before 1871 were returned as "remittent" or "continued fever." Even now, with all the attention that has been brought to bear on enteric fever, it is found extremely difficult, and often impossible, to recognise such cases in their early stage, and they are constantly entered, on admission into hospital, as cases of remittent or continued fever. In all the fatal cases the results are recorded as verified on *post-mortem* examination (*Report on Sanitary Measures in India*, 1877-8-9, Vol. XI., p. 45).

1876, there were no fewer than 368 deaths from enteric fever among the European troops in India, which represent a proportionally large number of recoveries. Hence these fatal cases represent a significant and important amount of clinical work in diagnosis and in morbid anatomy, by a large number of medical officers scattered over the length and breadth of India, who are, in the ordinary course of duty, quite as competent from previous training, education, and experience gained in the medical schools of the United Kingdom, to diagnose enteric or typhoid fever, as to distinguish any other grave specific disease or fever included in our nosology (Dr. Joseph Ewart, l. c.) The diagnosis of enteric fever in India, even by those who have recorded cases in reply to official circulars, does not involve a greater margin of error in diagnosis than would have occurred if a similar number of medical practitioners had been applied to by circulars in England (Netten Radcliffe in *Brit. Med. Journ.*, June 26, 1880).

In 1877 the European army throughout India furnished 233 cases of enteric fever with 92 deaths—*i. e.*, 39 per cent. were fatal. The mortality under this head (1·60 per 1,000 of the strength of British troops) exhibits no marked variation on former years. Since 1871, when this fever was first recorded separately, the ratio has fluctuated between 1·34 and 1·87. If Bengal be taken alone, the loss equalled 1·55, compared with a minimum of 1·53 and a maximum of 2·01 during the previous six years. In Madras the mortality was 2·00, the highest yet attained, excepting 2·34 in 1872. The death-rate from enteric fever in Bombay, during 1877, was somewhat lower than in either of the other Presidencies; it equalled 1·39. During previous six years the minimum had been 1·31 and the maximum 2·22. As usual the disease was confined to no particular time and to no particular places. The admissions for the most part took place in the hot weather and rains. Cases were recorded in fifty-seven different stations. In many of these the attacks were limited to one or two. In Bengal the greatest number of cases at any one place was 17 at the hill station of Subathu in 11 months, with a mortality of five. In Madras Secunderabad returns 33 and Bangalore 15. There were also 11 at the hill station of Wellington (*Army Sanitary Commission*, Vol. XI., p. 44). The following abstract is intended to show the relative monthly number of admissions in the whole British Indian army from enteric fever, as compared with those from all other fevers (average strength 57,260 in 1877):

	Enteric Fever.	Other Fevers.
January,	9	1224
February,	14	803
March,	7	874
April,	17	1221
May,	44	2404
June,	18	2289
July,	22	2414
August,	24	2708
September,	30	1890
October,	19	2398
November,	11	2068
December,	18	1866
Total Admissions,	233	22159
Total Deaths,	92	32

Taking for granted that the diagnoses were correct, this table shows that enteric fever did not follow the same monthly distribution as other fevers. It was most prevalent during the hot months, while the other fevers were frequent during and just after the rains. Again, every station of British troops had cases of "fevers" during the year. But out of 98 stations included in the statistical returns, 44 had cases of enteric fever; the others escaped. The following stations suffered the most from enteric fever, namely:—Meerut, Agra, Meean Meer, Subathu, Kurrachee, and Ghizree, Secunderabad, Bangalore, Wellington, Belgaum. These nine stations, with less than one-fifth part of the strength of the troops, furnished about one-half of the whole admissions and deaths from this disease (*Army San. Com.*, p. 97). In Bombay, for every 100 deaths enteric fever accounts for 14; in Bengal, for 13; and in Madras, for 10.

In 1877, British troops in Madras Presidency (strength 11,016) furnished 65 cases of enteric fever with 22 deaths. Three stations,—namely, Bangalore (12 cases), Secunderabad (33), and Wellington (11), furnished in all 56 cases and 29 deaths, out of a total of 65 cases and 22 deaths. In other words, 22 per cent. of the aggregate strength of Madras British troops yielded alone 86 per cent. of the total enteric fever mortality. Why should the fever be so prevalent and so much more fatal at these stations? is the question put by the Sanitary Commission (*l.c.*, p. 176). An abstract of the annual statistics of enteric fever in the Madras Presidency, from the date of the first recorded case in 1861 down to 1876, shows that for the sixteen years ending in 1876, during which cases had been entered on the medical records under the designation of "typhoid" or "enteric" fever, there had been 443 such cases recorded, of which 139 had proved fatal, and the diagnosis of each of these 139 cases had been verified by *post-mortem* examination (*l.c.*, p. 205). "Of what disease did those British soldiers die" if they did not die of enteric fever? And "the question," Dr. Bryden says, "has also been asked by military men, how it happens that no regiment or battery escapes enteric fever in the first year, whatever cantonment in India may be selected for it?" The fact implies that the existence of enteric fever has been recognised all over India.

One cannot help observing, after reading many official reports regarding diseases in India, that opinions are still in many quarters divided, crude, and quite unformed as regards the pathology of fever and of fevers in the Tropics, just as they were in this country up to 1846, before Sir William Jenner's analysis of cases during life and dissections after death, gave the data for distinguishing enteric from typhus fever (see Vol. I., p. 584). It is also curious to note, as Dr. Ewart observes, that the nature and character of the opposition to the views of those who contend for the endemic and widely scattered existence of enteric fever in India, in some measure resemble the nature and character of the opposition which Stewart and Jenner encountered in this country when they succeeded in splitting up common continued fever into three distinct and well-defined diseases. We have accounts for example, (1.) Of typical cases of enteric fever in India as we see them in this country. (2.) Cases of enteric fever, as in the Madras command, have been considered by Surgeon-General Gordon to be merely "malarial" or climatorial fever of the continued type, with intestinal complications, the *post-mortem* lesions in which are said to be the same as, and not to be distinguishable from, those of enteric fever. (3.) The enteric fever of the country is still regarded by many eminent physicians as common continued fever with bowel complaint, just as it is by some in this country and in Germany. (4.) Sir Joseph Fayrer has expressed his conviction that there is a form of "fever" in tropical climates, which, as regards its cause, is not identical with the enteric fever of this country (*Brit. Med. Journ.*, June 26, p. 937, 1880).

Some one is now required to arise and do for Indian fevers what Sir W. Jenner did for the fevers of this country. These many and varied views held regarding "fevers" in India, remind us of the state of medical opinion thirty-five years ago, before the pathology of enteric fever, and the difference existing between that disease and the other continued fevers were understood. Looking at the subject of Indian fevers by the light of experience gained in the study of continued fevers in Great Britain, attempts seem to be made in India to identify fevers—(1.) By means of some anatomical character; (2.) by means of clinical symptoms; (3.) by an appeal to considerations of cause. In seeking for a cause enteric fever in India is by some classified as one of the results of *malaria*, and so dealt with by preventive measures for malarious diseases. But considerations of cause are the most questionable means of identifying diseases, because of the uncertainty as to causation. Enteric fever in this country is as yet only known by the totality of its symptoms, and *post-mortem* appearances, and other pathological phenomena; and from such knowledge we are still in this country proceeding to the investigation of its causes. Clinically the disease has long been known; but we have not yet become fully acquainted with the circumstances of its production (Dr. BUCHANAN, *Brit. Med. Journ.*, June 26, 1880, p. 973). During the first week of a fever in this country, it may not be possible to form a diagnosis as to enteric fever; for it begins so insidiously, that its commencement in numerous cases cannot be fixed. But given a fever with more or less diarrhœa, with spleen enlargement or epistaxis, with nocturnal exacerbations of temperature each night becoming more and more severe, with special characters of the temperature curve, with successive crops of rose spots on the skin, and with certain anatomical characters in the fatal cases, we know that such a case is one of enteric fever. Even if there be no spots, or if those present be not sufficiently characteristic, the diagnosis of enteric fever may yet be positive in cases where pyrexia of a remittent type has lasted upwards of a week, and is associated with diarrhœa, ochry stools, tympanites, and abdominal pain, enlarged spleen or epistaxis. But, if both the eruption and abdominal symptoms are absent, the diagnosis of enteric fever can still be arrived at by a process of exclusion—after carefully comparing the symptoms with those of other diseases with which enteric fever is most apt to be confounded; and practically, the following rule will be found to hold good:—" A fever which in this country (aguish districts excepted) persists beyond seven days, and which may be unattended with cutaneous eruption, or by signs of local disease in the head, chest, or elsewhere, is in all probability enteric fever, even though there be no symptoms of intestinal lesions. Almost the only source of fallacy is latent tuberculosis. The disease most apt to be confounded with enteric fever in India is undoubtedly remittent fever, seeing that they may both prevail together. The pyrexia of enteric fever is essentially remittent; and cases are recorded by Murchison, Trousseau, and Bartlett in malarious countries which put on at first an intermittent type—in fact, all the phenomena of ague. Vomiting and diarrhœa may also occur in both diseases; while enlargement of the spleen, cerebral symptoms, and the typhoid state are common to both. The eruption is perhaps the only distinctive mark to be relied on; and in every case of remittent fever complicated with abdominal symptoms it ought to be carefully looked for. Europeans in India (especially in illness) are liable to such a variety of eruptions—marks of insect-bites and the like—that the difficulty of diagnosis in India is thus considerably increased. In natives, also, the lenticular rash is not so easily made out as in Europeans; but with care it can be distinguished in those whose shades of colour verge upon brown rather than black. In a certain proportion of Europeans the rash is not observed; but even in fatal cases of this kind the characteristic and unmistakable ulcerations of Peyer's patches are nevertheless found after death (EWART).

It is the close resemblance of enteric fever to remittent fever which accounts to some extent for the fact, that it is only within recent years that enteric fever has been recognised as occurring in India; and no doubt it has existed to a much greater extent in India than had been recognised or shown by official returns. It is also still highly probable that latent or less well-marked cases are still classified under other heads. A remittent fever may, indeed, be so severe as to have no appreciable remission; and to distinguish the exacerbations and remissions of some cases from the well-known periodicity of enteric fever is by no means an easy task. But then, in addition to other signs, we have the daily observation with the thermometer, which shows that while the type of enteric fever is no doubt essentially remittent, what is known as the curve of temperature throughout the case is really characteristic. "Given a patient who comes under our notice in the *first two days* of his illness, we can, by taking his temperature carefully night and morning, predict with almost absolute certainty, in uncomplicated cases, whether we are likely to have a case of 'remittent fever'—that is, a fever of climatic origin—to deal with, or a case of 'enteric.' The value of this knowledge to the physician in the treatment of the disease cannot be over-estimated" (Dr. GEO. S. RANKING).

(*b.*) *Causation.*—As to etiological relations in India as in this country, enteric fever is essentially an endemic disease; and new sanitary arrangements have sometimes increased its endemic area, such as the substitution of drains opening into the interior of houses for scavengers and nightmen, and with a water-supply insufficient to prevent the escape of sewer emanations, as in Edinburgh, when an increase of enteric fever took place under the new sewage system, without compensating or ventilating arrangements. As a rule, any outbreak of enteric fever in India is local and circumscribed—sometimes to a single house or village; and the names of the village or the house has sometimes been given to the fever in this and other countries—a very bad mode of nomenclature. It seems most prevalent in autumn in India, as it is here. Warm, damp, weather, when drains are most offensive, and animal putrefaction most active, is often followed in this country by an outbreak of enteric fever; and an increased rainfall may sweep such impurities into water used for drinking purposes.

Boudin and others have tried to show that an antagonism exists between enteric and malarious fever; but the facts suggest a similarity as to the mode of origin rather than any antagonism; for in countries like India and Burmah, where enteric fever and malarious fever both exist, and where enteric fever has often been mistaken for remittent fever, the diseases are generated under somewhat similar circumstances. There seems to be some analogy between cases of enteric and malarious diseases. The prevalence of both varies with season, temperature, moisture: but enteric fever occurs when no malarial fever exists, and the prevalent season of one is not the prevailing season for the other. Recent residence predisposes to both. Both only prevail under infractions of local sanitary law. Enteric fever seems independent of overcrowding and deficient ventilation—prevailing not only in most dense, but in least populous districts of large towns, and of common occurrence in country districts, in isolated houses, both here and in India. It is also a safe conclusion, that wherever enteric fever originates, *something* is radically defective in local sanitary arrangements, and that either air, food, or drinking-water is polluted with decomposing excrement. It is, at the same time, impossible to deny that enteric fever is in some way (not well known) communicable by the sick to persons in health; and there are cases which suggest its independent or spontaneous origin from decomposing sewage and other forms of putrefying animal matter finding its way to drinking-water, contaminating air or food, and so giving rise to a fever (enteric) differing from fever resulting from other causes.

The Surgeon-General of the Madras Army, Dr. C. A. Gordon, recently reported to Government that the great mortality ascribed by medical officers to enteric fever was not, in his opinion, due to that cause at all, but to the ordinary climatic fevers—fevers more properly belonging to the climate and topography of the country. In this report he "overrides the diagnosis made at the bedside of the patient, and prefers his own opinion thus formed, to that of the medical officer who treated the case and saw it in all its stages. Dr. Gordon has thus incurred a grave responsibility in imputing to the officers of the Army Medical Department the habit of treating cases not according to the recognised principles of medical science, but empirically" (*Lancet*, Nov. 9, 1878). The Army Sanitary Commission concurs in condemning Dr. Gordon's conclusions. Dr. Gordon says that "the phenomena stated of late years to constitute specific pythogenetic fever are none other than such as were known by old and experienced medical officers in India as pertaining to fevers of the country, endemic and climatorial." He would have us believe that the whole Indian medical service has mistaken what he calls forms and results of climatic fever, "and even of other diseases, such as heat-apoplexy" for typhoid fever. The method by which he has arrived at his conclusions is greatly to be deprecated. It appears to have been as follows, as stated in the report of the Sanitary Commission:—"He compares the scientific nosological definition which has been given in England by systematic writers of well-known eminence of typhoid fever with the notes of cases entered in medical officers' case-books; and, finding that the two accounts do not always agree, he either rejects the evidence of the records, or he gives a new diagnosis of the case altogether from the medical officers' facts. And then, on the assumption that the opinion held in England of the absolute etiological relations of typhoid fever to filth causes, especially to sewer air, ought to hold good everywhere, and finding from such reports as he has been able to procure that there is an apparent absence of such causes at stations where enteric fever cases have been reported in Madras Presidency, he considers this as an additional reason for calling in question the existence of the 'specific' disease known as typhoid fever." Then he proceeds to "ascertain how far the cases said to be enteric fever by the medical officers who saw and described them (in many instances founding their diagnoses, at the last, on the morbid appearances found on dissection) among British troops in the Madras command harmonise with the descriptions given. It does not appear that Dr. Gordon himself saw even one of the cases during life, or was present at a single *post-mortem* examination of the fatal cases. He appears to have read the case-books in his office; and if he found that the symptoms did not 'harmonise' with those given by the 'recognised authorities,' who describe the disease as they saw it in quite different conditions, and in a climate differing in every essential particular from that of India, he, without hesitation, pronounces the diagnosis wrong, even in cases where the *post-mortem* appearances 'harmonise' with those given by the 'recognised authorities' as characteristic of enteric fever." Dr. Gordon's materials cannot be otherwise than misleading; and having been obtained as the result of the issue of circulars to a large body of medical officers spread over a vast area, they were almost necessarily untrustworthy. Anyone having experience of the difficulties associated even in this country in investigating causes of epidemics, must know how little value is attached to data got together in such a manner (NETTEN RADCLIFFE). He admits as far as symptoms and pathology (by which I presume he means morbid anatomy) are concerned, that a disease resembling enteric fever does prevail in India; but he denies that it is etiologically the same, and throws doubts as to the view held as to the etiology of the disease in England. While he states that the continued fevers of India are but one single form of fever, he nevertheless divides them into as many as twelve groups. And, even accepting the facts recorded in the returns issued, they, in some important respects, lead to conclusions diametrically opposed to those embodied in Dr. Gordon's paper. As to the 175 cases on which Dr. Gordon based his conclusions, he admits that seven were real cases of enteric fever, the others being so-called "climatorial" fever. Of what value could seven cases, scattered over thirteen years and throughout a wide territory, be in determining a question of this kind? But accepting the diagnoses as given by the recorders, twenty-five per cent. of the total cases furnished no evidence whatever, either in one way or another, with reference to the question of etiology; and, deducting these cases, it is found that by far the greater proportion of the remainder, according to the reporters, had occurred under conditions of "fæcal effluvium from latrines," or the patients had been exposed to chances of infection to local conditions which are known to be chiefly associated with enteric fever. How can these data be reconciled with Dr. Gordon's statement that, in not a single one of these cases was there any evidence of the existence of such "causes" of enteric fever as govern the prevalence of that disease in Europe? (NETTEN RADCLIFFE.) In his report Dr. Gordon thus questions the existence of enteric fever within the Madras command—*i.e.*, in Southern India and British Burmah; and from the internal evidence contained in that report, it is very evident that Dr. Gordon set about his work with preconceived ideas and a foregone conclusion, having first made a *definition* for himself, according to which his inquiries were to be conducted. His definition led him to look for "a specific fever with enteric ulceration, due to the presence of filth in the air, earth, or water," with the results stated in his report (*The Lancet*, August 14, 1880).

With regard to causation, Dr. George Buchanan, the experienced sanitary head of the Local Government Board, writes that, "Having come to know the disease in England, and not till then, we had gone on to learn lessons about its association with one and another external condition, and then by degrees we were learning its causation. But the numerous and valuable results that inquiries about causation had heretofore yielded, did not prevent our having, but rather led us to have, ever fresh doubts whether we knew *all the conditions for the appearance of the disease*, and assuredly had taught us that we did not yet know *the conditions for its production*. Inquiry as to the way in which fever had made its appearance in a household—to say nothing of inquiry, in a stricter sense, as to 'cause'—had been found to involve a process of living over again, as it were, the life of each patient before his attack, with differentiation of his life from that of other people. That process was difficult enough of accomplishment under the conditions of English society, but must needs be incomparably more difficult where a fever occurred in individuals of an alien race, having traditions and caste-notions separating them by wide intervals from the people who were endeavouring to investigate the origin and spread of disease among them. The impression that Dr. Buchanan had reached was, that it was perfectly permissible to regard much of the described fever of India as being identical with European 'enteric fever.' It was modified, no doubt, by circumstances of Indian life, yet did not appear to differ more from our own enteric fever than one case or one outbreak might differ from another case or from another outbreak of this disease when it was witnessed in Europe" (Discussions at Epidem. Soc., *Brit. Med. Journ.*, June 26, 1880, p. 973). "Dr. Gordon's use of the term '*causation*' was misleading and unscientific. Points were referred to as causes which in reality were nothing more than conditions associated with the prevalence of disease. The study of climatology in connection with disease was of the utmost importance, but the assertion that an infectious fever was of climatic origin, when even the local circumstances under which it existed were not recorded, was damaging to the cause of epidemiology; for, although it was obvious that climate governs every condition of life, health, disease, and death, yet that did not necessarily constitute it a cause of any special disease" (NETTEN RADCLIFFE, l. c.). However much climate (*i.e.*, heat, moisture, and vicissitudes of temperature) might modify an attack of enteric fever; and however much fatigue, exposure, depressing influences generally, or a rapid transfer from England to India, might predispose to an attack of enteric fever, they—none of them—caused it. In India, as elsewhere, enteric fever is generated by a *specific morbid cause,* fostered by, if not actually born, of decomposing nitrogenous matter; and this *contagium* possesses dormant vital activity.*

An experienced and distinguished authority on Indian diseases writes :—"That it is evident from the whole tenour of Dr. Gordon's report, that the search for the causes of enteric fever has been confined by him to the barracks in which soldiers are quartered, and their immediate surroundings. Under the improved sanitation of the present day, we can well understand that such causes were not to be found where alone they appear to have been looked for. The soil of the native bazaars and villages within easy walking distance from most Indian cantonments is sodden with excremental impurities, and the water of the tanks and wells from which the communities dwelling therein draw their water-supplies is equally impure. Is Dr. Gordon prepared to maintain that in such places no causes capable of exciting pythogenic disease exist, or that soldiers, particularly young soldiers, do not constantly enter such places, and expose themselves to whatever *contagium* they may contain? Secondly, typhoid fever has been carried into houses in this country just as seemingly free from specific causes of 'specific disease,' as the barracks referred to in this report; and the practice of watering milk, for example, with impure water is quite as common in India as it is (or was) in England.* When Dr. Gordon confines his attention in a question of this kind merely to the sanitary condition of barracks and their immediate surroundings, sanitarians will tell him that he has only investigated half his case."

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* No one has put this argument better than Dr. J. B. Russell (the very able Medical Officer of Health of Glasgow) in a most interesting paper "On the Conveyance of the Contagium of Anthrax to Hair-Factory Workers as illustrative of the Particulate Theory of the *Contagia.*" "Climatic cosmical conditions are beyond the sphere of practical etiology. They necessarily condition the evolution of the phenomena of particulate contagia; they are concerned in the seasonal variations in the prevalence of the disease; in regional limitations of disease; in the circumstances which at one time, and in one place, give wings, so to speak, to the contagia, and at other places, and at other times, obstruct their progress. We MUST recognise these facts if we wish to avoid the risk of falling into erroneous assertions of success for preventive measures, and also to justify at times what might seem undue confidence or undue anxiety as to the probable course of some disease whose contagium is dominated in its activities by these cosmical conditions. Yet cosmical agencies have only the same relation to practical etiology as to all biological phenomena, and no other. . . . The etiologist observes that the seasons bring their special tendencies to disease, that temperature and rainfall, and hydrometric and electrical phenomena are attended by proclivities to some diseases while they are hostile to others. But, knowing all this full well, he also knows that none of those physical agents (singly or combined) can create contagia—they can only condition their existence." In an investigation into the origin of an outbreak of a communicable disease a fallacy is apt to creep into our results by regarding the *medium* of communication as the *cause* of the disease. We may have twenty different and distinct *media* for the conveyance of one contagium or poison, and popularly it may be said that any one of these media will poison or is a poison; but, as a scientific fact, there is only one poison, and that is the *specific contagium.* The contagium of enteric or typhoid fever is the most versatile of all the communicable diseases of this country in its choice of a medium. In reference to an outbreak we may say, for convenience, that the victims were poisoned by milk, or by water, or by sewer gas; but, as a scientific fact, they were no more poisoned by any one of these media than the suicide is poisoned by the bottle or medium which contained the laudanum or poison which he swallowed. This initial fallacy of speaking of a *medium* as a cause gives birth to a brood of minor fallacies, which at once disappear before a clear perception of their parentage—*e.g.*, (1.) Arguments based upon the erroneous apprehension that one medium is the only medium, or, in the phraseology of the critic, that one cause is the only cause. The inapplicability of one so-called cause to the circumstances of another so-called cause is triumphantly paraded: the fact being that *media* alone are meant; and the one cause acting through all the media is the *contagium.* (2.) The critic points to the fact that your cause is often present when your effect is absent, or *vice versa,* the truth being that in that case we have not got to do with the relation of cause and effect at all. The medium is not a medium when the contagium is absent. We must look elsewhere for a medium, or prove the intermittency of its action as a medium. Or, (3.) The natural and essential properties of the cause are adduced in evidence of the absurdity of attributing to it baneful effects; the fact being that the *contagium* is a foreign and casual ingredient in the medium. The milk is "bland and nutritious, the delight of youth and the solace of age;" the water is sparkling and palatable; it has quenched the thirst of generations of men—how can it be said that either is the cause of enteric fever? No such allegation is made. The contagium is the cause; the milk or water only the occasional medium.

"As regards drinks generally, the Sanitary Commissioners report that the men appear to exercise very little caution when they go into bazaars. In one case of enteric fever at Poona, in a man who was a total abstainer, it is stated that 'he was in the habit of almost every day drinking milk and native drinks purchased from any native carrying them about, also of eating pies of native manufacture.'" A case of enteric fever in connection with a bakery at Baroda is described as follows:—"The patient had for some time been employed in the Commissariat bakery at Baroda, which is in a dirty, close, hot shed in the middle of the bazaar, where two native latrines (in which at the time of the medical officer's visit, when bread was being made, urine and excrement were accumulated), are within a few paces of the table on which the bread is mixed. There was no filter in the place, and the water in use for mixing the flour into dough appeared to be very dirty. Leading out of the shed is a native dwelling-room. Private Wilson, the patient, was obliged to spend several hours of every day in this bakery, and there can be very little doubt contracted his fatal illness from typhoid poison present in the atmosphere" (*Report on Sanitary Measures in India,* Vol. XI., p. 212). In the Report of the Sanitary Commissioner for 1877, Dr. Cuningham has described sanitary defects at some twenty stations intense enough, in an Indian climate, to aid in developing (as *media*) the disease in subjects otherwise predisposed by age and the influence of hot moist climate. They are of *the usual kind,* but chiefly resulting in foul damp subsoils, bad barracks, cesspits, suspicious water, unwholesome state of adjacent native towns, bazaars, and the like. The local condition of the hill station of Subathoo exhibits an absence of fundamental knowledge of common sanitary principles in providing a station, the object of which was to protect the health of the troops. A special committee which was appointed to inquire why seventeen cases and five deaths from enteric fever had occurred at this station in eleven months, reported that several of the barracks were "most faulty in construction, and consequently quite unfit for habitation. In some there was overcrowding, and a few of the rooms contained four rows of beds. The lavatory accommodation was very limited. The water supply deficient. The sides of the hills and deep gullies were fouled by the native population. The latrines were ill ventilated." The men of the 73rd Regiment at Subathoo were described as "pale, anæmic, and having a generally unhealthy appearance, quite unlike what British soldiers should be after eighteen months' residence at a hill station." Five officers at this station were attacked by the fever. Sanitary defects were also found at Kasauli and Dugshaie, where cases of enteric fever had happened. The predisposing influence of age and service is shown by the fact that, on a five years' average, 75 per cent. of the deaths occurred within three years of the men having landed in India, and 94 per cent. were men under 30 years of age (BRYDEN, l. c., p. 98).

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* With regard to milk, Dr. Ewart relates an incident that happened to him when Civil Surgeon of Ajmeer. A complaint having been submitted concerning the quality of the milk provided for the consumption of the prisoners, he found that it had a most offensive taste and smell. His native doctor suggested that this arose from its being mixed with impure water. We visited the village from which the milk was supplied. Some fresh-drawn milk was then mixed with water obtained from a neighbouring stagnant pool, which was used for ablution and bathing by men, women, and children, and around which were the evidences of fæcal pollution. The mixture thus procured corresponded, in its offensiveness of taste and smell, to that which had been objected to as unfit for use and condemned. On Dr. Ewart urging these facts in the proper quarter the contractor was changed, and no more milk adulterated with stinking water was supplied during the remainder of his residence at Ajmeer.

In conclusion, the Army Sanitary Commission considers that the disease in India is substantially the same as that which prevails in England; but that in the former country, some (conditioning) elements, such as temperature and malaria, may exist which are not operative at home. An analysis of the outbreak at Bengal stations, shows in several cases a distinct connection with impure water, or with defective drainage or conservancy; and the greater prevalence among mounted troops is accounted for by the effect of foul stables. The influence of youth and of short experience of Indian service in predisposing to attacks of enteric fever, is dwelt upon with great urgency by the Army Sanitary Commission. Dr. Bryden shows that among soldiers landed in India at twenty-four years of age and under, one in 185 must expect to die of enteric fever during the first two years of service. Surgeon-General Innes (Bengal Army) observes that the facts "teach us that very much of the advantage which should have accrued from the large sanitary expenditure of late years has been negatived by the continued importation of a class of soldiers, a considerable proportion of whom are only brought to India to die. . . . It may be confidently affirmed that the age at which vast numbers of our recruits enlist is wholly fictitious; and if this has been the case in the past, it is clear the evil must assume more formidable proportions under a system of yet shorter service; and so long as immature youths are drafted to fill the ranks of regiments serving in the plains, so long will the resources of the country be burdened by a mortality which is, to a great extent, preventible" (p. 35). Dr. Cuningham shows very clearly that whatever be the exact nature of enteric fever, the true antidote is attention to local insanitary causes. The Sanitary Commissioner of the Punjaub (Dr. Bellew) made an examination of the registers of the different towns and villages in that district, which showed that by far the largest portion of the mortality occurred within and up to fifteen days of illness; also, that out of 15,432 deaths no less than 3,676 were children from a few days old to five years of age.

In the reports of the Sanitary Commissioner with the Government of India (Dr. J. M. Cuningham), it has been shown that enteric fever is in the main a disease of young soldiers new to India. Of 169 cases in 1876 summarised by the Surgeon-General of Her Majesty's British Forces, 142 occurred in men under twenty-six years of age; and of these 61, or more than one-half, were under twenty-two. As to length of tropical service, the facts are equally striking: 146 out of 169 had not been in India longer than three years. Of the 146, again, 112 had not been in India longer than two years, and of the 112 no less than 82 were in their first year's Indian service. Dr. Bryden's figures are not confined to Bengal; they include the whole European Army of India. The deaths alone are taken, because in such instances the diagnosis is verified by *post-mortem* examination, and, therefore, there can be no mistake in regard to the nature of the cases. Of the 105 deaths due to enteric fever in 1876, 43 occurred during the first year's Indian service, 18 in the second, and 20 in the third, 23 between the fourth and tenth, and one beyond the tenth year. Of the 105, again, 71 were under twenty-five years of age, 28 between twenty-five and twenty-nine, and the remaining 6 at or over thirty. The experience of 1877 is to the same effect. Of the 92 deaths from enteric fever in the Army of India, 28 were of men in their first, and 20 in their second year's service; 9 occurred in the third year, and 14 in the fourth; the remainder took place between the fourth and tenth year of service in India. As regards age, 47 of the 92 were under twenty-five, 31 between twenty-five and twenty-nine, and the rest at more advanced periods of life. The experience of the five years 1871 to 1875 taken together is in accord with the facts which have just been cited regarding 1876 and 1877. The whole subject of the relation of enteric fever in India to age and length of tropical service is dealt with by Dr. Bryden with remarkable completeness and variety of illustration. As to age, he shows that of the 472 deaths due to enteric fever during these five years, 323 were in men under twenty-five, and only 9 in men over thirty-four; or to give the result in ratios, the mortality from this cause in men under twenty-five equalled 2·86; between twenty-five and twenty-nine it was 1·44; and in men of thirty and upwards ·32. The liability at the three periods stood at 61·91, 31·17, and 6·92 per cent. The length of Indian service has been shown only since 1873, as one of the particulars which it is important to consider in all fatal cases. Of the 368 deaths from enteric fever during the four years 1873 to 1876, 185 were of men under one year's service in India, 41 of men between one and two years, 51 between two and three years, 31 between three and four years. Of men above seven years' service there were only 10, or, to sum up the result in Dr. Bryden's own words, "75 per cent. of these deaths occurred within three years of landing in India, and 94 per cent. of the total were among men under thirty." The question is then put:—What is the nature of this fever? Is it the same as the enteric fever of Europe, or is it the enteric fever of Europe with a malarial element superadded? Is it, in fact, a typho-malarial fever? It may be observed, that the prevalence of enteric fever among European soldiers in India commences, and is most marked, in the hot months, before the fevers usually ascribed to malaria become so rife. "*Typho-malaria*" is an unfortunate name, which has mainly come from America, and cases were recently described as such at the Medical Congress at Philadelphia, but the decision was altogether against their being correctly described by that name. They were decided to be cases of malarious fevers, attended with typhoid symptoms, or they were cases of typhus fever overlaid by malaria. We have to grapple with enteric fever and with malaria in all its forms in India; and it is so much the worse for the chances of recovery of the enteric patient, if he is at the same time suffering from malaria in any form. The two morbid conditions (enteric fever and malaria) may coexist in the same patient. Cases of malaria may also die with typhoid symptoms; but these symptoms are common to many other diseases, and have nothing whatever to do with enteric fever. (See Vol. I., pp. 78, 291.)

Formerly it was the fashion to ascribe small-pox, measles, and scarlet fever to the operation of a single causative agency; and prior to the investigations of Gerhard, Stewart, and Jenner, typhoid, typhus, and relapsing fevers were each regarded as springing from a common source. But the result of observation during the past thirty years has been to demonstrate more and more clearly the non-identity of the poisons exciting typhus, typhoid, and relapsing fevers, with as much precision and exactitude as clinical research had proved long before the non-identity of the poisons setting up an attack of variola, measles, and scarlet fever. These views are now accepted with very general consent; hence it is difficult to subscribe to the hypotheses recently promulgated, that, on the one hand, enteric fever in India is simply one of the various expressions of malarial disease, and that it is only malarious, remittent, or continued fever with intestinal complication; or, on the other hand, that it may be the product either of a specific cause, or of malaria, or of both. In the experience of Dr. Ewart, malaria is never the cause of enteric fever in India; and although it is not necessary to accept in its entirety the pythogenetic theory of its origin, or the spontaneous generation of the poison, yet the intensely insanitary condition of the cities, towns, villages, and hamlets throughout India renders it inexpedient to dispense with the filth theory as unquestionably the best working hypothesis hitherto advanced to explain the endemic prevalence of the disease in that country; or to fall back upon the existence of two or more efficient sets of so-called causes for the same specific affection. There is, indeed, no more ground for allowing that enteric fever, as observed in tropical latitudes, can be roused into activity in the human subject by two or more sets of poisons—namely, marsh miasmata or malaria, and a specific poison, than there is for saying that any of the contagious exanthemata can be originated by more than one specific poison. Dr. Ewart states that no word-painting that ever was invented could exaggerate the abominable filthiness of the sleeping accommodation set apart for the convicts in Ajmeer Jail. They were arranged in groups of fifteen to twenty in narrow low-roofed cells, and packed far too closely together. A few earthenware vessels were provided as urinals and commodes. In this way they were herded together from 8 P.M. to 6 A.M. daily. After the removal of the prisoners, generally for out-door labour on the roads, the filth was taken away; but much of it was washed into shallow channels leading to a neglected cesspool just outside the south-western aspect of the building, from which unspeakable odours emanated; or in such a site as to allow the effluvia issuing therefrom to be wafted back into the interior of the jail by the prevailing wind. The well from which the drinking-water was drawn was only a few yards distant. In addition to direct self-evident sources of contamination, it was so near the cells, drains, and cesspools, that it must have been exposed to pollution by percolation through the intervening gravelly soil. For numerous instances of stagnant tanks, employed equally for bathing purposes, for yielding drinking-water, and water for the dilution and adulteration of milk, and as receptacles for all sorts and conditions of abominations and filth, the reader is referred to Dr. Joseph Ewart's paper on Enteric Fever in India, and to the Reports of the Sanitary Commissions of the various Provinces of India, some of which are referred to in the Report of the Sanitary Commission.

The etiological points in the history of enteric fever may thus be summed up, as Dr. Murchison has done, under the following heads:—

(1.) It is an endemic disease, or its epidemics are circumscribed. (2.) It is most prevalent in the autumn of all climates, especially after unusually hot and moist weather. (3.) It does appear to be independent of overcrowding, attacking the well-to-do and the poor indiscriminately. (4.) There may be grounds for believing in its generation, *de novo*, by fermentive putrefaction of fæcal and perhaps other forms of organic matter, independently of a previous case—but the question is not yet completely determined. (5.) At the same time, it may be communicated by the sick to persons in health; but even then the poison is not like that of small-pox, given off from the body in a virulent form, but is developed by decomposition of excreta *after* they are discharged. (6.) Consequently, an endemic or epidemic outbreak of enteric fever has to do with local media of propagation, and implies poisoning of air, drinking-water, or food, with decomposing excrement products, so that even a single case of enteric fever should at once be held to prove something wrong with the mode of getting rid of the excretions.

(*c.*) *Diagnosis and Classification.*—With the object of identifying diseases, Mr. Cornish is rightly of opinion that "we do not tend to improve our knowledge by classifying only the bad cases as 'enteric,' and the milder fevers as continued" (p. 108). He admits the fatality of the disease in India known as "enteric fever;" but he says "it would be preferable, instead of selecting such cases of continued fever as cases of typhoid, to discard altogether 'the term' 'continued fever' in army returns, and describe all these continued fevers as enteric or typhoid" (p. 109). Such a method cannot be called diagnosis, and upon this point the Army Sanitary Commission makes the following memorandum:—This view changes considerably the aspect of the question, and there is no doubt that the whole series of returns since the commencement shows that, while enteric fever deaths have increased, deaths from continued fever have fallen off. But a question arises at this point, namely—is the "continued fever" of Indian returns the same disease as the "continued fever" of home returns? The complaint against this heading has all along been its indefiniteness for practical sanitary purposes. Is it a climatic fever in India without malarial poisoning? There is no doubt of the importance of the questions which have been raised in Madras in relation to those fevers classed as "enteric." But the same difficulty can scarcely be said to exist where *post-mortem* examinations have been made. Practised pathologists have no difficulty in recognising the characters of fatal enteric fever there. Indeed, if no further light can be thrown on the subject, the real question would appear to be this,—Is there such a specific fever as enteric fever at all? or do these *post-mortem* appearances take place in non-malarial fevers in India of the simple continued type? This is the question raised by Mr. Cornish, and it is not quite the same as that raised by Dr. Gordon, who appears to think that the appearances found after death are those which have always existed in fatal Indian "climatic fevers of malarial character." The question can only be decided by clinical and *post-mortem* examinations conducted in India upon the principles set forth in this work under "FEVER," Vol. I., Part i., p. 257; and Part iii., Chap. iii., p. 395. The importance of the question to efficient sanitary work cannot be over-estimated. Enteric fever may also be confounded with tuberculosis.

In the Punjaub, fever deaths are registered without distinction of type or form (l. c., p. 35). "That the phenomena stated of late years to constitute specific pythogenic fever are none other than such as were known by old and experienced medical officers in India," is no doubt true; but then, the "old and experienced medical officers" did not rightly interpret the true significance of the phenomena before them, any more than did hundreds of "old and experienced" physicians all over the world, until their real significance was demonstrated within comparatively recent living memory. It is quite true, as Dr. Gordon points out, that Annesley and other Indian physicians have accurately described the morbid anatomy of true enteric fever, which he and they alike thought resulted from the adynamic form of true malarial remittent fever. The question of correct diagnosis is not one of merely professional importance, but involves the practical question of the necessity for discovering and removing unwholesome conditions at military stations. The contention of those who differ from Dr. Gordon is that, so far back at least as European records of disease go, enteric fever has always existed in India, although the malarial elements, always present there, often so modify symptoms that those of enteric fever do not square exactly with those described by authors whose field of observation has been confined to non-malarious countries.

II. TYPHUS FEVER.—We must now be prepared to recognise the occurrence of typhus fever in places where it has not hitherto been observed. In the United States and British North America it has prevailed extensively as an epidemic at different times. With regard to the existence of typhus in India, it is on record that "within the last nineteen years (since 1861) a contagious continued fever in the jails of India has attracted much attention. First, an epidemic in the Central Prison at Agra is described by Dr. Walker in 1861. The same fever had previously prevailed in the North-Western Provinces of India. It was believed to be typhus, and described by Dr. Walker as typhus. It only seemed to differ from true typhus in the absence of any eruption, and in the frequent occurrence of jaundice. In 1863–4 a similar fever, unquestionably propagated by contagion, and ascribed in its origin to under-feeding and overcrowding, was described in official reports to Government by Drs. R. Gray and De Renzy. The fever was at first remittent, but soon became continued. Jaundice was common, but no eruption was noted. In 1869 a fever still more resembling typhus was seen to prevail in the prisons of the Punjaub, and was described by Dr. Fairweather in the jail of Rawul Pindee. He gives clinical histories of several cases. Like the other epidemics, at first intermittent in some instances, it soon became continued. Jaundice did not occur; but there was a continuous eruption, whose characters were identical with those of pure maculated typhus. Enteric fever prevailed in the jail at the same time. Typhus fever must henceforth now be regarded as one of the diseases of India. A "doubtful" fever is also referred to as prevailing in many parts of the Punjaub, with a mortality so large as one in three of the patients. It was suggested whether it might not be a hybrid of typhus, enteric, and relapsing fever!

Pure maculated typhus was again prevalent in 1873, in the jail at Rawul Pindee. It has been described by Surgeon-Major Dr. John Ince, Civil Surgeon at Rawul Pindee, in March and May, 1873, in the *Report to Surg.-Gen. of Ind. Med. Dept.*, also in *The Ind. Med. Gazette* of Sept. 1, 1873. The deaths were 9·45 per cent. of admissions. Fever had before occurred as an epidemic in the jail, but its nature is acknowledged not to have been satisfactorily determined. The epidemic of 1873 was most carefully analysed by cases during life and dissection after death. The ranges of temperature showed it to be decidedly remittent by daily increase of evening temperature. No similar disease prevailed among the free population. It was possibly introduced from Peshawur Jail; but how it came there "there is no evidence to show." It is at present in that state of information which suggests a belief in its spontaneous development.

With regard to the *co-existence* of malaria with the continued fevers, there are good authorities for holding that view, especially as to malaria with typhus. There are those who hold that it is not improbable that remittent fever may become adynamic, and at the same time infectious, in consequence of overcrowding and neglect. This view involves the theory of the spontaneous generation of typhus, and may explain some of the cases described by Dr. Walker at Agra, in 1861 (MOREHEAD). It is also not unreasonable to suppose that under such circumstances, the fever may originate in malaria, with a poison like that of typhus superadded, and so appear to be spontaneously developed (MURCHISON). The causes of typhus fever in the tropics should also be inquired into in relation to the causes of "yellow fever."

As to the question of its spontaneous development, observations ought to be definite, and reduced to a numerical expression, otherwise experience and observations are of little value. Experience to be of value to any one besides the immediate observer, must be something capable of definite expression. The mind is apt to attach an undue importance to accidental occurrences, unless regulated by statistical records.

"If medicine is ever to become a science, it must learn to submit its methods, facts, and conclusions to the same rigid examination as is required in physical science" (HELMHOLTZ).

FINIS.

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